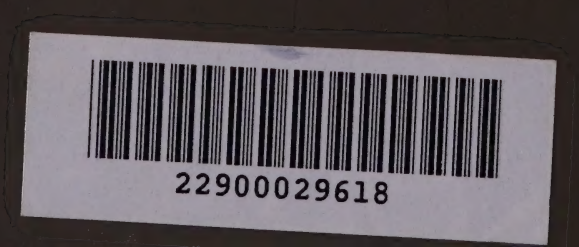
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THE

PRINCIPLES AND PRACTICE

OF

MEDICINE

EDITED AND COMPLETED FROM THE MANUSCRIPT

OF THE LATE

CHARLES HILTON FAGGE, M.D., F.R.C.P.

PHYSICIAN TO, AND LECTURER ON PATHOLOGY IN, GUY'S HOSPITAL; EXAMINER IN MEDICINE IN THE UNIVERSITY OF LONDON

BY

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PREFACE TO THE SECOND EDITION

In the present issue the whole work has been revised, and almost every page has received alterations or additions.

In the first volume, a fresh chapter has been introduced on Pyæmia in its medical aspects. In that on Enteric Fever, the cases given at length in proof of its origin by contagion have been curtailed, and the subject of treatment expanded. A section on Febricula and one on Vaccinia have been added. The chapter on Rubeola has been re-written. Additional facts will be found on the interesting subjects of the bacillus of Cholera and the contagium vivum of Malarial Fever. The account of the treatment of Diphtheria has been enlarged. The chapter on Syphilis has been transferred to its proper place among febrile specific diseases, and a short account of Actinomycosis has been added to the chapter on epizootic maladies which affect the human subject.

In the next section, that on Diseases of the Nervous System, a notice has been added of the condition now known as Peripheral Neuritis. Among Diseases of the Spinal Cord will be found short accounts of the remarkable affections known by the names of Friedreich and of Thomsen, and the chapters on Insular Sclerosis and Tumours of the Brain have received considerable additions.

The subject of Insanity, on which neither Dr Fagge nor myself could write except at second-hand, I entrusted to my friend and colleague Dr Savage, whose large experience and scientific habit of mind have enabled him to present in a short compass an account of mental disorders which will I hope answer the needs of the student of general medicine.

The section on the clinical aspects of Valvular Disease of the Heart, written by Dr Wilks for the first edition, has been left unaltered; but I have supplied what appeared to be wanted in other parts of the subject, and have added sections on the general prognosis of Disease of the Heart, on its peculiarities in children, and on the congenital lesions of the organ. Statistics on this subject and on several others have been inserted in the second volume, based either on cases which have come under my own care or on the records of Guy's Hospital: some of these were kindly compiled

for me by our Medical Registrar, Dr L. E. Shaw, others by former pupils of my own, whose names are stated in each case.

In the section on Diseases of the Lungs, the terms used in physical diagnosis have been revised, and the synonyms most used by French and German as well as by English writers have been inserted. Plastic Bronchitis has been transferred to the chapter on Bronchitis, because most readers would seek it there; and for the same reason Broncho-pneumonia (or Pulmonary Catarrh) has been placed immediately after Lobar or Fibrinous Pneumonia. The chief additions in this section are in the chapter on Inflammations of the Lungs and in that on Pulmonary Phthisis.

Among the chapters on Diseases of the Stomach and Bowels, fresh information will be found in that on Intestinal Obstruction.

The Diseases of the Liver have been rearranged in an order more easy to follow than that adopted in the first edition, which readers found practically inconvenient. If the considerations on nosology are valid which are submitted in this edition,* it must be hopeless to attempt a natural or permanent classification of diseases, and few pathological or ætiological facts should be strong enough to upset an arrangement which proves practically convenient.

The sections on Anæmia and on Arthritic Affections have been considerably expanded.

My own chapters on Diseases of the Skin, having been so recently written, needed only slight additions here and there. Zona and Xanthelasma have been restored to their natural places in this section.

Both Indices have been carefully revised and adapted to the present volumes.

By employing an ampler page and a bolder style of printing, the book has been made pleasanter to read, without its bulk being materially increased.

I have pleasure in acknowledging my obligations to numerous correspondents who pointed out errata or omissions in the first edition, as well as to much helpful and friendly criticism in the reviews which appeared at home and abroad. I have tried in the present edition to render the work more worthy of the reception it has met with, and trust it will remain a monument of the industry and judgment of my lamented colleague, and a reflexion of the principles and practice long associated with the school of medicine in which we both alike have been learners and teachers.

P. H. Pye-Smith.

Harley Street;
July, 1888.

* Vol. i, pp. 126, 385, 713 and vol. ii, pp. 745, 865, 1030.

PREFACE TO THE FIRST EDITION

Soon after Dr Fagge's lamented death, I was asked by his widow to complete and edit the Treatise on Medicine, which occupied his best energies for more than twelve years, and on which he was at work up to the last. I was also told that my late friend and colleague had himself desired that, if he were unable to finish his book, its completion should be entrusted to me.

A request thus enforced could scarcely be refused, and, in accordance with the wish of the publishers, the manuscript was put into my hands about eighteen months ago. I was the more encouraged to undertake what I foresaw would be a difficult and laborious task, by my honoured friend Dr Wilks, who, with characteristic kindness, not only collected and looked through the scattered parcels of manuscript and ascertained what progress the author had made, but offered to help in the work by supplying the most important chapter of Diseases of the Heart—that on Affections of the Valves. The brief but masterly account which Dr Wilks has given of this difficult branch of clinical medicine makes, in my judgment, one of the most valuable chapters of the book.

I was also so happy as to secure the intelligent and untiring help of Dr Carrington, Assistant Physician to Guy's Hospital, in seeing these sheets through the press. He has taken the greatest possible interest in its progress, and I have been continually indebted to his knowledge and care in verifying references and correcting errors.*

The whole of the vast field of internal pathology and therapeutics had been traversed, and the account of it now before the reader had been written —much of it rewritten—by Dr Fagge, with two considerable exceptions. He had left to the last the subject of Cardiac Diseases, to which he had made such valuable contributions in the 'Guy's Hospital Reports' and in Dr Reynolds' 'System of Medicine,' and that of Diseases of the Skin, with which also he was perfectly familiar. The part of the former subject which was unfinished when the pen dropped from his hand is well supplied by his old teacher Dr Wilks. The section on Cutaneous Diseases had not been begun, and this I have added to the best of my ability, much regretting, for my own sake as well as for the profession generally, that we have not the results of my late colleague's large experience, sound judgment, and wide knowledge in what was once his favourite field of work—or rather one of them.

Unfortunately we could find no table of contents or other indication of the plan and arrangement intended by the author. Here and there occur indications of the sequence of subjects proposed, and at the beginning of a few sections the order of the topics included under it is stated. But the general arrangement of chapters and the order of most of the subordinate sections had to be determined without such help. In so doing I have aimed at convenience rather than symmetry, and have been more guided by the clinical than the pathological relations of the several subjects.

* This promising physician and estimable man died of acute pneumonia a few months after the first edition appeared. In the short interval between the deaths of Dr Fagge and Dr Carrington, Guy's Hospital had experienced the loss of two other of its physicians, Dr Moxon and Dr Mahomed, a series of misfortunes happily unprecedented in its annals.

I am not sure whether the author would have placed Syphilis among the specific diseases or not. I should myself have done so, and have put Tubercle beside it. But the latter subject must clearly in this work be placed close to Inflammation, and the former, therefore, is allowed to hold an intermediate position. The order of the specific Fevers, the place assigned to such affections as Rheumatism and Rickets, and the grouping of the complicated series of Nervous diseases, must be always difficult to determine.

In drawing up the contents of each chapter, the head-lines of the pages, and the titles of the subcapitular sections, I have endeavoured to make the order easy to follow and also to facilitate reference.

In revising the language employed, though I have been anxious to make as few alterations as possible, there is, I suppose, scarcely a paragraph left untouched. The changes consisted chiefly in breaking up long sentences with relative clauses, and in omitting frequently recurrent adverbs—the corrections which commonly suggest themselves in proof sheets. I have, however, left the conversational style, which it would have been impossible, if desirable, to change without more extensive alteration than an editor is justified in making; and those who knew the writer will pardon a colloquial phrase, if, as I hope, they should sometimes while they read seem to hear his low rapid voice, and see his bright intelligent glance, and note his characteristic attitude of suspense, his quiet smile of doubt, or his eager nod of assent.

In the first volume, almost all of which had, I believe, been revised by the author, the corrections and additions have been few. In the second, I have been obliged, not only to add references to some of the most important papers which have appeared since the manuscript was written, but also here and there to supply footnotes or paragraphs.

I hope that the references to hospital Reports, weekly Journals, and Transactions of societies, whether English or foreign, will be sufficient for the student; in most cases I have thought it enough to indicate the year or the volume where a glance at the contents would identify the article referred to. In the case of authors whose systematic writings are again and again quoted, as Watson, Walshe, Murchison, Virchow, Cohnheim, Trousseau, Charcot, and the writers in Reynolds' 'System' and Ziemssen's 'Cyclopædia,' I have, in order to avoid repeated references, put against the name in the index of authors the title of the work or works cited *passim* in the text.

I feel that some apology is due for the frequent quotation from the 'Guy's Hospital Reports' and from other writings of those connected with that Medical School. But in truth one of the merits of this book is that while paying, I trust, due respect to the work of others, both in this country and abroad, it is essentially based upon experience gathered in the wards, the deadhouse, and the out-patient rooms of one of the great London hospitals. If some of the eminent physicians connected with the sister schools of medicine in London, Edinburgh, or Dublin would write a treatise on medicine like that of Dr Bristowe, and if they would put before the profession the invaluable traditions of pathology and of practice which each great school possesses, I am persuaded that we should all wish them to write with as little restraint as the author of these volumes.

P. H. P.-S.

Harley Street;
December, 1885.

CONTENTS OF THE FIRST VOLUME

PAGES

INTRODUCTORY CHAPTER

Definition of disease—Organic and functional diseases—Pathological anatomy and clinical characters as bases of nosology—Symptoms and diseases—Physical signs and subjective symptoms—General ætiology: exciting and predisposing causes of disease—Prognosis—Modes of dying—by paralysis of respiration—by asphyxia—by syncope—by hæmorrhage—by asthenia 1—19

CONTAGION

General pathological processes—Specific diseases—Contagion and miasm—Theory of infection—Contagium vivum—Microzymes—Their origin and life—Their exclusive action in producing specific diseases—Immunity from contagion—Protective inoculation—Modes of transference of miasmata and contagia—Theory and practice of disinfection . . . 20—36

FEVER

Pyrexia—Thermometry—Clinical course of pyrexia—Hyperpyrexia—Theory of fever—Possibly beneficial effect of pyrexia—Idiopathic and symptomatic fever—Raised temperature without fever—The physiological stages of fever—Concomitant phenomena of pyrexia in the nutrition, pulse, respiration, and muscles—Subnormal temperature . . . 37—51

INFLAMMATION

Historical review; Virchow; William Addison, Waller and Cohnheim; Goodsir—Direct observation of inflammation in animals—Redness, swelling, pain, heat, pyrexia—Exudation: membranous, catarrhal, purulent, diphtheritic, and gangrenous inflammation—Repair—Causes of inflammation: irritants and predisposition—Chronic inflammation . . . 52—69

PYÆMIA

Theories of pyæmia—Metastasis—Phlebitis—Septicæmia—Embolism—Infection—Microphytes—Practical application—External or traumatic and internal pyæmia—Channels of infection—Local distribution of secondary abscesses—Idiopathic pyæmia—Slighter forms—Diagnosis: especially from enteric fever and from tuberculosis—Prognosis—Treatment . . . 70—78

TUBERCLE

Anatomy and histology of tubercle: views of Laennec, Bayle, and Louis; of Virchow, Schüppel, and Ziegler—Tubercle a product of modified inflammation—Infiltrating tubercle—Caseous and fibroid transformations of tubercle—Koch's tubercle-bacillus—Result of experiments on animals: inoculation of tubercle: Perlsucht: infection by the mouth and by the lungs—Ætiology of tubercle: spread of tubercle in the body—Course of acute tuberculosis—Varieties of chronic tuberculosis: phthisis pulmonum, laryngis et ilei; tubercular inflammation of serous membranes: genito-renal tuberculosis; Addison's disease; tuberculosis of the lymph-glands and spleen; tubercular disease of joints; multiple tubercle generally—Historical retrospect 79—95

PAGES

TUMOURS

New growths—Innocent and malignant tumours—Virchow's characters of malignancy—Malignancy not an attribute of structure—Histological classification of tumours—Malignant osteoid sarcoma: round, fusiform, and giant cells; hæmorrhagic and alveolar sarcoma: melanotic sarcoma—Multiple sarcomata as a general disorder—Osteitis deformans—Lymphoma—Carcinoma—Heredity—Theory of new growths . . . 96—125

SPECIFIC INFECTIOUS DISEASES

AFFECTING THE WHOLE BODY AND ACCOMPANIED BY FEVER

Nosology—Bases of classification—General and local diseases—Specific Fevers. 126

TYPHUS

Historical sketch—Incubation—Course: first week: second week: crisis: recovery: death and post-mortem appearances—Complications and sequelæ—Ætiology; contagion of typhus—Prognosis—Treatment . 131—152

RELAPSING FEVER

History of its distinction from Typhus—Its occurrence in Epidemics—Its course—Occasional mortality, direct or indirect—The spirillum—Mode of contagion—Famine as a predisposing cause—Diagnosis—Treatment . 153—163

ENTERIC FEVER

History of the recognition of enteric fever and of its distinction from typhus—Nomenclature—Characteristic lesions—Incubation—clinical course and symptoms—recovery—abortive cases—fatal cases—complications—sequelæ—relapses—Diagnosis—Ætiology—a contagious disease—always produced from a previous case—methods of contagion: by air, water, &c.—illustrative cases—predisposing causes: soil-water, climate, age—Prognosis—Treatment—food, stimulants, drugs—cold baths and antipyretics—treatment of other symptoms—treatment during convalescence—Febricula . 164—208

SCARLATINA

History and distribution—Contagion—Modes of transference—Surgical and puerperal scarlatina—Incubation—Onset and course—The throat—The rash—Abortive and malignant varieties—Complications and sequelæ—Prognosis—Protection—Prophylaxis—Treatment . . . 209—216

MEASLES

History—Ætiology—Incubation—Onset—Course—Eruption—Varieties—Complications—Sequelæ—Protection—Diagnosis—Prognosis—Treatment 217—222

RUBEOLA

Recognition and nomenclature—Its characteristics—Discrepancies and probable explanation—Incubation—Onset and course—Diagnosis—Prognosis 223—226

SMALLPOX

Nomenclature—Incubation—Onset—Early rashes—Specific eruption—Varieties of the exanthem—Course and symptoms—Discrete, confluent, and modified smallpox—Complications and sequelæ—Prognosis and treatment.

Vaccinia—Inoculation—Introduction of vaccination—Course of the eruption—Its protective power—Its drawbacks—Its relation to variola . 227—242

VARICELLA

History of the recognition of chicken-pox—Its distinction from smallpox—Incubation and onset—Characters of the eruption—Symptoms and course—A disease of children—Diagnosis—Prognosis . . . 243—248

PAGES

ERYSIPELAS

Definition of the disease—Its contagion—A specific febrile exanthem—Relation of the phlegmonous form to facial idiopathic erysipelas—Incubation and onset—The eruption—Its histology—The symptoms and course—Sequelæ—Recurrent erysipelas—Diagnosis—Prognosis—Morbid anatomy—Treatment 249—258

DIPHTHERIA

History of the recognition of the disease—Its definition—Its pathology—Relation to croup and to "diphtheritic inflammation"—Micrococci and bacteria—Ætiology—Contagion—Course—Varieties—Complications and sequelæ—Albuminuria—Paralysis—Prognosis—Treatment . . 259—279

CHOLERA

History and name—Onset and early symptoms—Stage of collapse—Stage of reaction: the urine at this stage—So-called "cholera-typhoid"—Cholerine—Complications—Post-mortem conditions—Pathology—Ætiology—Theory of contagion—The Comma Bacillus—Prognosis—Treatment . 280—301

THE PLAGUE

History—Incubation, onset, and symptoms—Diagnosis—Anatomy—Fatality—Treatment—Conditions of contagion and causes of the decline of the plague 302—305

SYPHILIS

History of the disease—Nomenclature—Its place among specific exanthems—Origin and incubation—Primary lesion—Infecting and soft sores—Premonitory stage—Secondary lesions of skin, throat and mouth, eyes, &c.—Tertiary lesions of skin, tongue, palate, bones, and testes—Diagnosis—Infection—Special questions in syphilitic pathology—Prognosis—Treatment.

Congenital Syphilis—Its transmission to the child—Syphilitic placenta—Local manifestations—Its further transmission—Its treatment—Later effects 306—337

AGUE

Intermittent Fevers—Incubation—The cold, hot, and sweating stages—Regular and irregular varieties—Remittent Fever—Malarial cachexia—The spleen, liver, and other organs in ague—Melanæmia—Nature and laws of malarious contagion—Diagnosis—Treatment . . . . 338—355

YELLOW FEVER

History—Incubation, course, and events—Morbid anatomy—Diagnosis—Ætiology and pathology—Question of its contagious or miasmatic nature—Preventive, climatic, and curative treatment . . . 356—362

DENGUE

History—Names—Course and symptoms—The fever and the interval—The exanthem—Sequelæ—Pathology—Diagnosis—Prognosis—Treatment 363—366

EPIZOOTIC DISEASES AFFECTING MAN

Anthrax—Distribution—Varieties in animals—The Bacillus—Modes of infection—Varieties in man: Charbon, Intestinal and Thoracic Anthrax.

Glanders—Its history and nomenclature—Origin—Acute form—Eruption, ozæna, &c.—Pyrexia—Fatality—Chronic form.

Foot-and-Mouth Disease—An epidemic disorder among cattle—Occasionally occurs in man—Its mode of conveyance—Its symptoms.

Actinomycosis—Its occurrence in cattle—Its appearance in the human subject. 367—384

DISEASES OF THE NERVOUS SYSTEM

PAGES

Local or regional diseases compared with general and specific diseases—Principles of their classification and nomenclature—Order to be followed.

Nervous Diseases—Peculiarities of this group—Arrangement adopted 385—388

AFFECTIONS OF THE NERVE-TRUNKS

Peripheral Paralysis—Causes—Motor palsies of vertebral nerves—The reaction of degeneration—Sensory paralysis—Pain—Causalgia—Glossy skin and other trophic results—Diagnosis, prognosis, and treatment—Paralysis of cranial nerves—Motor, of the portio dura, hypoglossal, and oculo-motor—Sensory, of the trifacial and olfactory.

Neuralgia—Pathology and general characters—Trifacial neuralgia, &c.—Sciatica—Treatment of neuralgia.

Peripheral Neuritis—Its history and symptoms—Histology—Causes—Prognosis and treatment. . . . . 389—432

PARAPLEGIA

Difficulties of this section—Arrangement of diseases of the cord proposed.

Symptoms of paraplegia—Localisation—Superior and inferior limits—Incomplete paraplegia—Reflex movements—Knee-jerk and clonus—Muscular atrophy and rigidity—The bladder in paraplegia—The rectum and genitalia—Ammoniacal urine and bedsores.

Intrinsic Paraplegia—Myelitis—Histology of acute myelitis—Of chronic myelitis or sclerosis—Ætiology—Course and symptoms—Prognosis—Diagnosis from paraplegia due to spinal weakness, anæmia and congestion of the cord, from reflex and hysterical paraplegia, concussion of the spine, acute ascending paralysis, alcoholic and syphilitic paraplegia, spinal hæmorrhage, and tumours of the cord; with the pathology and symptoms of these affections severally—Treatment of intrinsic paraplegia generally.

Hemiparaplegia—Rachialgia.

Extrinsic or Compression-Paraplegia—from vertebral caries or cancer—from aneurysm, hydatid cyst, or meningeal tumour—Symptoms—Diagnosis—Prognosis and Treatment.

Spastic Paraplegia—in adults—in children—Primary and secondary—Anatomy—Symptoms—Prognosis and Treatment—Cases.

Meningeal Affections—Hæmorrhage—Acute and chronic spinal meningitis. 433—495

SPINAL PARALYSIS WITH WASTING OF MUSCLES

General characters of this group of diseases of the cord.

Sudden Atrophic Spinal Paralysis—Infantile palsy—In children—Its course, sequelæ, anatomy, and treatment—In adults.

Subacute Atrophic Spinal Paralysis—Its course and anatomy.

Lead Palsy—Distribution—Diagnosis—Tremors—Treatment.

Chronic Atrophic Spinal Paralysis—Progressive Muscular Atrophy—Symptoms—Pathology—Primary and secondary—Diagnosis—Treatment.

Bulbar paralysis—Progressive muscular atrophy in children—Hereditary form.

Pseudo-Hypertrophic Paralysis—Primary or myopathic muscular atrophy. 496—529

TABES DORSALIS, OR LOCOMOTOR ATAXY

History and nomenclature—Symptoms affecting movement and sensation—Lightning-pains—Visceral pains—Disorders of vision—Atrophy of joints—Perforating ulcer—Loss of knee-jerk—Complications—Course and event—Histology and anatomical seat of tabes—Discussion of the relation between the ataxy, dysæsthesia, and other symptoms—Ætiology—Diagnosis—Prognosis and treatment.

Hereditary tabes: Friedreich's disease . . . . 530—546

PAGES

INSULAR SCLEROSIS

History— Anatomy— Symptoms— Ætiology— Diagnosis— Course— Prognosis and treatment . . . . . 547—552

DISEASES OF THE BRAIN DUE TO LOCAL LESIONS OF THE CIRCULATION

Introductory remarks—Anatomical effects of obstructive disease of the arteries—Embolism, syphilis, &c.—Red and white softening—Effects of hæmorrhage—Renal disease, atheroma, miliary aneurysms.

Hemiplegia—Its characters—Diagnosis of the local lesion—Course and end.

Aphasia—Amnesia—Agraphia—Character, seat and physiology of aphasia.

Apoplexy—Symptoms, onset and course—Determination of locality—Its diagnosis from injury, poison, alcohol, pyæmia, uræmia, epilepsy—Diagnosis of the anatomical causes of apoplexy—Treatment of apoplexy and hemiplegia. 553—604

TUMOURS AND ABSCESS OF THE BRAIN

Tubercle—Syphilitic gumma—Glioma and other tumours—Symptoms: Headache and other cerebral symptoms—Vomiting—Temperature, pulse, and respiration—Optic neuritis—Localising symptoms—for the base—the cerebellum—the motor area and other parts—Diagnosis from hysteria, meningitis, &c.—Diagnosis between the several kinds of tumour—Prognosis—Treatment.

Red softening—Histology—Causes—Seldom or never primary.

Cerebral Abscess—Ætiology—Anatomy—Symptoms: general and localising—Diagnosis—Event. . . . . 605—644

CHRONIC AFFECTIONS OF THE BRAIN DUE TO DIFFUSED ORGANIC LESIONS

Chronic diffused inflammation: its great rarity—Hypertrophy of the brain.

Hydrocephalus: in children—Origin, pathology, and local anatomy—Effect upon the skull—Symptoms, course, and event—Treatment—Hydrocephalus in adults—Cases.

General Paralysis of the Insane—History—Ætiology—Symptoms and course—Anatomy—Diagnosis—Treatment.

Other forms of cerebral atrophy—Senile—Alcoholic—Saturnine, &c. . 645—673

AFFECTIONS OF THE MEMBRANES OF THE BRAIN

Tubercular Meningitis—History—Morbid anatomy—Relation of the tubercles to the inflammation—Their occasional absence in basal meningitis—Ætiology—Onset—Course—Later and final stages—Tubercular meningitis in adults—Diagnosis—Prognosis.

Epidemic Meningitis—History—Morbid anatomy—Symptoms and course—Varieties, complications, and sequelæ—Ætiology—Diagnosis.

Acute non-tubercular Meningitis—Anatomy—Varied causes—Secondary and idiopathic—Symptoms—Diagnosis—Prognosis.

Treatment of meningitis generally.

Adhesive thrombosis of cerebral sinuses—Hæmatoma of dura mater . 674—712

NEUROSES

OR FUNCTIONAL DISORDERS OF THE NERVOUS SYSTEM

PAGES

THE SPASMODIC NEUROSES

Arrangement of the Neuroses and particularly of Spasmodic Neuroses.

Facial or Histrionic Spasm—Spasmodic Wryneck.

Reflex and Functional or Movement-spasms: Saltatorial and Salaam-spasm—Scriveners' Palsy, Hammer-palsy, and other handicraft-spasms.

PARALYSIS AGITANS—Symptoms—Pathology—Ætiology—Treatment.

THOMSEN'S DISEASE—Its characters and symptoms.

TETANY—Description—Relation to Trismus neonatorum, Laryngismus and Carpopedal contractions, and of all to Rickets—Treatment.

CHOREA—Nomenclature — Symptoms and course — Chorea gravis—Morbid anatomy—Statistics—Pathology and Ætiology—Relation to Rheumatism and Cardiac disease—Embolic theory—Treatment.

TETANUS—Symptoms—Varieties—Ætiology—Pathology—Diagnosis—Events—Acute and chronic course—Mortality and Prognosis—Treatment.

HYDROPHOBIA—Inoculation—Premonitory symptoms—Characters when developed — Ætiology — Pathology — Diagnosis — Treatment — Prophylaxis—Pasteur's method of protective and curative inoculation . . 713—777

THE PAROXYSMAL NEUROSES

Introductory remarks—Relation to other neuroses and to each other.

MIGRAINE—Nomenclature—Symptoms—Pathology and causation—Treatment.

EPILEPSY—Definition—E. minor—E. major: aura: fit: sequelæ—Pathology, seat, and causation—Diagnosis—Treatment of the fit and of the intervals.

PAROXYSMAL VERTIGO—Symptoms—Auditory vertigo—Menière's disease.

PAROXYSMAL INSANITY—Peculiarities—Danger of violence or suicide—Relation to epilepsy, somnambulism and night terrors . . . 778—820

HYSTERIA AND HYPOCHONDRIASIS

HYSTERIA—A real and distinct disease—Mental and moral perversions—Hysterical affections of sensation and of movement—Contractures—Globus hystericus — Flatulence — The hysterical fit—Catalepsy—Sleep-walking—Ecstasy—Convulsions—Mania—Hystero-epilepsy—Anorexia and marasmus—Ætiology and Pathology of hysteria—Diagnosis—Prophylaxis—Treatment—Hysteria in men and boys.

HYPOCHONDRIASIS—Distinctions from hysteria—The supposition of imaginary diseases, commonly abdominal—Distinction from melancholia—May mask real disease—Treatment—Prognosis . . . . 821—845

PSYCHOSES OR DISORDERS AFFECTING THE MIND

(By G. H. SAVAGE, M.D.)

Insanity due to (1) disorder of functions of brain; (2) the expression by the nervous system of bodily disease; (3) the expression of disease of the brain—Classification—Causation.

Acute delirious mania—Acute and other forms of ordinary mania—Melancholia with bodily complaints—Hypochondriasis with mental complaints—Mental melancholia—Stupor, active or passive—Delusional insanity—Hallucinations of the senses—States of mental weakness—by defect—by instability—Specially named varieties of insanity: puerperal, alcoholic, moral, syphilitic, gouty, plumbic, febrile, phthisical, asthmatic, cardiac, renal and diabetic—Epileptic insanity—Paralytic and tabid insanity.

Testamentary capacity—Certificates and other medico-legal points.

Idiocy and imbecility . . . . . 846—875

PAGES

CRETINISM, DELIRIUM TREMENS, SUNSTROKE, ETC.

CRETINISM AND ALLIED CONDITIONS—The idiocy of cretins as an endemic disease—Its relation to goitre—Sporadic cretinism in England—The cretinoid condition in adults (myxœdema)—The condition which follows removal of a bronchocele in man—and removal of the thyroid in animals.

ALCOHOLIC POISONING—Chronic—its symptoms and treatment—Acute—delirium a potu—symptoms, prognosis and treatment.

HEAT-STROKE—Origin—Cardiac and cerebro-spinal forms—Mortality—Sequelæ—Diagnosis—Treatment.

MERCURIAL TREMORS—Course and symptoms—Diagnosis and treatment 876—896

DISEASES OF THE HEART

FUNCTIONAL DISORDERS

Changes in the frequency of the pulse—Irregular and intermittent pulse—Alternate and twin pulse—Palpitation—Cardialgia—Irritable Heart—Treatment of the above conditions—Tension, length, and volume of the pulse—Syncope—Distinction from epilepsy and from sunstroke—Treatment.

ANGINA PECTORIS—Onset—Symptoms—Event—Pathology and diagnosis—Pseudo-angina—Anatomy—Ætiology—Prognosis—Treatment . 897—917

AFFECTIONS OF THE MUSCULAR WALLS

DILATATION AND HYPERTROPHY—Physiology of the process—Its estimation—Its anatomy—Diagnosis—Symptoms—Ætiology—Treatment.

FIBROID DISEASE—Anatomy—Causes and pathology—Cardiac aneurysm—Symptoms—Course and event.

ACUTE MYOCARDITIS—Its origin, anatomy and results.

FATTY DEGENERATION—Fatty overgrowths and infiltration—Intrafibrous fatty degeneration: its histology, causes, and symptoms—Granular or pigmentary, and calcareous degenerations—Rupture of the heart . . 918—941

PERICARDITIS

Rarity of idiopathic pericarditis—Its antecedents—Anatomy—Fibrinous, serous, hæmorrhagic, and purulent effusion—Physical signs—Symptoms—Event—Adherent pericardium—Hydropericardium—Treatment of pericarditis 942—948

ENDOCARDITIS AND VALVULAR DISEASE

SUBACUTE, SIMPLE, OR BENIGN ENDOCARDITIS—Its relation to rheumatism and its ætiology generally—Its anatomy—Its signs.

CHRONIC, FIBROID, OR SCLEROTIC FORM—Its origin and anatomy.

ULCERATIVE, INFECTIVE, OR MALIGNANT FORM—Its origin, diagnosis, anatomy, and results.

VALVULAR LESIONS—Their anatomy and ætiology—Effects upon the heart itself and upon other organs—Diagnosis: the murmurs attending on valvular lesions—Their physical cause—Their rhythm—Their locality and conduction—Mitral stenosis—Its signs—Tricuspid stenosis—Mitral regurgitation, and tricuspid—Aortic and pulmonary stenosis and regurgitation—Special prognosis—Functional murmurs—The pulse in cardiac disease—Sphygmographic tracings—General symptoms of valvular lesions—Treatment of organic diseases of the heart.

Congenital diseases of the heart—Cardiac disease in children—Relative frequency and general prognosis of valvular lesions . . 949—986

DISEASES OF THE BLOOD-VESSELS

INCLUDING ANEURYSMS AND OTHER THORACIC TUMOURS

PAGES

ANEURYSM—Definition of the term—Varieties—Anatomy—Origin—Symptoms—of aneurysm of the ascending aorta—of the arch—of the descending aorta—Treatment—Abdominal aneurysms—Origin—Diagnosis—Treatment—Dissecting aneurysms—Statistics of aortic aneurysm.

INTRATHORACIC TUMOURS—Of the mediastinum—of the lung.

THROMBOSIS AND EMBOLISM—Causes of coagulation—Effects—Pulmonary embolism—Fibrinous infarction—Cases of recovery.

EXOPHTHALMIC GOITRE—History—Ætiology—Symptoms—Pathology—Event—Treatment . . . . . 987—1014

THE

PRINCIPLES AND PRACTICE OF MEDICINE

INTRODUCTORY CHAPTER

Definition of disease—Organic and functional diseases—Pathological anatomy and clinical characters as bases of nosology—Symptoms and diseases—Physical signs and subjective symptoms—General œtiology: exciting and predisposing causes of disease—Prognosis—Modes of dying—by paralysis of respiration—by asphyxia—by syncope—by hæmorrhage—by asthenia.

MANY attempts have been made to frame a satisfactory definition of *disease;* but hitherto no better result has been arrived at than that it is the opposite of *health*, a derangement of structure or of function in some part of the body or in the whole of it, causing pain or discomfort, or tending to shorten life.*

The art of medicine, however, is concerned not with disease but with *diseases.* And it is of the first importance that we should form correct opinions upon what constitutes *a disease*, and upon what principle diseases should be defined and named.

* Both Disease and Health are incapable of precise definition because they are not scientific terms. They are descriptive, popular and subjective.

"Disease" is "discomfort." Whatever causes bodily uneasiness or whatever by experience will sooner or later cause it, whatever interferes with our bodily functions, whatever tends to death, is disease. Health is the opposite condition, of comfort, ease, and ability to eat, sleep, move and perform the other functions of life.

Diseases have only this in common, that they all interfere with comfort or shorten life. There is no common cause for the pains of inflammation, of colic and of mechanical injury.

No line can be drawn between health and disease. Pathology is only physiology under various disturbing causes. Decay and death are as much physiological events as birth and life.

All diseases imply two things—an exciting cause, *quidquid irritans*, mechanical, thermal, chemical, parasitic, infective, or of unknown nature; and a reacting, living organism, *quidquid irritabile.* Stone in the bladder is not the disease; the disease is the reaction of the body. The severest injuries, the most violent poisons, produce no disease in a corpse.

There is a tendency after disturbance to return to the previous condition, if the equilibrium has not been too violently upset. This tendency has been called *Vis medicatrix Naturæ;* but there is no such force, and the so-called "efforts of Nature" often aggravate instead of curing the mischief. Our mortal bodies are not made to last for ever.

It is clear that if disease is not a single state nor the result of a single cause, it cannot be removed by any single method, or on any universal principle.

Hence all "Systems" of Medicine, like all "Universal remedies," are of necessity false. Iatro-mechanical and iatro-chemical schools, Brunonian and Antiphlogistic theories, Allopathy and Homœopathy, are all equally unreasonable; not wrong solutions of a scientific problem, but ignorant answers to an absurd question.

The art of medicine, therefore, as above stated, is not concerned with disease in the abstract, but with separate diseases, with pains and discomforts, the cause, seat, and origin of which the physician seeks to find, and, if possible, to cure in each case—*ἰατρεύει γὰρ καθ' ἕκαστα.*—ED.

From an early period in the present century, the utmost zeal and perseverance have been devoted to the search for morbid changes in the various organs and tissues of the dead body, and to tracing the relations between such changes after death and states of ill health during life. Numerous lesions have been thus discovered, the occurrence of which was altogether unknown to the physicians of former ages; and many of them have been found to betray their presence by signs which skilled observers can recognise without difficulty, even when the patient himself draws no attention to them, or is unaware that he is ill. On the other hand, there is a not inconsiderable number of disorders which force themselves into notice by the pain or discomfort they occasion, and which may in some cases actually destroy life; and yet after death all the organs and tissues seem to have their structure unaltered even when they are examined with the highest powers of the microscope and by every chemical test that can be devised. At first sight it appears as though the distinction thus suggested must be a very important and fundamental one, between *organic* or *structural* diseases attended with recognisable morbid changes and *functional* diseases in which no such lesions can be found.

By many thinkers, however, the occurrence of functional diseases is altogether denied. They are of opinion that if our inquiries could be carried far enough we should discover some morbid changes to account for every disorder or complaint, even the slightest. They say that they "cannot conceive" how any of the vital processes should fail to be carried on naturally so long as every part of the body retains an absolutely normal structure; and they seem to suppose that no further argument is needed to establish their opinion. But, as is well known, the history of philosophy is full of lessons which show how fallacious it is to assume that things are impossible because when submitted to the scrutiny of our faculties they seem to be inconceivable. The question is not whether an effect can arise without a cause but whether the cause must necessarily be of one particular kind because we know of no other kinds.

However, physiology suggests an analogy that may not be altogether without its application to pathology; the case of muscular fatigue. This was formerly supposed to be solely a result of the consumption of contractile material in the performance of work by the muscles. It is now known to be partly due to the accumulation in the tissues of those waste products which arise out of the contractile material so consumed. But the nerves and nerve-centres are also liable to exhaustion; and it seems not unlikely that the *neuroses*—as functional nerve disorders are called—may depend upon similar conditions. No doubt each of the factors which are concerned in the production of muscular fatigue can be conceived as within the range of our mental vision; the one appearing as a minute structural change, the other as a change of chemical composition. But it must still be important to maintain the distinction between diseases arising in such ways and those in which conspicuous lesions are present.

Unfortunately, however, the direction in which medical thought has tended during the last thirty years has been such as to lead writers to ignore functional diseases as far as possible, even apart from theoretical doubts as to their possible existence. Those who have been termed "pathologists"—whose studies have been devoted to the investigation of morbid changes in the dead body—have from the time of Rokitansky been disposed to maintain

that such changes are really the "diseases" in the cases in which they are present, and that their effects (which are generally the most obvious phenomena in the living patient and which used themselves to be called diseases) are only "symptoms." Thus whereas Paralysis and Dropsy, and Jaundice, were once conspicuous members of the nosology, they are now almost banished from it. Sometimes, as in the case of Apoplexy, the very name has been taken from a clinical condition, and transferred to the lesion which, according to modern investigations, is more or less constantly present. And to account for many of the slighter complaints to which mankind are liable, hypothetical lesions—such as hyperæmia and anæmia of a variety of organs—have been lavishly created on speculative grounds.

It seems to me that all this is as incorrect as it is inconvenient. The two principal objects of the art, and even of the science, of medicine are to prevent or to cure disease; and for those who alone wish to study medicine, the part of each disease which is of real importance is that which can be recognised during life.

It is time to admit frankly that some of the reasons which have generally been assigned for the cultivation of morbid anatomy can no longer be justified. It used to be said that no one could properly treat a patient without understanding the nature of his disease; and when the microscope was first brought to bear upon pathology, it was in the hope that improved methods of cure would directly result from the discoveries that might thus be made, one line of practice being applicable if a morbid change was inflammatory, another if it were degenerative, and so on. Now, within the last few years there has been much discussion as to the nature of tubercle, as to the part played by inflammation in pulmonary phthisis, and as to the degenerative or inflammatory character of arterial atheroma. But will anyone maintain that our treatment of the patients in whom these several affections occur has been based to any appreciable extent upon our theoretical views with regard to them? It rather seems to me that while writers have been insisting on the dogma that Pathology is the true basis of Therapeutics, practitioners have been too much disposed to fall back upon an unexpressed notion that structural changes of whatever kind are but little likely to be interrupted in their progress by the drugs which we can administer, or by any other curative measures which we can adopt. With the dogma itself I should, indeed, have little fault to find, if by "pathology" were to be understood, not merely the morbid anatomy of a disease, but the best attainable knowledge of its origin, course, and termination. In the latter sense the term has of late been seldom employed in England, but so I shall always endeavour to use it in the present work.

Morbid anatomy, however, may in one sense be said to be the true foundation upon which nearly the whole art of medicine, including therapeutics, should, as far as possible, be built. But the reason for this is a very different one from that suggested in the previous paragraphs. It is that until we have traced a disease to a definite organic lesion we can seldom be sure that cases which seem to us to be identical may not in fact differ from one another in their nature; and that upon their real identity depends all certainty of knowledge as to their prevention or cure. Probably no structural change has ever been discovered without leading to a further subdivision of the corresponding disease, in consequence of there being cases, more or less similar, in which the change in question is absent. Even in

an affection so restricted in its scope as "bulbar paralysis" this result has not failed to follow, since the discovery was made that it frequently depends upon a primary atrophy of certain nerve-nuclei.

The older writers on medicine have often been reproached for looking on diseases as entities rather than as morbid processes of which the living structures are the seats. But what can be more of an entity than a tissue affected with an organic lesion? And how could one go further in giving to a disease a substantive existence than by confining it to that which can be seen and handled, and regarding as "attributes" or "symptoms" all its attendant vital phenomena? It seems to me, therefore, that Dr Bristowe is right in maintaining, in the introductory chapters of his work on medicine, that the real disease is not merely the organic lesion, but a "complex," or "sum total, of morbid changes in both function and structure."

I hold, therefore, that in constructing a Nosology, the main thing to be attended to is that each disease should represent a homogeneous group of cases, resembling one another in their principal features. It is not necessary that all diseases should be constituted alike; we have in *scabies* one of which the fundamental character is the presence of a special cause, in *aneurysm* one in which it is a lesion, in *migraine* one in which it is a series of morbid sensations and disorders of function. Whenever the whole of a group of cases can be traced to a single structural change, this should doubtless be regarded as the most essential element in the definition of the corresponding disease, and it should, if possible, furnish the name. But if the effects of several lesions are identical, so that they are clinically undistinguishable from one another, it is often better to keep them united under a common designation. Such an arrangement may be regarded as merely provisional, but it is one which is likely to be needed for very many years to come. And for my own part, I should generally give to these comprehensive diseases the most prominent places in the list, and I should assign altogether subordinate positions to the affections included in them, notwithstanding that the latter are alone known to the morbid anatomist. It is, however, often necessary for the two sets of names to stand side by side.

The process by which we give a name to a case, or by which we determine to what disease it belongs, is one of analysis; and there is nothing surprising in the fact that in some cases we can carry this process further than in others. To say that a patient has "hepatic ascites" is far more accurate than to speak of him as affected with "cirrhosis of the liver," unless we are certain that after all the lesion may not prove perihepatitis, or cancer, or gummata. To define a cerebral case as one of "hemiplegia" is much better than to call it "softening of the brain," if the lesion is but little less likely to be a clot of effused blood, or a tumour, or an abscess. And yet there are other cases in which we can be sure that cirrhosis of the liver, or that softening of the brain is present; and it is obviously better to name such cases according to their respective lesions. It is just as though a chemist were sometimes unable to discern the ultimate elements of the substances submitted to him for analysis, and had to be contented with determining their proximate principles; and there is this further force in the comparison that for practical purposes it is often far more important to know the "proximate" than the "ultimate" diagnosis.

Above all, I wish to protest against the statement so commonly made, that it is "incorrect to call hemiplegia, or ascites, or any like condition, a

disease, because it is really only a symptom." I have already endeavoured to point out that the phenomena which are commonly spoken of as symptoms are parts of the disease to which they belong, no less than the lesion, or the specific cause, or whatever else is taken as its main characteristic. It is true that, when they afford the means by which we infer the existence of a hidden morbid change, they almost necessarily bear to it the relation of effects to a cause, so that one then naturally comes to look upon them as subordinate, and of secondary importance; but this only shows that the term symptom is in fact a relative one. So long as we are unable, either during life or after death, to discover any cause for a set of clinical phenomena, they certainly are not symptoms, for they indicate nothing. There can be no symptom until the disease of which it is a symptom is discovered. As the process of analysing a case, or a group of cases, advances, the name which we apply is rightly changed at every step; and each "disease" becomes in its turn a "symptom" of the disease which succeeds it. I suppose that itching was once hardly distinguished from having the itch; afterwards, when the characters of the eruption of scabies were made out, itching was looked on as a symptom, and the eruption was the disease; now that the *sarcoptes* has been discovered, the eruption is itself reduced to being a symptom. If, in an important class of cases, the analysis is always arrested at a certain point, it is absurd to say that the ultimate fact in those cases, which cannot be made to reveal anything further as to their action, is "only a symptom." So far from hesitating to classify clinical phenomena as diseases when we are unable to trace them to their causes, I only wish that we had a more complete supply of suitable names for them. It is very probable, as Dr Moxon has argued in the 'Guy's Hospital Reports' for 1870, that a good "clinical nomenclature" would be quickly followed by an immense advance in our knowledge of such conditions.

Moreover, even when we do understand the whole pathology of a case, and when the phenomena which characterise it clinically are fairly to be called symptoms, the distinction between them and the anatomical changes is often very uncertain and arbitrary. Thus, in acute tuberculosis, if with the ophthalmoscope one discovers a tubercle in the choroid membrane of the patient's eye, one is very likely to think of it as a symptom; but in reality the disease itself consists of nothing but a multitude of precisely similar tubercles. So, again, the enlarged liver of mitral disease, the osseous nodes of syphilis, the swollen joints of acute rheumatism, each belong in strictness to the pathological anatomy of these several complaints. Nor, if we would be logical, can we stop even here; the same thing must be said of all cutaneous eruptions, and of all affections of mucous membranes, down to the rose-rash of enteric fever, or the scanty sputum of spasmodic asthma. In that part of 'Ziemssen's Cyclopædia' which is devoted to the exanthemata, the appearances seen on the skin are in fact described in some instances (but not in all) apart from the "symptoms," and under the head of "anatomical changes." But for my own part I prefer to employ the former term in its natural sense, even though I should be driven to admit that such a use of it is conventional. On the other hand, in the case of a functional disorder, to speak of the clinical phenomena as symptoms is really to reduce the disorder itself to an abstraction.

Thus, it appears to me that all the morbid changes, whether of structure or of function, which are recognisable during life may properly be called symptoms, notwithstanding the logical objections to this proceeding.

Symptoms, so defined, naturally divide themselves into two distinct classes. Some of them are merely "subjective," or, in other words, they are appreciable by no one but the patient, so that our knowledge of them rests solely on his statements. Others are "objective;" that is, they can be directly observed by other persons. Indeed, they can be investigated, even though the patient himself is unconscious; or, if in possession of his senses, he may be the only person who cannot perceive them.

Now, subjective symptoms are open to many sources of fallacy. They can often be feigned by one who desires to deceive, pretending to be ill when he is not really so. Moreover, and this is of far greater importance, their severity depends almost entirely upon the nervous susceptibility of the individual in whom they occur. A hearty labouring man will take no notice whatever of a pain which would drive a delicate lady to her bed for several weeks. In the former case one must be as careful not to overlook the occurrence of disease, as in the latter case not to assume its presence without sufficient grounds. And yet it is essential that great and constant attention should be paid to subjective symptoms, for they often yield information which can be obtained in no other way.

The physician elicits objective symptoms by examining the patient's body with fingers, eyes, and ears. Until the time of Auenbrugger, Corvisart and Laennec, he could do little more than feel the pulse and look at the tongue. Then came the discoveries of percussion and of auscultation, which gave to the diagnosis of diseases a precision that no one before had dreamt of. It was natural that those who introduced the new methods should endeavour to mark their value by giving a special name to the indications afforded by them. And thus it became usual to speak of "physical signs" as opposed to ordinary "symptoms." In reality, however, the distinction is untenable. As Dr. Reynolds remarks, heat of skin, an eruption on its surface, the wasting of a muscle, a change in any one of the secretions, are each of them "physical signs," no less than a cardiac murmur or a dull percussion-note. Equally unimportant is the division of symptoms into "general" and "local," upon which some writers have laid stress. And lastly, it is misleading to pick out a particular symptom as "pathognomonic," or as bearing so close a relation to the disease that, whereas all the rest of the symptoms afford only presumptive indications of its presence, this one proves it absolutely.

Our knowledge of the *causes* of diseases, of their *œtiology*, is, as a rule, exceedingly fragmentary and imperfect. It may fairly be doubted whether in the whole domain of pathology any cause is to be found which invariably and under all circumstances produces a given effect. But in many instances there is little difficulty in perceiving that an illness, of whatever kind, has been preceded by some definite change in the outward circumstances of the patient, or in the action of some vital organ. He may have been chilled by exposure to cold, he may have eaten unwholesome food, he may have been over-excited or over-fatigued. Either of these conditions is then called the "*exciting cause*" of the disease. But, on further consideration, it appears that people often go through weather no less inclement, indulge their appetites in precisely similar ways, have their mental and bodily powers strained to quite as great an extent, without being attacked by the same complaint, and, indeed, without their health being in any degree impaired. We now have no alternative but to assume that some other cause or causes have been in operation. This may act in either of two ways: it may combine

with the exciting cause to produce the disease in the individual who falls ill, or its influence may be exerted upon those who remain well, counteracting and nullifying the operation of the exciting cause. If, for example, it is found that a complaint occurs only in persons at a certain time of life, this may either depend upon that particular age being favorable to its development, or upon other ages being unfavorable. Probably it sometimes happens that the one explanation is correct, sometimes that the other is; or both may apply in the same case. But in practice we generally assume that all causes of this kind have a positive instead of a negative action; and we are therefore accustomed to group them together under the name of "*predisposing causes.*"

It is, however, important to note that the conditions which are commonly included among the predisposing causes of diseases do not always operate in either of the ways just mentioned. Reverting to the case already put, of a complaint affecting chiefly or solely persons at a particular age, we may on consideration find that this is merely due to the fact that those who are at that age are more exposed than others to the action of the exciting cause. The simplest illustration is, perhaps, afforded by the preponderance of certain ages among those who meet with injuries of various kinds. Thus, cutaneous scalds and burns are particularly frequent among young children; and scalds of the throat from drinking hot water out of the mouth of a kettle scarcely occur in persons who are grown up. On the other hand, young adults are much more apt to get their limbs broken than very old men. Such differences arise, not from a predisposing influence exerted by one period of life, nor from a counteracting influence of another period of life, but merely from the ignorance of infants as to the effects of heat, and from the fact that men at their prime are obliged to work under conditions which expose them to accidents. So, again, if general paralysis and locomotor ataxy and many other diseases of the nervous centres are more frequent in males than in females, the reason may be, not that one sex rather than the other predisposes to their occurrence, but that more men than women come under the operation of the exciting causes of these complaints, which are believed to be chiefly overwork, sexual exhaustion, over strain of the mental faculties, and the like. All this is simple enough when we know what the exciting cause of a disease is, but if we are altogether ignorant of it, as is very often the case, our interpretation of the so-called predisposing causes must obviously be in the highest degree speculative and uncertain. Perhaps the most unsatisfactory of them all are those which are commonly included under the term "idiosyncrasy." It is well known, for example, that in certain persons nettlerash is invariably produced by strawberries, and that in other persons the odour of cats excites asthma. In such cases the exciting cause is obvious, and it is impossible to deny the title of predisposing causes to the conditions, whatever they may be, which lead to its operation in a particular individual and not in others. But as to the nature of these conditions we are altogether ignorant.

At first sight one might think it advisable to confine the term "predisposing cause" to those conditions which alone have logical claims to be so designated. Virchow many years ago made an attempt to introduce such a limitation, defining the "predisposition of an organ to disease as dependent upon a certain slight deviation from its normal structure which causes its constituent parts to be too loosely held together." And a similar statement is upheld by Wagner. But a little consideration will show that we shall

scarcely ever be able to speak of predisposing causes at all if we adopt such a definition. For my own part, however, I am so sensible of the inaccuracy of the common use of this term that I shall, as far as possible, avoid making use of it in the present work.

It is in reference to the prevention of diseases that a knowledge of their ætiology is primarily of practical use, but indirectly it not infrequently has an important bearing upon their cure. I have already shown that the process of analysing the clinical phenomena or symptoms, which constitutes the diagnosis of a case, is often arrested at a point short of our being able to trace it to a definite anatomical lesion; and, not infrequently, we are unable even to carry that process far enough to reach any solid basis on which to institute a sound plan of treatment. Every circumstance in regard to possibly exciting or predisposing causes of the patient's illness may then be of the greatest value in helping us to treat it successfully.

But before we presume to attempt to modify the progress of a disease by medicinal or other treatment we have something to do beyond observing its symptoms, searching into its causes, and determining its place in the nosology. We have to ascertain what is its natural course when undisturbed by our art, so that we may know as certainly as possible whether the remedies which we use are really effectual or otherwise. It is this study that enables us to give what is termed a *prognosis* of the case to the patient himself or at least to some near relative, a part of our duty which may be of the highest importance, and in the performance of which our credit and reputation are often at stake. In some cases, indeed, the prognosis is of such a character as almost to do away with the necessity of our rendering any further services to our patient. We may be able to declare that the disease will quickly and safely pass off, without tendency to relapse, and without risk of its leaving any ill effects behind it. A case in point is perhaps afforded by an attack of shingles in a child. Or we may have to say that the complaint is one which must inevitably prove rapidly fatal in spite of every effort that can be made to arrest it. Such a verdict, for instance, must be given in many cases of apoplexy, and in hydrophobia. But, as a rule, our prognosis is applicable, not so much to the natural and undisturbed course of the disorder, as to the course which we believe it to be likely to take under the most judicious treatment that we can devise for the benefit of the patient.

One of the ways in which a disease may end is in *death*. Now, in the mode of dying, and in the phenomena which attend the last moments or hours of life, there are certain varieties which require to be mentioned, and for the discussion of which the present is perhaps the most convenient occasion.

It was an ancient doctrine that different organs might in different cases be the *atria mortis*, "the vestibules or portals of death." But in modern times Bichat first endeavoured to distinguish three modes of dying, at least so far as sudden death is concerned, which begin respectively by interruption of the circulation, of the respiration, or of the action of the brain. His views were based upon the contrast which he drew between the "organic life" and the "animal life;" the heart and the lungs were regarded by him as bearing the same relation to the former as the brain to the latter. Now, it has long been recognised that the third of Bichat's modes of death—at least in the way in which he understood it—may be resolved into the other

two. And yet, although writers point out that apoplectic and other comatose patients often die by "asphyxia," and perhaps sometimes by "syncope," they still continue to include *coma* among the modes of dying. But it seems to me that the logical way of dealing with the question is to describe as forms of death only those conditions which lead directly to the extinction of life, and which are themselves incapable of further analysis. Now, during sleep, and under other circumstances as well, the movements of the chest walls and those of the heart constitute the two great signs of life, all obvious indications of cerebral activity being in abeyance. We may therefore, I think, reasonably suppose that if there are any fundamental differences in the phenomena which usher in death, the key to them should be found in the different order in which these two sets of movements successively cease. But, in Dr Michael Foster's 'Text-book of Physiology,' I find that little or no encouragement is given to the notion that any such distinctions can be drawn. Of the "respiratory centre" in the medulla oblongata, upon which the maintenance of the respiration depends, he says that "when this spot is excised or injured, breathing at once ceases, and since the inhibitory vagus centre is generally at the same time stimulated, and the heart's beat arrested, death ensues almost instantaneously. Hence this portion of the nervous system was styled by Flourens the 'vital knot' or ganglion of life—*nœud vital*." Dr Foster's statement appears to be derived from the results of experiments on animals rather than from observations upon the human subject. But there can be no doubt that an equally complete and sudden extinction of all the vital actions occurs—in persons who are killed by lightning, and in at least some of those who are shot dead, or who succumb to other violent and extensive injuries. Again, I am quite disposed to believe that in many cases in which the action of the heart is suddenly arrested, as, for example, where there is disease of that organ, or of the aorta, the respiratory centre is at the same instant paralysed, so that both sets of movements cease together. In all probability the same thing occurs in some of those very rare instances in which a large quantity of blood effused into the interior of the cranial cavity destroys life with almost absolute suddenness.

But, whatever may be the case in the physiological laboratory, it is certain that in the wards of a hospital some cases occur in which the respiration stops while the heart is still beating; while there are others in which the cardiac pulsations are arrested before the thoracic muscles and the diaphragm cease to draw air into the lungs. Thus we can clearly recognise two principal modes of dying which are fundamentally distinct from one another. Each of the vital movements in question, however, is dependent upon the influence of nervous ganglia; the respiratory (as we have already seen), upon that of a centre in the medulla oblongata; the circulatory, upon that of a series of minute ganglia situated at the base of the heart. Thus, it is obviously possible for death by arrest of the breathing to be directly due to a morbid condition of the respiratory centre just referred to. At first sight, we seem now to be reintroducing Bichat's third form of death—that by interruption of the action of the brain. But a little consideration will show that this is not really the case. For the conditions under which a *sudden* paralysis of the respiratory centre occurs are not those which cause cessation of the animal before the organic life, as Bichat supposed; nor does such paralysis in fact appear to bear a much closer relation to diseases of the brain than to those of the lungs and air-passages. A *gradual* exhaustion of the respiratory centre seems to be a necessary part of the process by which life is extin-

guished in all other cases in which the breathing ceases before the heart stops.

I. *Dying by the lungs.*—This may, as I have just hinted, occur in two different ways; one in which the centre in the medulla oblongata is suddenly paralysed, and another in which its vital activity becomes gradually, though perhaps rapidly, exhausted. This difference appears to me to be a most important one, since there is a complete contrast in the attendant phenomena.

(i) Death by stoppage of the breathing, dependent upon *sudden paralysis of the respiratory centre*, is often absolutely quiet. No doubt, if the person were sitting or standing up he would fall, and this would attract notice to the change in him. But, when lying in bed, he may draw his last breath without making the slightest noise, or moving a limb; and this, notwithstanding that he was awake and perfectly conscious a few minutes before.

On two occasions, in visiting patients in consultation, I have found them in the act of dying in this way when I first entered the room. In neither case had the relatives the slightest idea that anything was amiss, although they were standing by the side of the bed or at its foot. What at once arrested our attention was, in each instance, the exceedingly flickering and feeble state of the pulse; and a careful scrutiny soon showed us that there were no respiratory movements. One circumstance which seems very remarkable, and the explanation of which is not apparent to me, is that there was no obvious lividity, or change in colour of the face. We had hardly time to give a warning of what was impending before the pulse became imperceptible, and the beats of the heart very soon afterwards ceased. There was no autopsy in either case; but the accounts which I received of the previous symptoms seemed to leave little doubt as to their nature. The first patient was the wife of a tradesman who had been suffering for a considerable time from severe pain in the head, and from other symptoms of cerebral tumour; she had been morose, and unwilling to be disturbed, so that her taking no notice of the visit of a stranger did not appear surprising. The other patient, an old lady, had been ill with bronchitis for some weeks, but not so as to excite much anxiety; she was supposed to be asleep, having said that she would like to rest for a quarter of an hour before the time appointed for the consultation; but she was absolutely unconscious, and the strongest liquor ammoniæ had not the slightest effect when brought to her nostrils.

I believe that these instances illustrate the two chief conditions under which sudden paralysis of the respiratory centre is met with in medical practice. (1) Within the last few years several cases of tumour, or of abscess of the brain, have terminated, in this way suddenly and quite unexpectedly at Guy's Hospital. I know of at least one instance in which the same thing would have happened during an epileptic fit, had it not been for the diligent maintenance of artificial respiration. And in cerebral hæmorrhage I believe that this mode of death is not infrequent. (2) On the other hand, it seems to occur in various diseases of the lungs and air-passages. Three years ago, one of our students, a strong healthy young man, devoted to football, was admitted into the Clinical Ward labouring under a severe attack of asthma, to which complaint he was subject. A few hours later, the house physician, who was sleeping in an adjoining room, was hastily summoned to him on account of an alarming failure of the breathing. The respirations became more and more shallow, and at last they ceased altogether; he fell forwards in a state of insensibility, and remained unconscious for several minutes. Artificial respiration was at once resorted to: this was kept up for a little

while, and the patient recovered. I have no doubt that he would have died if no medical man had been at hand. It seems to me likely that when death occurs suddenly from obstruction to the entrance of air into the larynx, it is due, not (as is generally supposed) to syncope, but to paralysis of the respiratory centre. It is well known, for example, that persons choked by the impaction of food in the fauces not infrequently die without any of the symptoms which would ordinarily be expected for such an accident; so that no suspicion of it arises until an autopsy is made. I am not sure whether those cases of pleurisy, or of phthisis, in which sudden death takes place should, or should not, be explained in the same way. A chronic disease, especially if it causes much wasting, may fairly be supposed to impair the nutrition of the heart, and in other ways to interfere with the regular maintainance of its movements. The question is one upon which direct observations are needed, as to whether the pulse or the breathing is the first to stop; or whether they both cease together. (3) It is, of course, well known that many poisons kill by paralysing the centre in the medulla oblongata: this is one of the chief ways in which death is apt to occur during the administration of chloroform and other anæsthetics.

(ii) Death by stoppage of the breathing, dependent upon gradual (though often very rapid) exhaustion of the respiratory centre, has long been known under the name of *asphyxia*. Etymologically, indeed, this is incorrect, for the term properly means "pulselessness," whereas (as I have already explained) the heart goes on beating to the very last; but at the present day no misunderstanding can arise from the use of "asphyxia" in the usual sense, which has not only common acceptance, but the authority of many generations of writers, in its favour. Some years ago an attempt was made to substitute for it the name of *apnœa*. This, however, has never been widely adopted, and its employment is now to be discouraged as much as possible, since physiologists employ it to denote an exactly opposite condition, that which consists in an excessive supply of oxygen to the blood, whereby respiratory movements are for the time rendered unnecessary. We may, therefore, continue to describe as asphyxia the state which generally arises when a person is strangled, or smothered, or drowned, or when he is choked by a solid substance impacted in his throat. The primary effect of such an accident is that the face and other parts of the surface assume a livid or purple colour in consequence of the altered condition of the blood in the systemic arteries, which becomes far darker and contains much more carbonic acid than the venous blood of health. The respiratory movements at once become more forcible and more frequent. Dr Michael Foster sets forth how in experiments on animals it is very soon observed that the expiratory efforts are exaggerated out of all proportion to the inspiratory, and how they quickly pass into violent convulsions. During this period (which in the rabbit occupies a little more than a minute), the blood-pressure in the arteries rises very greatly and the cardiac pulsations are increased in frequency. At the end of it the breathing suddenly becomes deep and slow, the inspiratory movements being now the more marked of the two. The arterial pressure rapidly falls again, and the beats of the heart are reduced in number, although they still remain forcible. Gradually the rhythm of the respiratory movements ceases to be regular; long pauses occur, each separated by a series of quickly following inspirations, and each in its turn seeming as though it would be final. The inspirations themselves become more and more shallow, they have a gasping character, being accompanied by contrac-

tions of accessory muscles, especially of the face; at length the gasps spread into a convulsive stretching of the whole body; and with extended limbs, and a straightened trunk, with the head thrown back, the mouth widely open, the face drawn, and the nostrils dilated, the last breath is taken in. In rapid asphyxia all this occurs within four or five minutes. The heart continues to beat for some seconds longer, its strokes at last rapidly failing in frequency and strength.

In experiments on animals, in which it is possible to determine the state of the heart's chambers at the moment of death, they are seen to be all gorged with blood, the left as well as the right. If in ordinary autopsies in the human subject the left auricle and ventricle are found comparatively empty, while the right are distended, it is because the former unload themselves during the setting-in of rigor mortis.

To Dr Foster's account of the phenomena observed during life under such circumstances—which I have followed almost word for word—I may add the fact (in clinical practice a very important one) that when there is an actual obstruction to the entrance of air into the lungs the more yielding parts of the chest walls recede at every inspiration; thus the suprasternal, supra-clavicular, and lower intercostal spaces, the præcardial region, even the inferior part of the sternum, and the lower ribs, may appear to be violently sucked inwards. And after death the pleura may be found spotted with ecchymoses, which appear to result from the corresponding traction upon the subserous capillaries.

But asphyxia is by no means limited to cases in which a mechanical obstruction exists; it occurs in a scarcely less typical form when an animal or a human being is kept in a limited area into which no fresh air can enter, as, for instance, in the Black Hole of Calcutta. And a more or less chronic variety of asphyxia is seen in croup and in diseases of the larynx and trachea, as well as in bronchitis and other affections of the lungs, in which the air-passages become blocked by accumulated secretions or by extravasated blood. The lividity of the skin and mucous membranes, and the recession of the yielding parts of the chest walls may be well marked in such cases. Convulsions are perhaps less commonly observed; but death ultimately occurs in precisely the same way as above described, with a gradually increasing shallowness of the inspiratory movements. Again, in the case of apoplexy and of other cerebral diseases, asphyxia is often obviously the cause of death; fluid accumulates in the bronchial tubes, and at every breath the air bubbles through it. But under such circumstances, we can seldom be sure that a gradual paralysis of the respiratory centre (from the direct pressure of intracranial effusion) may not help in bringing about the cessation of the breathing, even if it be not the sole cause of it.

The timely performance of artificial respiration is sometimes effectual in saving the lives of persons in the act of dying by either of the two forms of stoppage of the breathing. Nor is it difficult to see what conditions are generally necessary to the success of this procedure. In cases of sudden paralysis of the respiratory centre, little can be expected from it, when the cause of the paralysis is an intracranial lesion. I think, however, that I remember one instance in which a patient with a cerebral tumour was in this way snatched from the grave for a time, although only to succumb to a recurrence of the same symptoms a few days later. Where the breathing is suddenly arrested in the course of an epileptic fit, or of an asthmatic seizure, artificial respiration appears to offer a good chance, as is shown by the case

which I have narrated. And during the administration of chloroform it has often been as successful as could possibly be desired. In asphyxia, on the other hand, artificial respiration is useless unless the cause of the asphyxia can first be removed. If a man has been hanged or strangled, the throat must first be relieved from pressure ; if he has been choked, one must first clear out the fauces and the entrance of the larynx, or open the trachea, so as to admit the entrance of air. If he has been drowned, the fluids in the bronchial tubes must first be allowed to drain away. Thus in bronchitis and in other thoracic diseases, in which asphyxia gradually advances to a fatal termination, artificial respiration is seldom of any use.

Various methods of performing artificial respiration have been recommended by different observers. They all consist in the repetition, at regular intervals, about sixteen times in the minute, of alternate movements, by one of which air is driven out of the chest, while by the other it is sucked in.

The oldest plan of all is simply to compress the thoracic walls and then to relax the pressure, the patient lying on his back. In 1856 Dr Marshall Hall introduced what he termed the "postural" method. He first placed the body face downwards, when its weight caused flattening of the chest and abdomen, and therefore expiration ; afterwards he turned it over upon its side, when the elasticity of the ribs caused them to rise, and so led to a slight inspiration. One supposed advantage of the prone position was that the tongue fell forward against the teeth rather than backwards over the entrance of the larynx ; but as this was at the time when air was escaping from the air-passages, and not when it was entering, the advantage was probably small. A drawback obviously was that only one side of the chest was likely to expand. Dr Marshall Hall's method has therefore been generally abandoned in favour of one which was suggested by Dr Henry Silvester in 1857.

According to his plan the patient is placed on his back with the shoulders raised and supported on a folded article of dress. The feet are fastened down or held by an assistant. The mouth and nostrils are then wiped if necessary. The tongue is drawn forwards, and kept projecting between the lips. One way of doing this is gently to raise the lower jaw so that the teeth hold the tongue forwards. The operator now kneels at the patient's head, grasps his arms just above the elbows, and carries them upwards and a little forwards until they nearly meet, holding them there for two seconds ; this action enlarges the chest, the ribs being drawn up by the pectoral and other muscles. Next he turns down the arms and presses them gently but firmly against the sides of the body. This diminishes the capacity of the chest, especially if the operator's hands are made to exert further pressure upon it. The Royal Humane Society soon began to recommend Dr Silvester's plan, and it has since been generally adopted.

But in 1877 Dr Howard, of New York, proposed a third method, an account of which may be found in the 'Lancet' for that year. His first step is to tear off the patient's clothing to the waist, and make of it a large solid bolster. He next turns the body face downwards so that this bolster comes under the epigastrium. He then presses heavily upon the back for a few seconds, two or three times, so as to empty, as far as possible, both the lungs and the stomach. Next, the body is replaced on its back, the bolster beneath it making the epigastrium and the costal cartilages the highest points, while the shoulders and the occiput rest upon the ground at a lower level. The patient's wrists are now seized, crossed behind the head, and fixed there. His tongue is drawn forwards with the thumb and forefinger (protected by the corner of

a handkerchief), and it is held out of the right corner of the mouth. This part of the procedure may be confided to an assistant. The operator himself kneels astride the patient's hips. He places his two hands one on each side of the chest, the thumbs close to the xiphoid cartilage, the fingers over the lower intercostal spaces. He then slowly and steadily throws his whole weight forwards until his face nearly touches that of the patient, and afterwards, by a final push, springs suddenly back into an erect but still kneeling posture. This procedure he repeats eight or ten times a minute. A point which Dr Howard claims for his "direct method" (as he terms it) is that the entrance of the larynx is kept open by the backward curvature of the patient's neck. There seems to be no doubt that it is far less fatiguing to the operator than Dr Silvester's plan, and this is a very important consideration, inasmuch as artificial respiration has often to be persevered with for three or four hours at a time. It is surprising what a long interval will elapse, even in cases which recover, before a single spontaneous respiratory effort is discernible. Of course, where any such effort, however slight, is perceived great care should be taken not to let the artificial movements clash with it.

Whatever mode of artificial respiration may be adopted there is no reason why it should not be supplemented by the employment of warmth and friction to the limbs. The nostrils may also be excited by ammonia, and cold water may be dashed upon the face at intervals; but in cases that are really critical very little is to be hoped for from such means.

If a Faradic apparatus be at hand it may often be used with advantage. One mode of applying it is simply to stimulate the cutaneous nerves, placing, for example, one pole inside the cheek and another over the xiphoid cartilage, and allowing the current to pass without regard to the rhythm of the respiratory movements. I suppose, however, that this would probably fail if the respiratory centre were exhausted. Even then success may sometimes be attained by stimulating the phrenic nerves directly. The most exact method of doing this will be described elsewhere. But, for the purpose now in view, it is sufficient to use a large wetted sponge for each pole, and to put one on each side of the neck just outside the sterno-mastoid muscle. This brings some of the shoulder-muscles into play as well as the diaphragm, but this is no disadvantage. The essential thing is to stop and to reapply the current at regular intervals; so that each contraction may be followed by a pause, during which an assistant may compress the chest and the abdomen, so as to drive as much air as possible out of the lungs.

II. *Death by failure of the heart's action* is attended with different symptoms in different cases, according to the circumstances in which it occurs.

By some writers, including no less an authority than Sir Thomas Watson, the term *syncope* is employed as a general appellation for all forms of death beginning at the heart. And probably this is the best use to which the word can be put, though many instances might be found of its being taken with a narrower meaning, one nearly equivalent to fainting, of which an essential feature is stoppage of the organ in diastole.

(1) In some cases death by the heart is absolutely *sudden*, the patient falling flat upon the ground or sinking back in his chair. The respiration may perhaps cease at the same instant as the beats of the heart, or a few deep gasps occur, and then a kind of shiver runs through the frame, pallor spreads over the surface, and all is over. It is often impossible in such

cases to determine whether paralysis of the heart or spasm is the cause of the cessation of its movements. If an autopsy is made, its chambers may be found either relaxed or contracted, either empty or more or less full of blood. But it is difficult to say how far its state may be modified by post-mortem contraction of the ventricular walls. If they are flabby, while the muscles generally are in a state of rigor mortis, it seems fair to conclude that at the time when life became extinct the heart stopped in a condition of diastole. On the other hand, stoppage in systole is not necessarily due to spasm; it may be the result of pressure upon the outside of the heart, as when the pericardial sac becomes filled with blood by rupture of the aorta. An appearance which is, so far as I know, never seen when death begins at the heart, is an overloading of the right cavities with blood, while the left are empty, as in cases of asphyxia.

The causes of sudden cessation of the cardiac movements are various. There may be disease of the heart itself, or of its valves, or of the base of the aorta; and in many cases of this kind it is probable that lesions of the nervous ganglia situated near the base of the organ might be discovered if carefully sought for. Or the functions of the heart may be arrested by affections of distant parts of the body. Upon such cases a flood of light has been thrown by modern physiological researches. It has been found that in animals stimulation of one of the *nervi vagi*, if sufficiently powerful, almost instantly arrests the cardiac beats; a similar effect follows irritation of a spot in the medulla oblongata which, therefore, is called the "cardio-inhibitory centre;" and, lastly, it can be produced as a reflex effect by a violent impression upon peripheral nerves (as by suddenly crushing the foot), by comparatively slight irritation of the mesenteric nerves (in the frog, by sharply striking the exposed intestines with the handle of a scalpel), or even by gentle stimulation of them when peritonitis has first been set up.

In human pathology, all these varieties of inhibition seem to occur more or less frequently. That of which in medical practice we know least is perhaps direct inhibition by stimulation of the vagus. Czermak, however, was able at will to stop the beating of his own heart, by pressing the trunk of the pneumogastric nerve against a small osseous tumour in the neck. In cases of aneurysm of the aorta it is not uncommon for sudden death to occur without the autopsy revealing a rupture of the sac or any other definite change; in such cases the fibres of the left vagus nerve are commonly spread out over and inseparably blended with the walls of the aneurysm; and it seems very likely that this ought to be regarded as the cause of the arrest of the cardiac pulsations. Inhibition of the heart by stimulation of the centre in the medulla oblongata probably occurs when fainting is caused by emotion. Whether intracranial diseases ever destroy life in this way is perhaps doubtful; at least I am not aware of any observations in which it has been shown that the heart's beats ceased before the respiratory movements. On the other hand, death by reflex inhibition appears to be of frequent occurrence. There can be little doubt that the "fatal shock" which accompanies severe injuries of the limbs or of any part of the body is of this nature. But what is perhaps most striking is that abdominal diseases are specially apt to be attended with sudden death, just as we have seen that in experiments on animals the cardiac beats are most easily arrested by impressions on the mesenteric nerves. It is not necessary to appeal to the oft-cited cases in which persons have been killed instantaneously by blows on the epigastrium. In the collapse of acute peritonitis

life is often suddenly extinguished, as I shall hereafter have occasion to point out. Whether diseases of the lung ever produce sudden death in the same manner I do not know.

In other cases death by failure of the heart's action is more *gradual;* minutes or even hours pass before life becomes finally extinct. The symptoms may then vary according to circumstances, so that two or three different forms require description.

(2) Sometimes the fatal termination occurs by fainting, for which the ancient designations were *deliquium animi* and *lipothymia.* The patient experiences distressing sensations of giddiness, nausea, and sinking at the epigastrium. He turns cold and pale, and perhaps breaks out in a clammy sweat. His sight becomes dim, and everything may appear black to him. He hears rushing noises in the ears. More or less rapidly he becomes completely insensible. His pulse is rapid and very weak; and it soon ceases to be felt at the wrist, though it may still for a time be counted in the carotids. The heart's impulse grows more and more feeble, until it may be no longer perceptible. Still, however, one may be able to hear with the stethoscope that the organ continues to beat, though the sounds are very feeble, and the second one is audible only at the base. The respiration, in the meantime, becomes infrequent, irregular, and shallow. The pupils are dilated. Sometimes there is an involuntary discharge of urine and of fæces. When the heart's beats cease altogether, if may be considered that death has actually occurred. At the autopsy its walls are found relaxed, and its chambers may be more or less full of blood.

(3) Somewhat different in its phenomena is that form of death which is due to *hæmorrhage.* There is then a waxy pallor of the face and lips, of the hands and finger-nails, and, indeed, of the surface of the body generally, which is more extreme than that accompanying mere fainting. Consciousness is perhaps more gradually lost, though much depends upon the rapidity of the bleeding. Delirium is often present, and in many cases epileptiform convulsions are developed. Sometimes the patient lapses from time to time into a state of insensibility, regaining consciousness in the intervals: it is then generally said that "fainting fits" occur. They are especially apt to arise if the patient attempts to sit up, and at the same time the pulse at the wrist becomes much more feeble than before, or is even temporarily imperceptible. After death from hæmorrhage the heart is found contracted and empty.

The physician sees this form of death in cases of hæmatemesis or hæmoptysis, or ruptured aneurysm. Sometimes when the giving way of a large vessel is the direct cause of the cessation of the heart's action, there is no external bleeding at all; the blood is extravasated into some large space within the body, such as the peritoneal cavity, or one of the pleural sacs, or even the alimentary canal. But I am not sure that these can be cited as examples of death by hæmorrhage, for the symptoms seem to be seldom altogether typical.

(4) In marked contrast with the forms of death hitherto described, although also beginning with failure of the heart, is that which is called a state of *collapse.* This form, like the others, is attended with an extreme feebleness of the heart's action, the pulse at the wrist becoming imperceptible, and the cardiac sounds being heard with difficulty even through the stethoscope. The surface of the body, and especially of the hands and feet, becomes cold and deathlike. The features are sunken, the eyes

retracted in their sockets, and the orbits surrounded by deep brown rings. Yet the patient is often entirely free from all subjective sensations of giddiness, nausea, or faintness. He may still have a fair amount of muscular power, may get out of bed, and even walk for some distance. His consciousness is often retained up to the last moment; death, when it does occur, being absolutely sudden.

I am not aware that any satisfactory explanation has been given of the remarkable differences between the two conditions of fainting and collapse. The retention of the functions of the brain and even of the muscles in the latter state would seem to imply that these organs continue to receive a certain amount of arterial blood. Is it possible that the feeble stream which still flows from the heart, until its beats finally cease, is mainly directed to the deeper parts, by an adjustment of the vaso-motor apparatus, while the flow through the various superficial arteries is more or less cut off?

Either fainting or collapse may result from affections of the abdominal organs, and are then probably due to reflex inhibition from the solar plexus, as in the cases to which I have already alluded, of sudden death from blows upon the epigastrium. But either of them may also be due to other causes; collapse, for example, occurs in certain cases of diphtheria, and it may also be produced by pulmonary embolism. If the analogy with the inhibitory phenomena observed in the physiological laboratory can be looked upon as tolerably complete, it is an interesting question whether in any circumstances the supervention of collapse or of fainting can be prevented by the injection of atropine, which in animals completely annuls the normal inhibitory action of the vagus.

Treatment.—In many cases of death by cardiac failure life is extinct before aid can reach the patient. But sometimes the administration of a full dose of brandy or of ether by the mouth is effectual in arresting the danger; and the hypodermic injection of these remedies, when the syringe is at hand, is still more likely to be successful. Our house physicians frequently adopt this practice, and in many cases with marked success in stimulating the action of the heart, although the result is too often temporary. Another method, advanced by Dr J. C. Reid ('Brit. Med. Journ.,' 1880, vol. ii, p. 1014), is that of pouring hot, but not scalding, water over the præcordial region. He relates the case of an old man of seventy, who was thus restored from a condition of apparent death, and who lived for many years afterwards. It does not appear very clear whether galvanism can be used with advantage in such circumstances. Ziemssen recently found ('Deutsch. Arch.,' xxx, 1881) in a patient whose chest wall was deficient so that the heart was covered only by the skin, that the heart's beats could be accelerated by powerful currents. Erb recommends that in galvanizing the heart large electrodes should be used, one being applied over the surface of the organ, the other over the dorsal vertebræ; a current of the highest intensity should then be passed, its direction being reversed seventy or eighty times in the minute. Previous observers, as cited by Walshe, had found that with strong currents there was a risk of inhibiting the cardiac contractions instead of stimulating them. And although it was admitted that, if the irritability of the organ were lowered, a powerful current, which, under ordinary circumstances, might have arrested its action, would perhaps have the effect of reviving it, there yet seems to be need of caution; so that it would be advisable first to make trial of a feeble current

and afterwards to increase its strength. But in the cases now under consideration there is seldom time for such a method of proceeding.

III. If now we turn our attention to the phenomena which accompany the *gradual* fading away of existence under the influence of some wasting disease, and endeavour to analyse them, we find that although this also is commonly spoken of as death by *asthenia*, we can hardly identify satisfactorily the characters of that mode of dying, any more than we can do so in a person who succumbs to mere old age. It is to be observed, however, that very few cases occur in which such diseases really run on to what appears to be their natural termination. If we take, for example, a series of cases of cancer of the œsophagus, or of any non-vital organ, and if we critically investigate them, we find that in a very large majority of them it is some accidental complication which actually destroys life. Still there are some few exceptions to this rule, and in these exceptions all the vital functions are brought to so low an ebb before any one of them is absolutely arrested, that to determine the order in which their ultimate extinction takes place is superfluous, even if it be not impossible.

Slowly, and week by week, the sick man becomes more and more indifferent to those about him. His memory, his power of attention, all his mental faculties, gradually fail him. He passes days and nights alike in a dreamy doze or stupor. His special senses one by one disappear; smell and taste seem to go first; then he loses his sight so that everything appears dark to him; last of all he becomes unable to hear. The sensibility of the skin is often greatly diminished; flies crawl unnoticed over the face and hands, so that these parts must be covered with gauze; and the conjunctivæ may lose their sensibility to impressions to such an extent that the dull lustreless corneæ become dry between the motionless, half-opened eyelids. The pupils are generally contracted, but before death they are said to become dilated. The eyes can no longer be brought to bear upon an object, but remain fixed, with their axes parallel. The muscular weakness is extreme; the body sinks down in the bed, the head cannot be raised from the pillow, the limbs lie powerless in any position into which they may happen to fall. The organic functions are reduced to the lowest possible point. Wagner, to whom I am indebted for several of the details above mentioned, speaks of fluids as falling down into the stomach through the paralysed œsophagus with a splashing noise; but for many days before death it often happens that nothing whatever is swallowed, only from time to time a feather moistened with a little weak brandy-and-water is introduced a little way between the lips. The evacuations, both of urine and fæces, are passed into the sheets without exciting any consciousness. The temperature of the body usually falls, until it reaches several degrees below normal, the hands and the feet become obviously cold, sometimes, when the stupor is not very marked and the advance of death not very slow, the patient himself feels the cold creeping up from his limbs to his trunk. The respiration becomes infrequent, irregular, and shallow. Towards the last the movements of the chest walls may be so slight that it is scarcely possible to perceive them; one way of ascertaining whether they still go on is to hold before the mouth a looking-glass, the cold surface of which becomes dimmed by the moisture of the breath.*

* . . . . Lend me a looking-glass;
If that her breath will mist or stain the stone,
Why, then she lives.
KING LEAR, v, 3.

Sometimes the expiratory efforts are deep and sighing, and may be separated from one another by intervals of a minute or even longer, so that those who are watching by the bedside expect each breath in turn to be the last. Long before this, it has become impossible to detect the pulse at the wrist, which for many days has been small and rapid and flickering. At length, even with the stethoscope, no sign of the beating of the heart can be discovered, and then, often not without some hesitation, one pronounces the verdict that all is over.

In most cases there is no difficulty in determining the exact moment at which death occurs. But sometimes it cannot be fixed with certainty, and there are some altogether exceptional instances (though I have never myself met with one) in which for hours, or even for days, it remains uncertain whether life is extinct or merely suspended. I believe that the only sign of death which is both certain to manifest itself in the course of a few days, and also absolutely conclusive and infallible, is the occurrence of putrefaction, which is generally first indicated by discolouration of the surface of the abdomen. And in any case admitting of doubt, the coffin should not be closed until this has shown itself. But rigor mortis, chilling of the body, the cadaveric odour, the loss of transparency or flattening of the cornea, livid staining of the dependent parts of the body—each of these signs, when present and when developed in the usual way after suspension of all obvious vital phenomena, may be taken as proofs that life is really at an end. I do not think that there is any foundation for the strong fear which many persons entertain of being buried alive, after supposed death. The cases really requiring caution are some very few instances of persons found in the streets, or losing consciousness unexpectedly and in unusual circumstances. Dr Walshe says that the only serious difficulty lies in diagnosing what he terms "prolonged syncopal trance," but of this condition he gives no detailed account.

CONTAGION

General pathological processes—Specific diseases—Contagion and miasm—Theory of infection—Contagium vivum—Microzymes—Their origin and life—Their exclusive action in producing specific diseases—Immunity from contagion—Protective inoculation—Modes of transference of miasmata and contagia—Theory and practice of disinfection.

We have still an imperfect knowledge of the nature of many of the general morbid changes to which the human body is liable; and no doubt this explains the fact that the current classifications of the different forms of Hypertrophy, Atrophy, and Degeneration are unsatisfactory, and to a great extent artificial. Since most of these affections are limited to particular structures, they will be best discussed hereafter, when we come to them in dealing with the diseases of the several organs.

But there are other pathological processes, which are met with in almost every organ, and which form the basis of the whole science of pathology. Such are Inflammation, Tuberculosis, and certain kinds of New Growth. To these I must necessarily devote preliminary chapters; but it will be convenient to deal first with the closely-allied subjects of Contagion and Pyrexia.

Specific diseases.—Probably there is no medical man who does not from time to time apply the epithet *specific* to certain diseases, yet, as is the case with so many other terms that are habitually employed, there is no little difficulty in defining its meaning, and in determining what diseases should be classed as specific, and on what grounds. Most writers seem to be of opinion that their characters and their course differ from those of diseases which, by way of contrast, may be called "simple." Thus, Sir James Paget lays stress on the statement that "each specific disease constantly observes a certain phase or construction in its morbid processes." According to Liebermeister, the fundamental point is that among specific diseases the affection produced by a morbific cause is always of a particular kind. He instances the fact that a chill may in one person give rise to a common cold, in another to a bronchial catarrh, in a third to diarrhœa, in a fourth to toothache, and in yet other persons to a variety of "rheumatic" complaints; whereas, he says, the poison of syphilis produces only syphilis, that of measles only measles, that of smallpox only smallpox, and so forth. But I think that we shall hereafter find that there is no disease whatever of which the phenomena do not in certain cases vary so widely as to compel us to base our belief in their "specific" nature entirely upon our knowledge of their ætiology. And it seems to me that the only logical way is to frame our conceptions in accordance with this fact, unless, indeed, we give up the use of the epithet altogether.

I would therefore propose to define as "specific," *those diseases which have a definite cause, arising from without but acting within the body, distinct from the causes of all other diseases.*

The clause "acting within the body" is introduced for the purpose of excluding wounds and injuries and external cold, which set up "simple" or

"common" inflammation. It may be also taken as excluding injuries inflicted upon the mucous surface of the alimentary tract, whether by caustic poisons or intestinal worms, since this is for physiological purposes outside the body. But lead-poisoning and trichiniasis should certainly be regarded as specific diseases, and so, I think, should the affections caused by animal or vegetable parasites which penetrate into the substance of the skin, such as the *Sarcoptes scabiei* and the fungus of ringworm.

The difference of opinion as to the infective character of tubercular affections involves the question of their being specific or otherwise. Malignant and other tumours are, I think, certainly not specific. In reference to them and to some other diseases, a useful test of specificity is that of the existence of transitional forms, connecting one affection with another. Between simple morbid processes transitional forms are very frequent; in the case of specific diseases I do not think that they ever occur. It is true that Dr Braxton Hicks brought before the Obstetrical Society, in 1870, a large series of observations showing that when parturient women are exposed to the poison of scarlet fever the resulting disease is often not attended with either rash or obvious sore-throat, and has rather the characters of a common puerperal septicæmia. But, as he himself pointed out, it may be that the scarlatinal virus disturbs the processes which go on after parturition in the bruised and ecchymosed uterus, and causes local inflammatory changes, and therefore that these, rather than the scarlatinal virus itself, give origin to the septicæmia. This view has since been powerfully supported by Mr Howse in the 'Guy's Hospital Reports' for 1879; he finds that when scarlatina affects a surgical case after operation, septicæmic symptoms are not developed if antiseptic treatment is being carried out, and if the occurrence of putre faction in the wound is prevented.

I have defined the causes of specific diseases as arising from without although they act within the body. Now, there are two (or perhaps three) groups of specific diseases, of which the ætiological relations require to be carefully studied. They are as follows:

(1) *Miasmatic diseases.*—These are caused by a morbific agency which is derived from the soil, or from the water, or from the air of a place, altogether independently of the occurrence of illness of a similar kind in any other individual. Ague is the chief of them, but another miasmatic disease is Influenza.

(2) *Contagious diseases.*—Each of these maladies owes its origin in most cases, if not in all, to a *virus* derived from a person already suffering from it, or sometimes from one of the lower animals. Among these are the exanthemata, Typhus, the Plague, Mumps, Whooping-cough, and Glanders.

(3) *Miasmatic-contagious diseases.*—This group, which is acknowledged by all German observers, is little, if at all, recognised in England. The epithet "miasmatic-contagious" was, I believe, first proposed by Henle in 1840. He, however, only had in his mind the circumstance that smallpox, scarlet fever, typhus and other maladies often seem at first to break out epidemically, as though they might have been caused by a miasma, whereas afterwards the individual cases afford clear evidence of contagion. The question whether the exanthemata, or any other contagious diseases, do, as a matter of fact, ever arise independently of previous cases, is one with which I shall hereafter have to deal. But Liebermeister and other recent writers have assigned quite a fresh meaning to the term "miasmatic-contagious," and have limited its application to a special class of maladies. The typical examples are Enteric

fever and Cholera, diseases which are undoubtedly spread by human intercourse, and which are usually, if not always, traceable to previous cases, but which are nevertheless believed not to be directly communicable from one sick person to another. Liebermeister's conception with regard to them is briefly, that patients give off a contagion which is inoperative until it has passed through further stages of development outside the human body, and that these changes may be taken as converting it into a kind of *miasm*.

It is often convenient to speak of contagious and miasmatic-contagious diseases under the common name of *infective diseases*, from which it is understood that miasmatic diseases are excluded, and also those other specific diseases which are due to an ordinary animal or vegetable parasite, or to a mineral or vegetable poison. And the process by which contagia act may be designated "infection" with sufficient accuracy for all practical purposes, although this term is also applied to inflammation and to cancer.

Theory of infection.—Within the last few years the subject of contagion in general has been profoundly studied. Contagia often appear as fluids; this is the case, for example, with the vaccine lymph, with the contents of smallpox vesicles or pustules, with the nasal discharge of measles, and with the faucial secretion of diphtheria. Chauveau in France, and Burdon Sanderson in England, have been at great pains to demonstrate the fact that the activity of such fluids is not inherent in themselves, and belongs, not to matters dissolved, but to very minute particles suspended in them. They have shown, for example, that when vaccine lymph is allowed to diffuse into distilled water, the diffused liquid is incapable of conveying cow-pox by inoculation. It would almost seem, however, as if the same conclusions might have been safely deduced from the circumstance that most kinds of contagia are capable of being conveyed by the air. For a liquid, or a dissolved solid, must under such circumstances pass into the state of vapour, and must rapidly undergo attenuation, so as to become inert; while, during the brief interval in which it could be supposed to retain its infective power, it would be likely to affect a number of persons indiscriminately, instead of attacking only a few, and those capriciously, as is actually the case.

That contagia are "particulate" may therefore be taken as a fact beyond question. The next point is as to the nature of the particles. For a long time the dominant view was that they were inorganic, or at least devoid of life, that their properties were essentially chemical, and that they acted "catalytically" or after the manner of *ferments*. On the ground of this analogy a new epithet—"zymotic"—was coined, and it is still sometimes applied to the whole class of infective diseases.

But of late evidence has been accumulating from various quarters, which almost, if not quite, amounts to proof, that the contagia are living organisms of exceedingly minute size. There is, in the first place, the fact that in at least two infective diseases—Relapsing fever and Anthrax—the *constant presence* of such organisms has been positively demonstrated.* Certain

* To these may be added: Tuberculosis, Lupus and Leprosy; probably also Glanders Erysipelas, Gonorrhœa, Pneumonia, and several forms of Septicæmia in the lower animals. The cases of Cholera, Syphilis, and certain other specific diseases are more doubtful.

The conditions which justify our regarding a given organism as the material cause, the *contagium vivum*, of a given malady may be stated as follows:

(1) The malady must have such distinct and constant features, whether clinical or anatomical, as shall enable it to be identified.

(2) The microphyte must be itself distinguishable from others by its size and shape, its staining properties, but above all by its mode of propagation in "a pure cultivation" (*i. e.*

pathologists believe that they have detected similar bodies in ague, in enteric fever, in measles, in cow-pox, and in sheep-pox; but with regard to the value and the significance of their observations there appears to be more doubt. It is, of course, to be admitted that the mere discovery of organisms in a particular malady does not warrant the conclusion that they are its cause. The real proof that the probable hypothesis is correct, and that the true organism in any particular case has been discovered must be experimental. When I am discussing relapsing fever and anthrax I shall bring forward other reasons in support of that view so far as each of these diseases is concerned.

As to *a priori* probability, the facts which seems to me to tell most strongly are that so many other processes which bear a clear analogy to specific infection are constantly associated with the presence of minute organisms, and that some are proved to be caused by them. This is the case, for example, with several varieties of fermentation. Lister, in a most interesting paper read before the Pathological Society in 1877, established, I think conclusively, that the lactic acid fermentation or souring of milk is essentially dependent upon what he termed the *Bacterium lactis;* and Pasteur had previously shown that the alcoholic and the butyric fermentations have a similar origin. So, again, that putrefaction is set up by organisms now is certain; the success of Lister's method of treating wounds is nothing less than a demonstration that the discharges from them remain sweet so long as the organisms which induce decomposition can be kept at a distance.

The organisms concerned in these various processes differ remarkably in size, and therefore in the readiness with which the microscope reveals them. The *Torula cerevisiæ*, or yeast plant, which brings about the alcoholic fermentation, is made up of rounded or oval cells, with granules in their interior, which cells develop from one another by budding, and have a diameter of $\frac{3}{10000}$th or $\frac{4}{10000}$th of an inch. The *Bacterium lactis*, which

artificially separated from other organisms) and by the form and colour of the colonies it produces.

(3) The organism thus identified must occur in the blood or tissues—not merely on the surface, cutaneous or intestinal, but below the epithelium, in the lymph-spaces or blood-vessels—in every case of the disease in question.

(4) It must not occur in the human body except in cases of the particular disease in question. It may, however, conceivably occur in other animals without giving rise to the same pathological symptoms, or in air or water, as of course it does occur in the test-tubes and plates of "pure cultivations," and in whatever media convey the contagion from one person to another.

(5) When a pure cultivation of the organism is introduced into the blood and tissues of an animal, the phenomena, clinical and anatomical, of the disease in question must be reproduced.

These conditions in all their rigor have been satisfied in the case of Anthrax, and with scarcely less abundant proof in that of Relapsing fever.

The micrococcus of Erysipelas has also been established as the *contagium vivum* of that disease by good evidence.

In Variola and Vaccinia micrococci are present, but they have not been proved pathogenic by experiment. The same is true of those found in Diphtheria. In Pneumonia several microphytes occur, but not constantly, and which, if any, is the true pathogenic organism is still uncertain. The micrococci of Gonorrhœa and of ulcerative Endocarditis are constantly present, and probably pathogenic, but they are less clearly identified and the proof is incomplete.

The bacilli of Enteric fever, of Ague, and the so-called bacillus of Cholera are still the subjects of controversy as to identity, constancy, and pathogeny. That of Tubercle is constant, specific, and pathogenic, but its relation to much of what is clinically and anatomically tubercle in man is not fully established. Here the first of the above conditions is wanting. —Ed.

causes the souring of milk, consists of oval or rounded bodies, arranged in pairs or sometimes in chains, multiplying by fission, and measuring at the most $\frac{1}{20000}$th of an inch, that is, being no larger than the granules which are contained in the cells of the torula. Consequently, as Lister suggests, there is nothing improbable in the supposition that yet other organisms may exist which may be as much smaller than the *Bacterium lactis* as it is smaller than the *torula*. The characters of the *spirochæta* of relapsing fever, and of the *bacillus* of charbon, will be given when I am describing those diseases; but I may here quote the apt remark of Heydenreich that, were it not for the length of the spiral threads which constitute the former organism, it would in all probability have still escaped detection; rounded or oval bodies not exceeding in diameter the breadth of the spirochæta would be almost, if not quite, invisible with the highest powers of the microscope. In his observations on the traumatic infective diseases, indeed, Koch, by the use of staining liquids, and by a peculiar mode of illumination, has succeeded in demonstrating micrococci which far surpass in minuteness those which had before been recognised. It remains to be seen whether a like success will attend the application of his methods of investigation to the exanthemata and to the other specific fevers.

As the application of so many different names is not a little confusing, it may be convenient that I should append the following classification:

1. Moulds (*Mucorini, Hyphomycetes, Schimmelpilze* of Nägeli). These are long-branched filaments, which form numerous spores. To the pathologist they possess scarcely any interest, but they are sometimes found lining the interior of dry vomicæ in the lungs.

2. Budding fungi (*Saccharomycetes, Sprosspilze* of Nägeli). These consist of rounded or oval cells, which give off buds, and may form beaded threads. I shall have to describe them as causing certain cutaneous affections.

3. Fission-fungi (*Schizomycetes, Spaltpilze* of Nägeli). These are exceedingly small bodies, which multiply by fission, and are often arranged in pairs. They also sometimes cohere into delicate threads or into cubical packets. Several different names are given to them:

α *Spirilla* and *spirochætæ* are spiral filaments (*spirobacteria* of Cohn), having a well-marked cork-screw motion.

β *Bacilli (desmobacteria* of Cohn) are cylindrical bodies, which sometimes remain united after they have undergone fission, so as to form threads of considerable length.

γ *Rod-shaped bacteria* (*microbacteria* of Cohn) are very much smaller than bacilli. They are often slightly constricted in the centre or dumb-bell shaped.

δ *Spherical bacteria* (*spherobacteria* of Cohn, *coccaceæ* of Zopf) are also exceedingly minute. They may be called *micrococci*, but it is necessary to bear in mind that different writers have not always used this term with precisely the same meaning.

Sarcinæ are spherobacteria arranged in square or cubical packets, the result of fission in different planes.

By Billroth rod-shaped and spherical bacteria were associated together under the name of *coccobacteria*.

Bacteria sometimes cohere into chains; they are sometimes aggregated into masses held together by a jelly-like material, in which case the name *zooglœa* is given to them.*

* Microphytes are also classified as *aerobic* and *anaerobic* by Pasteur, according as they

It can be easily understood that there is often very great difficulty in distinguishing micrococci from lifeless granules of organic or inorganic matter. Active movements, when they are seen, are of course conclusive; but an oscillating motion is of no significance, being dependent upon vibrations transmitted from without to the liquid in which the granules float. Brunonian movements also affect dead particles. Bodies which subdivide or which cohere into chains may safely be set down as organisms. Irregularity in size is almost sufficient to show that the granules which differ among themselves are not micrococci; but uniformity in this respect affords no positive proof that organisms are present. The power of resisting liquor potassæ has sometimes been supposed to be a criterion; but I believe that it is valid against granules of caseine or of other protein compounds or minute drops of fat or animal cells.

It must be clearly understood that the above classification is one of names rather than of things, for there is still the greatest uncertainty as to the real relations between the different forms of these minute organisms. Consequently it is often advantageous to be able to refer to them under a common name, without specifying their exact characters; and for that purpose the word "microzyme" was suggested by Béchamp and adopted by Sanderson. Microphyte is perhaps better.

Cohn admits several species of schizomycetes. Nägeli says that he does not feel sure of the existence of more than one species, exclusive of the sarcinæ. At one time Lister believed that he had obtained a spirillum from the *Bacterium lactis;* but he has since become convinced that this was a mistake. There are, in fact, almost innumerable sources of error, which may vitiate the results obtained in long chains of experiments, such as those of Buchner and Dr W. Nägeli, which Prof. von Nägeli himself cites with approval, and from which it appeared that "*Bacteria lactis*, when placed in liquids containing extract of meat and sugar, undergo such an alteration that if afterwards introduced into milk they render it ammoniacal, and do not for a hundred or more generations regain the power of setting up the lactic acid fermentation." Lister himself found that this kind of bacteria, after cultivation in urine for several days, became somewhat less energetic in its action upon milk. Conversely, several observers have thought that septicæmic blood, and the products of acute inflammation, acquire an enormously increased virulence when transmitted through a series of animals in succession.*

grow in contact with oxygen, or protected from the air; and according to their physiological effects, by Flügge, as *septic* or putrefactive, *zymogenic* or fermentative, *chromogenic* or pigment-forming, and *pathogenic* or productive of specific diseases.—Ed.

* Zopf carried Nägeli's and Billroth's doctrine of the "pleomorphism" of the schizomycetes to its utmost limit. But more critical biologists admit variation in form of one and the same organism only within certain narrow limits. This was first proved by Lankester in the case of *Bacterium rubescens* (a chromogenic microphyte, since named *Clathrocystis roseopersicina* by Cohn), 'Quart. Journ. Mic. Soc.,' 1876.

Nor is there reason to believe that chromogenic microphytes can be cultivated into zymogenic or septic into pathogenic. The assertion by Buchner that the common *Bacillus subtilis* of infusion of hay was interchangeable with the Bacillus of anthrax and *vice versâ*, has been refuted by Klein. Pathogenic bacteria, however, may undoubtedly suffer diminution of their malignancy (or may acquire it in intenser degree) by being passed through a series of hosts. This is the explanation of Jenner's vaccination, for there is now no question that the cow-pox is variola passed through successive generations of cattle. And on the same principle Pasteur has succeeded in "attenuating" the virus of anthrax, of so-called "chicken cholera" and as he believes of hydrophobia, so as to protect against the unmodified disease by inoculation, or, as he calls it, "vaccination," with the attenuated contagium.—Ed.

Contagious pus.—Mr Hutchinson has repeatedly expressed the opinion that the "contagious inflammations" (as he terms them) differ essentially from the "specific fevers," and that they are conveyed from one person to another by leucocytes or living pus-corpuscles, and not by microzymes. The instances he enumerated in the discussion on the Germ Theory, held by the Pathological Society in 1875, were Gonorrhœa, Erysipelas, purulent Ophthalmia, and Porrigo. The points on which he relied in support of his views were chiefly two; first, the frequency with which some of these diseases occur apart from any apparent origin in contagion; and secondly, the fact that the severity of the inflammatory process in each disease varies widely in different cases, and may be modified at will in experimental inoculations, as when gonorrhœal pus is applied for the cure of pannus. This last argument seems, however, to have very little force, since we shall find that as much may be said of some maladies which are typically specific. But it is impossible to deny that erysipelas often appears to be directly caused by exposure to cold; and a similar origin can sometimes be traced for diphtheria—an affection, however, of which the specific character has been doubted on other grounds.

It seems a simple way out of the difficulty to suppose that the diseases in question begin as simple inflammations, and that they acquire infective characters secondarily. But such a view by no means excludes microzymes from being, in all probability, the bearers of contagion. It seems to me that Lister's observations on the lactic acid fermentation teach an important lesson in regard to this question. One would certainly have thought that the souring of milk, which so constantly occurs when that fluid is left standing for a few hours in warm weather, must depend upon causes which are in operation everywhere, and that any microzymes concerned in the process would be likely to be devoid of specific characters, at least until they had germinated within the milk itself. But Lister found that although the *Bacterium lactis* appears to be universally present in dairies, it is scarce in the world at large: milk which has been boiled may be exposed to the air in ordinary rooms for half an hour at a time; but while other bacteria and filamentary fungi may develop, it will rarely, under such circumstances, become curdled and sour, nor will it present the *Bacterium lactis*. Is it not probable that even though the occurrence of a common catarrhal inflammation favours the development of erysipelas or diphtheria, yet access of specific microzymes is the essential factor in the ætiology?

Spontaneous origin of contagia.—Some physicians of well-deserved authority, of whom the late Dr Murchison was one, think that certain typically specific diseases sometimes arise *de novo*, independently of contagion or of any virus derived, directly or indirectly, from previous cases. In reference to this question, it seems to me useless to insist on the fact that all such diseases must have had a commencement at some period of the world's history, just as a similar argument may fairly be disregarded when employed to support the theory of spontaneous generation of any of the lower plants or animals at the present time. On the other hand, I think that little stress can be laid on the circumstance that the inhabitants of certain countries, especially islands, remain free from particular exanthemata for many years, but afterwards suffer severely, when a case is introduced from without: for even if a disease were capable of springing up *de novo* under certain conditions, a great length of time might obviously pass in a given locality without the conditions being fulfilled. What does appear to my mind an argument of importance is that, when the origin of an outbreak of fever or of any other

contagious malady has been investigated with sufficient care, it has so often been traced in the most conclusive manner to a previous case, perhaps at a great distance, from which it may have been derived in some most indirect and unexpected way. The instances which I shall have to relate when discussing the ætiology of the several infective diseases render it, as I think, almost impossible that the development of any one of them *de novo* can ever be positively proved. And it is worthy of notice that particular instances, of which no satisfactory explanation can be given, occur in the case of smallpox —of which a spontaneous origin would generally be regarded as most unlikely—no less than in that of diphtheria, or typhus, or enteric fever, which are held by some authorities to be frequently so generated. Sir Thomas Watson cites Dr Gregory as having stated that, of the cases admitted into the Smallpox Hospital in his day, not one in twenty could be referred to any known source of infection. I am therefore strongly disposed to reject the doctrine that any of the specific diseases ever result from such causes alone as overcrowding or starvation; or even from the inhalation of sewer gas which contains no specific virus, but only the excrement of healthy persons.

It is, however, another question whether the microzymes of an infective malady, after escaping from patients, may not sometimes go on multiplying for long periods of time, and spread to distant localities, before they again penetrate into the human body. In the miasmatic contagious diseases we believe that such a process of development always occurs. In the strictly contagious diseases there seems to be no reason why it should not occur sometimes. Again, we have yet to learn whether some of these maladies may not occasionally be derived from the lower animals, as favus certainly is from cats.

Course of infection.—When the virus of an infective malady enters the human body an interval occurs during which the health of the patient remains apparently undisturbed; this is called the period of *incubation.* In some diseases, as in smallpox, it is almost always of the same length. This fact is of great importance, because it enables us to say, when a person has been exposed to infection, that after the lapse of a certain number of days, if not already attacked, he is safe, and may mix with other people without risk to them. It is, in other words, the foundation upon which rests the practice of *Quarantine.* The duration of the incubation of each specific disease has accordingly been studied with care. The only cases in which it can be positively determined are of course those in which there has been but a single exposure to contagion; but others, in which the exposure (though repeated) began only a few days before the patient's illness showed itself, are valid as proofs of a short incubation; and yet others, in which the exposure ceased many days before he felt ill, are valid as proofs of a long incubation. It seems now to be established, beyond all doubt, that whereas the incubation of each specific disease is fixed within certain limits for the majority of cases, there are yet some cases in which the limits are widely overstepped. Thus scarlet fever, which ordinarily has an incubation of nearly a week, sometimes breaks out within twenty-four hours of the first exposure to its infection. It is undoubtedly hard to reconcile this fact with what is otherwise a most reasonable explanation of the usual occurrence of a longer interval, namely, that it is occupied by the multiplication of the virus within the body. According to Nägeli, schizomycetes commonly double their numbers in from twenty to twenty-five minutes at the temperature of the blood. Lister found

that the *Bacterium lactis* took about an hour in completing the process of growth and subdivision. In diseases, such as syphilis, in which a local lesion precedes the development of constitutional symptoms, the virus may be supposed at first to multiply itself locally (in syphilis, within the hard chancre) and not to enter the blood in any considerable quantity until a later period. There is evidence that in the rare instances of the syphilitic poison being communicated by vaccination, the resulting chancre has yielded an infective material within ten days afterwards. On the other hand, an unfortunate case once occurred at the Charité Hospital of Berlin, in which variola was conveyed to a patient by the operation of skin-grafting, the graft having been taken from a person who happened to be in the incubation stage of the disease. This seems to show that from a very early period the whole of the skin contains the virus, which can have reached it only through the blood; and if so, the same thing must occur likewise in the exceptional instances in which smallpox is inoculated into the skin at a particular spot. In such cases, however, there is developed a "primary" or "mother-vesicle" which precedes the general eruption by some days. It therefore seems to be very unlikely that in any disease the multiplication of the virus during the incubation remains altogether limited to a local lesion, even if it is more active there than elsewhere.

Such speculations are of considerable importance, because they touch a question which is still discussed, namely, whether in specific diseases generally the various local lesions that characterise them are to be regarded as sources of blood-infection or as consequences of it. Thus in diphtheria, one opinion is that microzymes first settle upon the mucous surface of the fauces, or upon whatever part happens to be the seat of the diphtheritic process, and that they afterwards penetrate its tissues and enter the blood. Some observers have even thought that in enteric fever blood-infection is secondary to the intestinal lesions; and Liebermeister endeavours to prove that in yellow fever there is a primary parenchymatous hepatitis. But in typhus and in relapsing fever we have diseases to which such a view is altogether inapplicable; and, in the exanthemata, fever or other constitutional symptoms generally precede the cutaneous eruptions by a considerable interval of time. Moreover, in diphtheria itself there is a well-marked tendency for the throat to be affected when the virus has originally been deposited upon some other mucous membrane or even upon the cutaneous surface, and when it may therefore be supposed to reach the fauces through the blood.

Physiological action of the microzymes.—With regard to the mode of production of the pyrexia and of the other symptoms of specific diseases by their contagia we know at present scarcely anything. The microzymes, in multiplying so enormously as they do, must of course abstract nutrient materials from the blood and from the tissues. By Nägeli the idea of a "struggle for existence" between these rival organisms is carried out in detail. It is shown that when a person is exposed to contagion his taking the disease or resisting it depends probably in part upon the number of microzymes which enter his body, in part upon the condition of his blood and of his tissues at the time. Everyone who has experimentally cultivated the lower organisms knows that when more kinds of organisms than one are present, a slight change in the composition of the fluid in which they are placed may completely alter the result, enabling a microzyme which previously was weaker to outgrow that which had been the stronger. And so Nägeli imagines that slight alterations

in the state of the blood may greatly favour or oppose a contagion in the competition which it undergoes. The analogy of the various fermentations suggests (as Lister has pointed out) that, besides appropriating the materials for their own growth, microzymes may also cause further disturbance by catalytically decomposing other substances of which they make no use. It is therefore not surprising that their presence sets up pyrexia, which I believe to be a quasi-physiological process, having for its object the destruction and removal of noxious matters of whatever kind from the circulating fluid. The evidence in support of such a view will be discussed hereafter (cf. p. 44).

Such a destruction of the contagious microzymes within the blood, as the result of the febrile process, would explain what has been, in the minds of many pathologists, one of the greatest obstacles to the acceptance of the so-called "germ-theory" of infective diseases, namely, the fact that so many of them run a definite course, and terminate in the recovery of the patient at the end of a fixed period of time. But a further hypothesis seems to be absolutely necessary. It is well known that most of these diseases occur only once in the life of the same individual; and perhaps no one of them is entirely devoid of such a protecting influence. I fail to see how this can be accounted for except on the supposition that in the course of each disease the blood or the tissues undergo such a change that they no longer afford, and never afterwards afford, the conditions requisite for the development of the corresponding microzymes. Perhaps it is not necessary to imagine that a special pabulum is exhausted, and there certainly are very obvious objections to the notion that human beings in general are born provided with a number of different materials which serve no known purpose but that of affording nutriment to the various contagia, if they ever find their way into the body, at however advanced a period of life. It is, however, to be borne in mind that the facts of contagiousness to other individuals, and that of subsequent immunity for the person already attacked, are almost necessarily connected together. Were it not so, almost everybody who passes through any one of these maladies would catch it from himself again and again.

The duration of the protective action seems to be very variable. In some diseases, as in relapsing fever and in diphtheria, its duration is perhaps no greater than is just sufficient to prevent the patient from reinfecting himself. In others it lasts during the whole of life. Indeed, a very interesting question is whether it may not be transmitted from parent to child. As is well known, the exanthemata sometimes rage with extraordinary virulence when they are introduced into communities that have been free from them for a great length of time. No doubt other explanations of this fact may be given; but it is at least possible that the reason may be that the victims come of a stock which has not for many generations been exposed to the contagion. It would be very interesting to investigate to what extent differences in the severity of a specific disease, attacking various individuals under the same conditions, in countries where it is always more or less prevalent, may depend upon whether one or both of the parents of the several patients had had that disease in childhood, or at least before the patients themselves were born. It might even happen, in the case of a malady which commonly affects adults, that successive generations should in turn succumb to it, in consequence of the continued absence of such an inherited protection.

Prevention of infective diseases.—These considerations seem to me to render very doubtful the advisability of attempting, even if it were practicable, to

eradicate infectious diseases from this or any other country, and then to keep them off by a system of quarantine. Sir Thomas Watson gave to such a scheme the weight of his authority. But sooner or later each disease would be sure to find an entrance, and it would probably commit unheard-of ravages among a population so long free from it. I doubt whether it is even right to teach the public to look upon the maladies in question as in a special sense "preventable." They are certainly not so much so as those affections which result from over-indulgence, from exposure to cold, or from hard work unsuited to the physical powers of the individual. The true method of combating infection seems to be that of which we have an illustration in the practice of vaccination. And there is reason to hope that in the future this method may be far more widely extended than hitherto. Pasteur and Toussaint in France, and Dr Greenfield in this country, have independently demonstrated the fact that by passing the virus of anthrax through guinea-pigs, its intensity can be diminished so that it is no longer fatal to bovine animals, and that after inoculation with this modified or attenuated virus they are no longer susceptible of the disease. One could not render a greater service to humanity than by discovering how to transmit the contagion of scarlet fever through any of the lower animals in such a way that when reintroduced into the human subject it should convey protection without risk. I am not even sure whether it might not be well to expose children on purpose to infection from this disease, when it happens to be epidemic in a mild form. Such a procedure has sometimes been advocated, but medical opinion has hitherto been that it cannot be done with safety, because a mild case often generates a severe one. But it is at least possible that this may only be true when the disease was originally of a severe type, and when it has chanced to produce a slight illness in an individual who (having inherited protection) is but little susceptible of it, so that it reverts to its original character when it passes on to a subject not thus guarded against it. For it is well known that, as a matter of fact, different epidemics of scarlet fever differ widely in intensity. Sometimes it is the most fatal of all contagious diseases; but sometimes it passes from house to house through an entire village, without destroying a single patient or giving rise to any dangerous sequelæ. May it not be that this is where the activity of the virus has been lowered by passing through a number of persons in succession, each of whom was only slightly susceptible of it? If so, we may possibly find a very simple means of obtaining a virus suitable for inoculation, without having recourse to the lower animals.

Method of transference of contagia.—We have still to consider how the microzymes of contagion or of miasm gain access to the human body.

According to Nägeli, the way in which they enter when suspended in the air is generally by penetrating into the capillaries of the lungs through the walls of the pulmonary alveoli. He is inclined to doubt whether they ever make their way through the mucous membrane of the mouth or the fauces, except when they settle upon it and first give rise to an intense local inflammation, as he (with so many other German writers) believes to be the case in diphtheria. But he thinks that they sometimes enter the blood through wounds or abrasions. It has been supposed that examples of such a mode of infection are sometimes afforded by the so-called "surgical scarlet fever," which so often breaks out in children after operations; but, as Dr Goodhart has shown, in the 'Guy's Hospital Reports' for 1879, that its occurrence is not prevented by the strictest "antiseptic" treatment, there appears to be

more probability of its arising in the ordinary way. This, however, is a point which I shall have to discuss hereafter.

Contagia have traditionally been divided into those which are "fixed" and those which are "volatile." Miasmata have been regarded as of necessity volatile. But, as we have seen, it is certain that none of them are gases or vapours, and that they all consist of solid particles of exceedingly small size. Nevertheless, even recent writers have taught that the particles in question are capable of escaping with the water which evaporates from liquids or from moist solid surfaces, and of diffusing themselves in the air.

Nägeli, however, seems to have conclusively demonstrated the very important fact that this is impossible. He performed a series of experiments with U-shaped tubes connected together. In one bend he placed a liquid suitable for the growth of microzymes; another bend he filled with sand saturated with a putrefying liquid. Even when air was drawn through the apparatus he found that no microzymes passed from one tube to the other. And on theoretical grounds he shows that no other result could have been anticipated.

The full acceptance of Nägeli's conclusions means little less than a revolution in our conceptions of the process by which contagia escape from the human body or miasmata from the soil. We have been accustomed to think that the breath of persons suffering under an infective disease is apt to be highly charged with the virus, and that the offensive odours exhaled from his moist skin, or from his excreta, may be highly dangerous. According to Nägeli all such notions are erroneous; he even declares that the presence of fœtor is generally a sign that the substances which give rise to it are still moist, and are therefore incapable of setting free any microzymes which they may contain. But it is obvious that some parts of an infected mass may remain covered with fluid, while others have already undergone desiccation.

Of course it is not disputed that liquids in which the microzymes of a specific disease are floating may convey it to healthy persons. Indeed there are some maladies, such as hydrophobia and syphilis, which seem scarcely to be communicable except by a process of inoculation, the poison being introduced into a bite or a wound, or a crack in the skin. And diphtheria is often spread by the direct transference of fluid secretions from one individual to another as in the act of kissing, or when a patient coughs and expels mucus into the open mouth of a medical man who is looking into his throat. It is also to be remembered that the stings of insects may sometimes be the means of conveying the virus; cattle, for instance, are liable to be infected with anthrax by gadflies.

Moreover, there is the clearest evidence (although this Nägeli rejects) that certain specific diseases are frequently transmitted by means of liquids which are swallowed. So far as drinking water is concerned I shall have to cite the facts in full detail when I am describing enteric fever and cholera. Still more interest attaches to the remarkable facts which have recently been made out by English observers as to the conveyance of the poisons of enterica and of scarlatina by means of milk. It is probable that, even in water, the specific mycrozymes multiply greatly in the interval which elapses, after it becomes contaminated by them and before it reaches the human stomach. But there can be little doubt that milk affords far more favourable conditions for their growth; for Lister proved in his experiments with this fluid that (unlike even Pasteur's solution) it was capable of serving as a

pabulum for almost any kind of microscopic organism. Once only did he meet with a bacterium that could not live in it. Now, I shall hereafter have to point out that in some of the cases in which specific diseases have been widely communicated in this way the quantity of virus which has been allowed to enter the milk has probably been very small. It, therefore, seems to me that the occurrence of milk-epidemics tends powerfully to support the germ-theory. I may observe that probably the only effectual method of purifying either milk or water, when contaminated with microzymes, is by boiling. Filtration seems to be altogether useless; and it is not likely that the addition of minute quantities of Condy's fluid would suffice to destroy them.

With these important reservations, however, I think that Nägeli is right in maintaining that infection generally occurs in a very different manner, namely, by means of microzymes which have undergone desiccation, and which rise into the air and remain suspended in it in the form of a fine dust: the particles, perhaps, even more minute than those which become visible in the track of a sunbeam. How little influence gravitation has in causing the subsidence of such particles is well illustrated by the facts that volcanic dust from Vesuvius is carried in the atmosphere to distant parts of Europe, and that the trade-winds convey another kind of dust right across the Atlantic Ocean from America.

The conditions under which microzymes thus become suspended in the air must be discussed separately for miasmata and for contagia.

(1) In the case of a *miasm*, at least any *miasm* of telluric origin, the first condition of its development is the presence of water in the interior or upon the surface of the ground, and the presence in this water of substances fit to serve as pabulum for the microzymes. Further, it is necessary that the level of the water should vary from time to time, so that particles of the soil itself, or the stems and the leaves of plants growing upon it, should undergo desiccation, and should receive deposits of microzymes, also in a dry state. So far as the miasm of ague is concerned, I do not know that there is any necessity for supposing that it is ever derived from any depth below the surface of the ground, or for invoking the assistance of any force beyond that of the wind, for the purpose of carrying up the dried microzymes into the air. But if any value at all is to be attached to the observations which Pettenkofer and others have made at Munich with regard to enteric fever and to cholera, microzymes concerned in the propagation of these "miasmatic-contagious" diseases must be assumed to come in some cases from the soil at a depth of several feet. The conditions which may be supposed to render this possible are very carefully argued out by Nägeli. In the first place, he thinks that in the ground-water which saturates a porous soil below a certain level, the growth of such organisms is likely to be especially active towards the surface of this water. Hence the slightest fall in the level of the water must cause an abundant settlement of microzymes upon stones, pieces of sand, and fragments of clay and humus, which come into contact with air as soon as the water recedes from them. In this subterranean atmosphere he conceives that currents are produced by various causes: changes of temperature, changes of pressure, winds, and lastly, the suction action of warm air into the interior of houses having deep foundations. Such currents, he thinks, may easily carry away the dry microzymes, especially if there is no precipitate of colloid matter from the water, to make them adhere closely to the substances on which they are deposited. A further condition, at least

so far as concerns their escape into the air above, is that the superficial layers of the soil through which they must pass should not be damp, and should not be covered with a uniform carpet of vegetation.

(2) The microzymes of a *contagion* doubtless leave the human body mainly in the fluid secretions and excretions, the fæces, the urine, and the sweat. They may also be attached to the solid particles of cuticle which are constantly being shed from the surface; in the case of scarlet fever and in that of smallpox this mode of diffusion of the poison is believed to be of great importance. In either case it is hardly necessary to say that their escape is due to no process of "elimination," and is of no advantage whatever to the patient. Indeed, the microzymes which find their way out of the body are probably altogether insignificant in number, when compared with those which remain in the blood or in the tissues, and undergo destruction there. The microzymes which adhere to fragments of cuticle are already dry; those which are contained in fæces, in urine, or sweat, must undergo desiccation before they can reach the air. Nothing is more favourable to this than the soaking of linen or other fabrics with any of the infected secretions. The microzymes remain upon the surface of the cloth when it dries, and are afterwards shaken off by the slightest movement. It is a most natural circumstance, therefore, that washerwomen and their families should be very liable to attack by contagious diseases; and instances in which infection has been traced to the hanging out of clothes to dry are readily explicable if one considers how imperfectly the process of washing is often performed.

Persistence of contagia.—One cannot state with any approach to certainty, how long a patient remains capable of infecting others after his recovery from a contagious disease. It is not unlikely that his secretions cease to contain microzymes, except in very small quantities, as soon as the pyrexia is at an end. This at least appears to be the natural inference from the observations which have been made, of a rapid disappearance of *spirillum* from the blood in relapsing fever. But for a considerable time afterwards fresh portions of infected cuticle may be supposed to be continually shed.

On the other hand, contagia sometimes adhere with great tenacity to the walls of an apartment which has been tenanted by a patient suffering under an infective disease, and to bedding or clothes which have been used by him. Nägeli mentions that at Munich several masons fell ill with smallpox after scraping the ceiling of a room in which smallpox cases had been treated six or seven years before, and which had then been whitewashed. Sir Thomas Watson relates the following instance: A house in which several persons had been attacked by scarlet fever was left empty for a year. When the family returned, a drawer in one of the bedrooms resisted for some time attempts to pull it open. A strip of flannel had got between the drawer and its frame, and had made the drawer stick. This piece of flannel the housemaid put playfully round her neck. An old nurse who was present, recognising it as having been used as an application to the throat of one of the subjects of scarlet fever, snatched it away and burnt it. The girl, however, soon sickened with the disease. Woollen substances seem to afford the most favourable conditions that can be imagined for the preservation of microzymes in a state of activity, short of their being enclosed in sealed glass tubes. By the hygrometric properties of such substances, contagia adherent to them may probably be prevented from undergoing too complete a desiccation, while they are at the same time protected from currents of air.

Destruction of contagia.—Steps must always be taken to prevent a person affected with a contagious disease from being a source of danger to others. But what can really be done in this direction does not seem always to have been clearly apprehended by those who have laid down rules for the purpose. It would appear:

A. That during an illness, the utmost care should be exerted to hinder the contagion from being carried away from the sick-room by means of attendants, or in the patient's excreta, or upon the linen of his bed or body, or in any other way. In all probability its escape into the open air through windows or chimneys is of no consequence.

B. That at this time, it is impracticable effectually to disinfect the patient himself or his surroundings, and therefore that the attempt should not be made.

C. That the patient, after recovery, and the nurses also, should be most carefully disinfected; that minute pains should be taken to prevent clothes, books, and other articles from conveying contagion; and that the sick-room with all its contents should be thoroughly purified before healthy persons are allowed access to it.

The measures which should practically be adopted to ensure these objects seem to be the following:

1. The patient himself must be isolated in a separate room or suite of rooms, which, if possible, should be at the top of the house. An intervening passage, of which the windows can be opened, is, of course, an advantage. Ventilation should be carefully attended to, and it is well that there should be a fire, so as to maintain a draught up the chimney. The doors should be kept closed, and outside them there should be hung an old sheet saturated with a liquid solution of carbolic acid or chloride of lime, and never allowed to become dry. The addition of glycerine to this liquid is probably advisable. It seems to be useless to expose vessels containing carbolic acid in the sick-room itself, or to scatter chloride of lime upon the floor.

Those who nurse or wait upon the patient should not be allowed to enter other parts of the house, and, if possible, such persons should be chosen for this duty as have already had the disease. The medical attendant should take care by ablutions, by change of clothing, and by exposing himself freely to the open air after each visit, to avoid carrying contagion abroad. One is often asked whether the father of a sick child should be allowed to continue his work in an office or chambers, so as to come into contact with fellow-clerks and with the public generally. My own opinion is, that if he avoids contact with the patient and takes a good walk when he leaves the house in the morning, no appreciable risk is run, so long as he himself remains well.

All curtains, carpets, and padded chairs, all articles of wearing apparel in cupboards or in drawers, all unnecessary articles of furniture of whatever kind, should be removed from the sick-room before the patient is placed in it, because their presence will make unnecessary difficulties afterwards. The supply of books or toys should be limited to such as are not too valuable to be burnt when they are done with. Any food or drink which the patient or the nurse may leave should be thrown away, and must never be taken by other persons. Cups, plates, and spoons should be washed in very hot water before they are allowed to go back to the kitchen; but, as far as possible, it is well to keep the same articles from day to day in use in the sick-room, and to wash them there.

2. All the excreta of the patient should be rendered innocuous by the addition of some disinfectant.

The experiments of the late Dr Baxter, recorded in the sixth volume of the new series of Mr Simon's 'Reports to the Privy Council,' have shown that few at any rate of the substances commonly used really possess the power of destroying contagia. He employed *carbolic acid*, *sulphur dioxide*, *potassic permanganate*, and *chlorine*. Each of these substances was added in definite proportions to vaccine lymph, and a series of vaccinations were performed with mixed liquids upon children, one arm of the child being inoculated with the lymph supposed to be disinfected, while the other arm was at the same time vaccinated with lymph that had been diluted with water to a corresponding degree. The results which he obtained were: that with chlorine and with potassic permanganate there is no security for the effectual disinfection short of the presence of free chlorine or of undecomposed permanganate in the liquid, after all chemical action has had time to subside. In the case of sulphur dioxide the criterion of success is that the liquid should be permanently and strongly acid; in the case of carbolic acid that it should contain at least 2 per cent. by weight of the pure acid. It is further necessary that the disinfectant should be thoroughly incorporated with the liquid; there must be no solid matters capable of shielding the contagious particles.

An additional point on which Dr Baxter lays stress, and on which Nägeli had previously insisted, is that the addition of disinfectants in too small quantities may do harm instead of good. Probably all the substances that can destroy contagia are also antiseptic agents; that is, they can prevent putrefaction. And it appears that they are capable of doing this even when present in too small a quantity to disinfect. At first sight this seems contradictory. But it is not really so; for Dr Baxter has shown, that when an infusion to which (for example) 2 or 3 per cent. of carbolic acid has been added fails to putrefy, the bacteria in it nevertheless remain alive, and that they begin to multiply if a little of the infusion is mixed with another liquid that can afford them pabulum. Now, it is this power of affecting other liquids, and not its capacity for undergoing putrefactive changes itself, which is really comparable with pathological infectiveness. And it is a well-ascertained fact that (except, perhaps, in the case of glanders) the occurrence of putrefaction in a liquid deprives it at once of whatever contagious property it may possess. Thus, if a disinfectant is added in too small quantity, it may easily happen that its action is precisely the opposite of what is intended: by preventing putrefaction, it keeps the contagious microzymes alive.

3. For the purpose of wiping off discharges from the nose or the mouth of the patient, pieces of rag should be used, and they should be burnt immediately afterwards. The sheets and body-linen should be placed in a tub of water as soon as they are changed, and they should of course be washed apart from those of other persons. It is usual to soak them in a weak solution of carbolic acid or in diluted Condy's fluid; but this is probably of little use. The really important point is that they should be submitted to the action of water at a high temperature. The safest plan is to boil them in a copper. But there is reason to believe that a degree of heat far below 212° may be sufficient. Thus, Davaine found that the virus of anthrax diluted with water was destroyed in five minutes by a temperature of 131° F., and Dr Baxter that even dry vaccine was rendered inert in thirty minutes by a temperature of 185° or upwards. There appears to be no doubt that in the

dry state contagia resist heat much better than when they are suspended in water.

The linen worn by the nurse should be treated in the same way as that of the patient. The dress should always be made of a material that will bear washing.

4. At the end of the illness, when it is believed that the patient is no longer giving off contagion from his body, he must be carefully washed, carbolic soap being perhaps preferable to other kinds. Special pains should be taken to cleanse the hair thoroughly. He should then be dressed in clothes none of which have been in the sick-room, and should be moved into another apartment. Similar precautions should be taken whenever a nurse or an attendant goes away, at whatever period of the case.

5. The room which has been occupied by the patient during the disease must be carefully disinfected before healthy individuals are allowed to occupy it.

In the first place, moveable articles that cannot be washed should, if possible, be exposed to dry heat. As I have already remarked, the temperature required for the destruction of contagia in the dry state appears to be considerably higher than that which suffices when they are suspended in a fluid. It varies with the length of exposure, the time needed being longer in proportion as the temperature is lower. Some hospitals for infective diseases have hot-air chambers, erected for the purpose, in which there is no difficulty in raising the temperature, even in the centre of a flock-bed, to 220°, 240°, or 300° F. There seems to be no doubt that this must be completely effectual in killing any kind of virus.

The next step is to destroy any microzymes that may be floating in the atmosphere, or adhering to the sides of the apartment, or to the furniture. With this object it may be exposed to the fumes of sulphur or to chlorine gas. All the openings into the room, chimney, windows, and doors, must be made as air-tight as possible. A good plan is to set fire to a few ounces of bisulphide of carbon in an iron pan supported over a pail of water. Or from a quarter to half a pound of brimstone broken into pieces may be placed in the pan and live coals used to ignite them. The room is then closed and left so for several hours. The ceiling should also be scraped and whitewashed, the walls should be re-papered, the floor should be thoroughly scrubbed, and all the paint in the room, as well as the furniture should be very carefully washed.

FEVER

Pyrexia—Thermometry—Clinical course of pyrexia—Hyperpyrexia—Theory of fever—Possibly beneficial effect of pyrexia—Idiopathic and symptomatic fever—Raised temperature without fever—The physiological stages of fever—Concomitant phenomena of pyrexia: nutrition: pulse: respiration: muscles—Subnormal temperature.

AMONG the varied effects of disease there is, perhaps, none which is more commonly met with—and certainly there is none which is more important—than PYREXIA or FEVER. It is not surprising, therefore, that the ancient writers should have recognised this condition, which by Galen was defined as "*Calor præter naturam.*"

But it is not a little strange that in modern times scarcely any attention was paid to it until very recently. The specific fevers, of course, were studied; but of the general febrile state little or no notice was taken. An increased action of the heart was considered as its chief characteristic.

It would be difficult to overstate the value of an advance which was made early in the second half of the present century, in the introduction into medical practice of the clinical thermometer, as a direct and simple method of determining the presence or absence of pyrexia. This was not, indeed, a novelty. About a hundred years previously the instrument had been used by Boerhaave and by his pupils Van Swieten and De Haen; and the last of these, who was the first clinical teacher at Vienna, had discovered the striking fact that in ague, during the cold stage, the temperature of the blood is increased. But when in 1850–51, Traube and Bärensprung independently published papers on the subject, no one else was in the habit of making thermometric observations at the bedside. From that date Wunderlich, of Leipzig, devoted infinite pains to the study of temperature in all diseases. In this country the practice was soon adopted by Parkes, Ringer, Aitken, and a host of others; and we may safely assert that it will never again be abandoned.

Methods.—The use of the thermometer in clinical practice requires considerable care, if the results are to be relied on as being even tolerably accurate. The instrument is most commonly placed in the axilla, but sometimes in the closed mouth, the rectum, the fold of the groin, or elsewhere.* Now, it is essential to observe that whichever of these places may be selected for the purpose, the temperature which is to be obtained is that not of any part of the cutaneous surface, but of the deeper structures of the body, an inch or more below the level of the skin. To determine the surface temperature of any region is quite another, and a most difficult matter. Instruments which have been called surface-thermometers have been constructed,

* By far the most rapid method of taking the temperature of the body accurately is for the patient to hold the bulb of the thermometer in the stream of urine when emptying his bladder. The glass and mercury are rapidly heated, for the cooled fluid passes at once away. The method is most applicable for taking the temperature in the evening before going to bed, and also for ascertaining the presence or absence of pyrexia when a patient is first seen. I learnt it from a short paper by Dr Oertmann in 'Pflüger's Archiv,' Bd. xvi, S. 101.—ED.

with the receptacle for the mercury of such a shape that one side of it can lie flat on the skin while the other side is protected with a non-conducting material, so as to diminish as much as possible any loss of heat from it. But to cover up any part of the surface of the body is, *ipso facto*, to raise its temperature above that at which it previously stood.

When a thermometer-bulb is placed deeply in the axilla, the fold of the groin, or elsewhere, the mercury goes on rising until it indicates a temperature corresponding very closely with that of the solid substance of a limb, at the same distance from the skin as that at which the instrument is buried. Consequently it is extremely uncertain what length of time such an observation may take for its completion. A great deal depends upon whether the parts were closely in contact before the thermometer was introduced. Thus, if the patient is lying on one side in bed, one should always choose the more dependent of the two armpits, since it will give a fixed temperature far more quickly than the other one. When a thermometer has once risen to its full height in the axilla, a second one placed in the same spot immediately afterwards will rise to the same point in from three to five minutes. But it is a very different matter if the arm has been separated from the chest, so that the axilla has contained air, and perhaps a thick fold of the underclothing. It must then itself rise through several degrees before it can bring the thermometer to a stationary point. And the length of time required for this to take place will vary indefinitely, according as the circulation of blood in the peripheral parts of the body is active or otherwise. When the skin is hot and turgid, as in scarlet fever, a comparatively short period will suffice ; in the cold stage of ague, and still more during the collapse of cholera, it is doubtful whether an axillary temperature can ever be relied on. It is to be observed that this uncertainty is altogether independent of any defect of sensitiveness in the instrument which happens to be employed.

I have gone into the matter thus minutely because there is reason to believe that the scientific value of thermometric observations made in this country is largely impaired by the comparatively short space of time devoted to them. It is commonly thought that from three to five minutes suffice for this purpose ; and it must of course be admitted that a temperature above the normal, however quickly obtained, is a positive fact, which from a practical point of view can never be worthless. Dr Bäumler, however, has shown ('Brit. Med. Journ.,' 1864) by direct experiments that, even when all ordinary precautions are taken, it may easily happen that too low a reading by ·3 to ·8 of a degree Fahrenheit is arrived at, if the thermometer is withdrawn from the axilla at the end of five minutes. He found that from eleven to twenty-four minutes are required to give an absolutely trustworthy result. Liebermeister goes still further : for even of a period of from fifteen to thirty minutes, he does not say more than that it suffices for the majority of cases. The rule which he lays down is that the instrument should be observed to remain stationary for five minuees before it is removed.

It is essential in placing the bulb in the axilla that no clothes should be allowed to remain in contact with it, and that the skin should grasp it firmly. If there be any perspiration, the hollow must first be wiped dry. The arm should then be folded across the chest, and the hand may be made to take hold of the opposite arm, while the opposite hand supports the elbow of the side on which the thermometer is. Whenever the parts do not seem to meet closely round the instrument, some other region should be selected. The mouth is suitable, if one can depend upon the patient's keeping it constantly

closed and breathing entirely through the nose. The bulb may be placed either beneath the side of the tongue or at the back of the cheek. Dr Bäumler found that from nine to eleven minutes sufficed to raise it to a fixed point there. But of all possible localities for thermometric observations, the rectum is by far the best. It would always be preferred, did not considerations of decency stand in the way; and in very young children as well as in old people this may often be neglected. An important advantage is the saving of time, for from three to six minutes are enough to give a temperature of scientific value. Consequently it is advisable to make use of the rectum whenever much depends upon the exact temperature at the moment, or when an ice-bath may perhaps be required. And it is to be noted that a rectal temperature can be taken with a registering thermometer while the body is immersed in cold water.

When, after the withdrawal of the instrument, the index has been read, it is common to dot down the result on a sheet of paper ruled for the purpose. Observations are repeated at regular intervals twice a day, every two hours, or even oftener, and each successive reading is recorded in a similar manner. Lines are then often traced from dot to dot so as to form what is termed a "chart." It is, however, very important to remember that the result is entirely artificial. However short may be the intervals at which the thermometer is applied, there is no reason for supposing that the patient's temperature moves straight upwards or straight downwards from one point to another. And when the intervals are long, as when the instrument is employed only twice in the twenty-four hours, there is not even a probability that the dots marked on the chart each day represent respectively the true maximum and the true minimum. We shall, indeed, presently see that in disease as in health there are, as a rule, certain daily fluctuations, the temperature being generally highest at a particular hour in the evening and lowest in the morning. But even when the observations are made just at those times, there is no security that the rule is observed in that special case on any one day. And it is always highly probable that between each pair of observations there may have been two or three or even several ascents and descents of the temperature which altogether escape notice. I therefore prefer to make no chart at all, but to place the figures themselves in two vertical lines, one for the morning the other for the evening.

The course of pyrexia is naturally divisible into certain periods or stages, which, however, vary greatly in length in different diseases. First comes the "pyrogenetic" or "initial" stage; during its continuance the temperature rises more or less steadily, the rise being interrupted, if it extends one, two, or more days, by the daily fluctuations already alluded to. When it is of short duration or when the rise is at first rapid, there is commonly a shivering fit or *rigor*. The second stage is the *fastigium;* in it the temperature reaches its *acme* or highest point; but, not infrequently, since this stage may last for several days or even two or three weeks, it is characterised (independently of the daily fluctuations) by a series of irregular slight ascents and descents, so that the highest point or one very near it may be touched again and again at longer or shorter intervals. The third stage is that of "defervescence," during which the temperature falls again to normal. Sometimes, however, it is separated from the second stage by an intervening period, called by Wunderlich the "amphibolic" stage, in which irregular exacerbations and remissions are observed. Or, again, there may at the end of the second stage

be a marked rise of temperature termed by Wunderlich the "*perturbatio critica.*" The third period, that of defervescence, is sometimes protracted, sometimes short and sudden. In the former case the pyrexia has been said to end by *lysis*, in the latter by *crisis*. The rule is to speak of a critical termination only when the fall to a normal temperature is completed within thirty-six hours; but sometimes the third stage begins with a slight and gradual descent, which after two or three days ends in a crisis. When defervescence is rapidly accomplished, it is generally accompanied by profuse sweating and sometimes by an abundant flow of some other secretion.

That Wunderlich and others should thus have adopted the term "crisis" is, I think, to be regretted. For it is scarcely possible for anyone who has had occasion to refer to the medical writings of the ancients to shake off entirely the remembrance of earlier mystical notions which formed a very important part of their teaching. When I come to describe the specific forms of fever, we shall find that each of them has a more or less definite duration, and that one can often confidently look forward to a future day, a week or two distant, as that in which defervescence is likely to take place. But this is very far from what the Greek writers or even some moderns have meant by critical days, their idea being that all fevers, without reference to differences between them, should come to an end on certain days rather than on others, so that when one critical day was passed, the disease might be expected to run on until the next critical day occurred. The whole of the doctrine in question is absolutely without foundation.

After defervescence the temperature often remains for some days slightly subnormal. It is also less stable than in health, being easily disturbed by slight causes, so that, for example, a rise of two or three degrees is apt to follow the first solid meal.

It is often convenient to be able to express in general terms the degree of pyrexia without giving the actual temperature. And for this purpose Wunderlich's classification may be adopted; it is as follows:

1. *Subfebrile*, temp. 99·5°—100·4° Fahr. (37·5°—38° C.).
2. *Slightly febrile*, temp. 100·4°—101·3° (38°—38·5° C.).
3. *Moderately febrile*, temp. 101·3°—102·2° in morning; 101·3°—103·1° in evening (38·5°—39° C. in morning; 38·5°—39·5° C. in evening).
4. *Decidedly febrile*, temp. about 103·1° in morning, about 104° in evening (39·5° C. in morning, 40·5° C. in evening).
5. *Highly febrile*, temp. above 103·1° in morning, above 104·9° in evening (39·5° C. in morning, 40·5° C. in evening).
6. *Hyperpyretic*, temp. approaching 107·6° or even higher (42° C.).

The routine use of these expressions may, however, under certain circumstances, suggest a false impression as to the gravity of particular cases. In children fever is set up very easily; an evening temperature of 105° may be due to comparatively trifling causes; and on the following morning the thermometer may not rise far above the normal point. So again, sensitive women sometimes show a "highly febrile" condition, when the result proves that no apparently adequate cause for it has been present. On the other hand, in old people the temperature is apt to be below what one would have expected from the gravity of the case.

With regard to the term *hyperpyrexia*, I shall have to make some remarks further on. At what point it should begin to be used is somewhat uncertain;

but it is generally understood to mean such a temperature as is sufficient of itself to endanger life if continued for any length of time, Cases in which the thermometer rises to 109° or 110° (43·5° C.) are very exceptional, and most observers think that they are invariably accompanied by severe and alarming symptoms. Just before the death of a patient from tetanus Wunderlich obtained a temperature of 112·55° (44·75° C.): this is said both by him and by Liebermeister to be the highest reading that has hitherto been certainly taken.

On the other hand, there have in this country been recorded a few instances in which, if they can be relied on, it would seem that far higher temperatures have occurred, not only without being followed by death, but sometimes without being attended with any serious symptoms whatever.

The first case of this kind was observed by Mr J. W. Teale ('Clin Trans.,' 1875) in the person of a young lady, who by a severe accident had several ribs broken, and afterwards suffered from great tenderness over the dorsal vertebræ. Two months later her temperature was one day taken at 110°; and afterwards the index of the thermometer was on four occasions buried in the bulb at the top of the instrument, at a point above 122°. Sometimes these extraordinary temperatures were taken in the axillæ, sometimes between the thighs, or even in the rectum. She was at first in an exceedingly weak state, but she gradually improved and regained fair health.

Other examples of "paradoxical temperatures," as they have been called, have been met with by Dr Donkin ('Brit. Med. Journ.,' 1879). His first case was that of a nurse who was recovering from enteric fever, when the thermometer was found one night to register 110°. Afterwards very high temperatures were repeatedly taken, on a single occasion one of 111·6°, yet no symptoms could be discovered accompanying this reading beyond a feeling described by the patient as one of "flushing" or "rushes of heat." But perhaps the most singular circumstance of all was the evanescent character of this pyrexia, if it deserves that name; once the thermometer rose to 107·2° in the right axilla, whereas five minutes later it stood at 98·6°. In the mouth a temperature of 106° was once observed. It does not appear that the instrument was ever held *in situ* while such extraordinary results were being obtained, but the patient's hands were watched, the idea of imposture being well kept in mind. Seven other cases are cited by Dr Donkin, all but one of them being in females.

In 1879 a remarkable instance of this kind occurred in Guy's Hospital under Dr Moxon. The patient, a girl of 22, had been in the ward for phthisis during ten months, when on the evening of July 25th her temperature was taken at 107·4° and about an hour afterwards at 110·8°. She appeared to be suffering somewhat from dyspnœa. On the following morning the thermometer stood at 99·8°. During the next few months the most extravagant variations of temperature were recorded. On one occasion Dr Mahomed obtained simultaneously a reading of 102° in one axilla, one of about 114° in the other axilla, one of 107° in the mouth. On changing over the instruments the highest temperature was attained in the axilla where it had before been lowest, that of the mouth being now 104°. Another day a small registering thermometer gave 102·6° in one axilla, while another one in the other axilla gave 109·4°; but directly afterwards, when two large instruments without indices were used, and when the patient's arms were held all the time, the temperature stood at 103° on each side. Dr Mahomed noted that the skin always felt moist and of the ordinary temperature, even when

a very high reading of the thermometer had just before been obtained. He never got a high temperature with a non-registering thermometer, when he himself held the instrument in the axilla, keeping his hand pressed against the patient's arm. It is certainly difficult to avoid the conclusion that, in this case, some deception was practised, although its nature was never discovered. The girl died of disease of the lungs on March 22nd, 1880. Can a patient without being observed, squeeze or rub the bulb of a thermometer, so as to drive the mercury up ?

Theory of fever.—In studying the nature of pyrexia we may, in the first place, safely assume that the *sources* of febrile heat do not differ in kind from those which normally keep the human body at a temperature above that of the external air.

In 1863, the late Prof. Traube, of Berlin, propounded the theory that the *quantity* of heat generated during fever is also the same as in health, and that in fact pyrexia consists in a diminution in the amount of heat given off from the body. His theory has since been shown to be incorrect, but it is right to add that it formed the starting-point of very numerous observations and experiments, by which our knowledge has been immensely increased.

There would be no difficulty in attributing either a rise or fall in the temperature of the body to an alteration in the loss of heat, while its production remains unchanged. But in pyrexia, when the temperature has risen to a certain point, it is often stationary there for some hours; and for several days it may oscillate upwards and downwards, always remaining above normal; exactly as in health it oscillates above and below 98·4°. Now, it is clear that such a maintenance of a fixed high temperature is of itself fatal to Traube's doctrine as it would equally be fatal to any hypothesis that should attribute fever merely to an increased *generation* of heat. For, in order that the temperature should remain stationary, the amount of heat generated in a given period of time and the amount of heat lost in the same period must be equal. Thus it is certain that in fever, as in health, the regulation of temperature still goes on, although the point at which the thermometer stands is different. Liebermeister, in 1864, took advantage of an attack of tonsillitis in his own person to demonstrate this fact very clearly. His temperature having risen to between 102° and 103°, he exposed his body to cold air, and washed himself with cold water; and he found that this caused a slight rise of the axillary temperature from contraction of the superficial vessels and diminution of loss of heat from the skin, exactly as would have occurred under normal circumstances. The same observer, in 1868, by noting the changes in the temperature of the water of a cold bath, in which he placed a man suffering from acute pneumonia, was able to calculate that the immersion caused a greatly increased production of heat, far more heat being given off than would have corresponded with the mere lowering of the temperature of the patient.* So also, as Cohnheim remarks, one may throw a fever patient into the most profuse perspiration by the subcutaneous injection of pilocarpine, when the loss of heat from the cutaneous surface must necessarily be enormously increased, yet without causing any fall in his temperature.

* The accuracy of this calculation has since been disputed by Winternitz. "Der Einfluss v. Wärmeentziehungen auf die Wärmeproduction," 'Med. Jahrb. Wien,' 1872; and 'Virch. Arch.,' Bd. lvi, S. 181.—Ed.

Another striking illustration of the fact that the physiological process of heat-regulation is still in action during the course of pyrexia, is afforded by the persistence of the regular daily fluctuations of temperature to which I have already more than once alluded. Wunderlich, indeed, formerly thought that the range of these fluctuations was wider than in health, even when the fever was such as would be commonly called continuous. But Jurgensen and others seem now to have shown that they are identical with the normal fluctuations in every respect except that they take place at a higher thermometric level. In pyrexia, as in health, the rule is for the temperature to rise more or less constantly during the day, and to fall during the night. The minimum occurs at about 6 or 7 a.m., the maximum at about 6 p.m. The range of fluctuation appears to be generally as much as 1° C., or even a little more, so that it approaches 2° Fahr.

According to Liebermeister, these daily curves of temperature are probably due to the influence of muscular exertion and of food upon the body, notwithstanding certain obvious objections to such an interpretation of them. One of the objections is that healthy men work and eat to a much later hour in the day than 6 o'clock, p.m., after which the temperature begins to fall; this Liebermeister meets by what is termed the "principle of compensation," according to which every rise in temperature tends to be followed after an interval by a fall below the normal point; he supposes, in fact, that the power of food and of exertion to raise the bodily heat is exhausted during the early part of the day, after which the inevitable recoil takes place in spite of them. The other objection is that the daily fluctuations do not disappear when a person remains in bed all day, and takes no food. Liebermeister accounts for this by appealing to the influence of habit. Evidently the persistence of the fluctuations during pyrexia, when the patient is perhaps absolutely helpless and has fluid nourishment at regular intervals through the twenty-four hours, requires and admits of the very same explanation. It seems to me, therefore, that Liebermeister fully establishes the position which he takes up, namely, that pyrexia consists, not in a mere rise of the temperature of the body, still less in increase of heat-generation or in diminution of loss of heat, but in a change in the normal function of heat-regulation, by which the production of heat and its loss are so balanced as to create and maintain, while the pyrexia lasts, a higher temperature instead of the normal temperature. One might imagine the index of the regulating machinery to be shifted upwards, so that it is "set," not at 98·4°, but at 101°, 102°, 103°, or even a still higher point. Cohnheim, indeed, rejects this view, summarily and almost with contempt, evidently imagining that it contains only a verbal solution of the problem, but in reality (as I think) failing to appreciate what the problem is. Of course we are at once met by the difficulty that of the nature and seat of the function of heat-regulation we have no certain knowledge, although its existence is indisputable. Liebermeister's notion is that somewhere in the upper part of the cord there are two centres, more or less closely connected, but opposed in their action, one of which is termed by him the "excito-caloric" system, the other the "moderating" system. It must, however, be allowed that the stability of the process of heat-regulation in fever is by no means so great as in health.

The view that pyrexia is a definite modification of a physiological process accords well with a doctrine which is now gaining ground, and which seems likely to bear valuable fruit, namely, that this morbid condition fulfils a pur-

pose in the economy, comparable with that which we shall see to be fulfilled by inflammation. The conception of fever as having a salutary influence is, indeed, as old as Hippocrates, and it can be traced all through the middle ages, and down to our own time. But our immediate predecessors recoiled from all explanations that could be called "teleological." And it is only of late, since the contagious principles of some at least of the specific diseases have been shown to be living organisms, that it has been possible to understand how pyrexia may bring about its own cure, by destroying the very agents which set it up. In discussing Relapsing Fever, Ague, and Enteric Fever, I shall give evidence that this is the case.

The chief conditions under which pyrexia occurs, fall naturally into two groups: sometimes it is "symptomatic" or secondary to a local inflammation, sometimes it is "idiopathic" and "essential," and depends on the entrance of a contagion into the blood. Now, recent researches seem to lead to the conclusion that "symptomatic" fever may likewise serve the purpose of freeing the blood from living organisms. It was experimentally shown by Breuer and Chrobak that the division of the nerves of the limb of an animal has no effect in preventing the development of fever as the result of inflammation in it. The inference seems to be inevitable that inflammatory fever is the result of the entrance of a morbid agent into the blood from the inflamed tissues, and this entirely accords with the observations of Billroth and Otto Weber, who, so far back as 1864, showed that pyrexia could be produced by injecting into the blood either fresh pus or decomposing substances of various kinds. With regard to the nature of the "pyretogenous" material in symptomatic fever there is more uncertainty. At one time the prevalent view was that it is possibly excited by the products of inflammation when they have re-entered the blood either directly or through the lymph stream. But at present the more usual opinion is that local inflammations are themselves set up by the entrance of a poison from without, and that when this is the case the same poison making its way into the blood sets up the pyrexia.

One must, however, observe that the fact that a particular disturbance of a physiological function can, in certain cases, be shown to be for the benefit of the economy, is no reason whatever why the same kind of disturbance should not arise in other cases when no purpose is served by it. If there be centres for heat-regulation in the cord, nothing is more likely than that they should be modified in their action by cerebral hæmorrhage or the status epilepticus. And, on the other hand, there is sometimes an absence of pyrexia as an accompaniment of intense inflammations (especially of the meninges and of the peritoneum) which can hardly be accounted for except on the supposition that some counter-influence is exerted upon these centres, either directly or through the sympathetic or other nerves.

The irregular fever of idiopathic anæmia, of leuchæmia, and of Hodgkin's disease has not yet received a satisfactory explanation; nor has that which accompanies the various forms of acute tuberculous affections.*

According to Liebermeister's definition of fever, the mere fact that the temperature of the body is raised is not of itself a proof that fever is present. In Tetanus the temperature is sometimes exceedingly high. But Dr Parkes,

* I may take this opportunity of remarking that it is very advisable to give up describing "hectic" as an independent variety of fever; such peculiarities and symptoms as belong to the pyrexia of phthisis will be mentioned when that disease is under discussion.

in his Croonian Lectures for 1871, suggested that the heat of the body in that disease might perhaps have a different origin from that of the specific and symptomatic fevers. A similar opinion has recently been expressed by Cohnheim, who would also exclude from pyrexia the phenomena of "heat-stroke." In each case it is probable that what occurs is not a shifting upwards of the point to which heat-regulation is adjusted, but a generation of heat in excess of that with which the process of heat-regulation is able to deal. The same view may perhaps be taken of hyperpyrexia in general, the proof of its correctness being that the temperature never remains stationary at a very high point, and does not continue to oscillate backwards and forwards, but either goes on rising until death occurs, or falls again to a moderate level.

Physiological course of fever.—As we have seen, there is no ground for the belief that in pyrexia either the amount of heat generated or that which is lost is in any way fixed. Probably each of them may vary within very wide limits in different diseases, or in different periods of the same disease. All that is necessary is that they should constantly bear such a relation to one another, that the bodily temperature is at first raised to, and afterwards maintained at, a point more or less above the normal.

Now, in the *initial stage* of fever, there can be no question that the loss of heat from the cutaneous surface is greatly diminished. And as there is no reason whatever for supposing that the evaporation from the lungs undergoes a proportionate increase, we are probably justified in assuming that the total loss of heat is much less than under ordinary circumstances. Since the introduction of the thermometer into clinical practice, writers seem to have fallen into a most extraordinary mistake with regard to the surface-temperature during rigor. We have seen that, as De Haen long ago pointed out, the temperature of the deeper parts of the body is actually higher than normal, and in fact rises rapidly. Now, this has commonly been taken as proof of an altogether false conclusion, namely, that the coldness of the limbs and of the skin generally, which gives the patient so much suffering, is merely a "subjective sensation produced by the state of the peripheral nerves." Of course the truth is that the low temperature of the surface is just as much a physical fact as the high temperature of the blood in internal organs. One has only to feel the shrunken hand of a man in the cold stage of ague to be satisfied that he is under no illusion when he complains of being cold, and a surface thermometer affords precisely the same indication by the slowness with which it rises above the temperature of the surrounding air, and the comparatively low point which it reaches, however much time may be given to it. Thus Schülein, among certain observations of surface temperatures to which I shall presently allude, gives a case of tertian ague; and in his chart it is most striking to notice, how, during each attack, at the precise moment when the temperature in the axilla was rising from 98° to 104° or 105° F., that between the toes fell still more sharply, from 95° to 86°, or even below 77°. The cause of the chilliness experienced by such a patient is that the peripheral arteries are contracted and do not allow of the passage of a sufficient quantity of blood to warm the superficial tissues, and to compensate for the loss which is always going on by radiation and conduction, to whatever extent the exhalation of fluid from the skin may be diminished. A necessary consequence is a great fall in the temperature of the skin, and one must not forget that this in turn involves a lowering of the amount of heat-loss, which affords one of the means by which the rapid rise

of temperature during rigor is brought about. It has, however, been caculated by Zimmermann and by Liebermeister that there is, besides, a very great increase of heat-production during this stage.

During the *fastigium* the temperature of the body may remain stationary. or at any rate undergoes comparatively gentle oscillations, so that for its maintenance no such antagonism between heat-production and heat-loss is required as for the rapid rise in the initial period. However, in many instances there is still a marked distinction between the temperature of the skin and that of the deeper structures. These cases afford special proof of the importance of the clinical thermometer, inasmuch as the axillary or the rectal temperature is found to be above normal while the patient's skin feels cool to one's hand and he has no sensation of feverishness. But of course the comparatively low temperature of the surface is a reality; and since the heat-loss by conduction and radiation is certainly less than when the skin is hot, it is fair to conclude that for the same degree of internal temperature the production of heat must also be less. On the other hand, there are certain diseases in which, when one places one's hand upon the patient's skin, one feels it to be pungent and burning in a very remarkable degree. Addison used to teach that in acute pneumonia the skin possessed a heat which was not observed in any other disease except perhaps scarlet fever. And I well recollect how disappointed I was when the thermometer was introduced into practice to find that his dictum seemed to have no physical foundation. An attempt was made to explain away the difficulty by saying that the surface was peculiarly dry in acute pneumonia; but this obviously was beside the question, since at any temperature above that of one's own hand a moist skin must undoubtedly feel hotter than a dry one, if it were possible to prevent the temperature from being lowered by the evaporation which inevitably occurs. The true solution of the difficulty is that in acute pneumonia the temperature of the surface is maintained at a point which is very nearly as high as that of the deeper parts, instead of being far below it, as in most other diseases, even when there is no moisture upon the skin. This has recently been established by Schülein, as the result of a series of careful observations published in 'Virchow's Archiv' for 1876, upon the relation between surface temperature and internal temperature in various diseases. His method was to insert a thermometer with a very small bulb between the first two toes, fastening them together by means of an elastic ring. And he found that whereas in other diseases (including enteric fever, acute rheumatism, and phthisis) the instrument in this position always indicated a much lower temperature than one placed in the axilla, there were three diseases in which the difference was very slight indeed, namely, acute pneumonia, measles, and scarlet fever. In these almost every movement of the axillary temperature upwards or downwards was accompanied by precisely the same movement of the temperature between the toes; whereas no such relation could be traced between the two sets of observations in any other febrile complaint. It must, however, be remarked that in many diseases surface temperatures are from time to time taken which are absolutely as high as those in acute pneumonia; and it is probable, as I have taught for a long time in my lectures, that a still more striking confirmation of Addison's doctrine would be obtained if we were only to note the rapidity with which a surface-thermometer rises, when applied to the skin in different complaints. For in that way we should be estimating the amount of heat which the surface is giving off by conduction; and it is this that one's hand appre-

ciates when laid upon a patient's skin. I have no doubt, however, that at least one other disease would have to be added to those enumerated by Addison, namely, acute rheumatism when complicated by hyperpyrexia. It is evident that in order to maintain such a uniform high temperature of the superficial as well as of the deeper parts, in spite of the great loss of heat which must necessarily occur, the generation of heat must be far greater in all these complaints than in others in which only the interior of the body is raised to a similar degree of heat. So, also, when the skin perspires freely during the *fastigium*, the temperature within remaining high, there can be no doubt that the production of heat must be proportionately increased.

Of the stage of *defervescence*, I have only to remark, that the profuse sweating which so often accompanies it, when crisis takes place, plays an important part in bringing about the rapid fall of internal temperature. But it is not to be regarded as the cause of the defervescence; the fact being that the state of pyrexia has come to an end, and that the heat-regulation is, therefore, no longer set for an abnormally high point.

Concomitants of fever: tissue-change and wasting.—Liebermeister's theory of fever, which I have adopted, renders it almost unnecessary to enter into any detail as to the sources of febrile heat, these being in fact identical with those which are enumerated in works on physiology as maintaining the normal temperature of the body. But I must not omit to point out that science has now advanced beyond the point at which Virchow stood when, in 1854, he declared that the elevation of temperature in pyrexia "must arise from an increased tissue-change." Liebermeister and other observers have indeed shown that the amount of urea excreted in the urine during fever surpasses by at least 70 per cent. that which is voided by a healthy person living on the same diet. But it is now well known that the production of heat may be largely dependent upon an increased oxidation of various substances, such as sugar, which have never formed part of the substance of the body. And both Leyden and Liebermeister have proved that the excretion of carbonic acid gas also is excessive in fever, the increase amounting probably to at least 50 per cent. It is essential to bear in mind, however, that neither the quantity of urea, nor that of carbonic acid, is nearly so great as that which may be discharged without any rise of temperature by a person who is taking active exercise, or even by one who has eaten a large meal of animal food with much fat. Thus we are brought back again to the same point as before, namely, that pyrexia cannot be due to any special rate of heat-production, but must depend upon an alteration in the point for which heat-regulation is set.

There is, however, no more striking feature of pyrexia than the wasting of the body which accompanies it; and this often seems to go on quite as rapidly as one could have expected, even if Virchow's dictum had been true in the strictest sense. Formerly there was a tendency to pay too little attention to fever as being, in itself, a cause of loss of weight, and to ascribe this result rather to a drain of pus, or of any other discharges which might occur in a particular case. It may therefore be worth while to cite a case of Liebermeister's in which a patient who had an abscess discharging about three ounces of pus daily, lost from fifteen to twenty pounds in weight during two months, while his temperature was at 101° or 102°, but regained it afterwards when he had no fever, notwithstanding that the suppuration still went on as before. No doubt an inability to digest food plays an im-

portant part in bringing about the wasting; it is well known that the secretion of milk ceases during pyrexia, and one may fairly assume that there is a similar interruption in the formation of saliva (to which, in part, the dryness of the mouth is due), and also of the gastric and intestinal juices. That it is not merely a question of loss of appetite appears probable from an observation of Niemeyer's, who, comparing together a healthy man and one suffering from fever during a period of two days in which they lived on exactly the same food, found that the latter lost much more weight than the former. Liebermeister remarks that the wasting is sometimes concealed by the presence of an excessive amount of water in the tissues: he has observed that from this cause the weight of the body may be raised by as much as ten pounds, although there may be only a very slight œdema of the ankles to indicate the presence of such an abnormal condition. He believes that persons who are in good health and well nourished invariably lose flesh when they become the subjects of pyrexia; but he admits that those who are already wasted may undergo no further emaciation, and may even gain weight in spite of the supervention of a febrile illness. According to a calculation made by Leyden, the daily loss of substance in fever amounts on an average to 7 parts per 1000 of the whole body-weight. But, as Cohnheim has pointed out, if this be correct, it should take only about eight weeks of a tolerably severe fever to kill a fairly-nourished man by the mere destruction of his tissues; at least, if one may appeal to the observations of Chossat, which showed that to the higher animals a loss of 40 per cent. of their weight is directly fatal.

The blood doubtless wastes simultaneously with the solid structures, and Parkes and others have supposed that the excess of pigment in the urine depends upon an undue disintegration of red discs. But hæmometric observations which have been made by Drs Baxter and Willcocks, lead to the conclusion that, with a single exception, febrile diseases cause no diminution in the proportion, either of discs or of hæmoglobin, in a given amount of blood. The exception is ague, in which Kelsch is said to have demonstrated a very great decrease of corpuscles. In all other febrile states the whole volume of the blood is lowered, while the relation of its fluid or solid constituents remains normal. If so, a point on which Dr Parkes used to insist can hardly be maintained, namely, that there is a special retention of water in the body during fever.

Increased rapidity of pulse.—We have seen that it is only of late years that elevation of temperature has been recognised as the characteristic and essential feature of pyrexia. Previously, acceleration of the pulse, rapidity of breathing, diminution of the secretions, and a variety of other phenomena, were supposed to be not less important indications of it. Now, it is a matter of some consequence to determine how far these several symptoms are directly dependent upon the increased heat of the body, or whether they must be regarded as independent effects of the various causes of fever. And in the opinion of the best modern writers, the rapid pulse which generally accompanies pyrexia is a result of the heightened temperature; Cohnheim adduces in support of this view the well-known fact that the frog's heart beats more quickly in proportion to the warmth of the surrounding air; Liebermeister insists on the effect which hot and cold baths respectively have, when they raise or lower the temperature of the body, of simultaneously accelerating or slackening the pulse. Liebermeister even lays down as the rule, based upon a considerable number of observations (which, how-

ever, vary widely among themselves), that for each degree Centigrade above the normal temperature there should be a rise of the pulse by eight beats above the standard rate of eighty. It is admitted, of course, that various other circumstances may affect the pulse-rate of febrile patients, as of healthy persons, or of those suffering from other diseases. Cohnheim insists on the effect which stimulation of the vagi may have in slowing it, and cites the fact that in basilar meningitis the pulse is often infrequent in spite of high fever. But neither he nor Liebermeister seem to have looked out for those negative instances by which their theory could best be brought to the test, namely, the exceptional cases in which a severe inflammatory process, or some other condition that usually gives rise to pyrexia, happens to run its course without elevation of temperature. Such instances are sometimes met with in peritonitis, in diphtheria, and in meningitis; and I think that the pulse is often, though not always, quickened. Does not this fact, if it be one, cast some doubt on the view which the German authors adopt?

Other febrile symptoms.—The increased frequency of the *breathing* in pyrexia is also attributed to the action of heated blood upon the respiratory centre; and this view is supported by a very interesting experiment, performed by Goldstein in Fick's laboratory, of warming the carotid blood alone; he found that the respiration was thereby accelerated. Cohnheim adduces in illustration of this theory, the fact that in acute pneumonia the breathing ceases to be rapid after the crisis of the fever, notwithstanding that the affected part of the lung still remains hepatized.

On the other hand, this writer is indisposed to admit that the *cerebral symptoms* which attend so many cases of fever are dependent solely upon the increased temperature of the blood and of the substance of the brain. He points out that in relapsing fever the thermometer rises to a great height with but little disturbance of the sensorium, and that in febrile diseases generally no relation can be traced between the intensity of the fever and the severity of the delirium or stupor. He does not even allow that the question is settled by the marked effect of a cold bath in restoring the mental faculties of a patient suffering from fever; for, he observes, it may be a consequence rather of the improvement of circulation which results from the bath.

It is important to observe that the rapidity with which the blood flows in pyrexia, and the arterial *blood-tension*, are by no means proportioned to the acceleration of the heart's action. At the commencement of fever, when the pulse feels full and hard, it is probable that the circulation is often really more active than in health. Much stress was formerly laid upon the "*sthenic*" character of such cases. In the 'Med. Times and Gaz.' for 1873, Dr Mahomed has given sphygmographic tracings of the pulse in various forms of fever, showing that this term is not without meaning; for in certain cases the vessels are full, the arterial tension is high, and the pulse bears considerable pressure. It is therefore a mistake to say that a febrile pulse is necessarily dicrotic. But, on the other hand, when pyrexia has lasted for any length of time, it almost always assumes an "*asthenic*" type, and in many instances this is the case from the very first. It would then seem that the circulation is more sluggish than natural, one proof of this being that the blood tends to stagnate in the more dependent parts of the body, so that what are termed hypostatic congestions arise in the lower lobes of the lungs, and beneath the integument of the back, of the gluteal region and elsewhere. The pulse still remains quick, but it is now soft,

feeble, and often small. The sphygmograph shows a highly characteristic condition of arterial relaxation, with low pressure; dicrotism becomes a prominent feature; and in many cases the pulse is even hyperdicrotic. In fact at this stage of fever the heart, although its beats follow one another so quickly, empties itself incompletely and discharges less rather than more blood into the arteries. Often, indeed, thrombi form in those parts of the heart's chambers which are farthest from the orifices; but this is a matter into which I shall have fully to enter hereafter.

Until recently, the impairment of the cardiac beats was held to be sufficiently explained by the fact that its muscle, like all the other tissues, becomes badly nourished as pyrexia goes on. Of late, however, some observers have thought that the cause of the enfeeblement of the heart's action in pyrexia is not merely its wasted condition, but the occurrence of definite morbid changes in its texture. When I come to speak of enteric fever I shall have to describe an affection, known as "parenchymatous degeneration" or "cloudy swelling," which is found in the liver, in the kidneys, in the heart, in the voluntary muscles, and elsewhere. This, however, is by no means peculiar to any one febrile disease, and Liebermeister and others have maintained that it is the direct result of elevation of the body-heat, reaching a certain degree of intensity, and protracted over a somewhat long period of time. This view is supported by the experiments of Dr Wickham Legg, who found ('Path. Trans.,' vol. xxiv) that the liver, the heart, and kidneys were granular in animals killed by the action of heat, even when its duration was only for a few hours. On the other hand, Cohnheim maintains that, although guinea-pigs kept for several days in a high temperature exhibit a fatty change in their tissues, there is nothing really resembling "cloudy swelling." And he insists on the absence of such an appearance in acute pleuro-pneumonia, and its occasional presence when there has been no fever (as in carbonic oxide poisoning) as proofs that Liebermeister's opinion is erroneous. He even declares that the statements as to the frequency of degenerative changes in febrile diseases are overdrawn, and that in a large majority of the bodies of those who have had intense pyrexia, fatty gland-cells and muscle-fibres are not at all more common than they would have been if death had taken place in a different way. The question is one of great importance, and bears upon the supposed causes of cardiac dilatation and of permanent failure of the muscular substance of the heart.

I shall not attempt in this place to discuss the *treatment* of pyrexia by baths and "antipyretic" remedies. Enteric fever and acute rheumatism afford by far the most frequent occasions for their employment, and it will be more convenient, when I am describing these diseases, to go into the details which are necessary.

Subnormal temperature.—It is an interesting question whether the temperature of the body ever undergoes a change in the direction opposite to pyrexia, the heat-regulation centre being set at a point below the normal instead of above it. I shall hereafter have to cite many instances in which a thermometer placed in the axilla remains unduly low, but probably in these cases the internal organs generally maintain a temperature nearly as high as ever, at least until death is obviously impending. Cohnheim says that this is the case with those who suffer from inanition as the result of

stricture of the œsophagus, from diffused paralysis, or from extreme anæmia. And even were it not so, we should still have to show that an alteration in the heat-regulation had occurred, and that the failure to maintain the normal temperature was not a result of an insufficient production of heat. Such a lowering of temperature by "*force majeure*," as Liebermeister terms it, is well seen in persons who are picked up insensible in the streets during cold weather. Cohnheim alludes to cases of this kind, in which the thermometer in the rectum has not risen above 86°, 79°, or even 75° F. The pupils are then dilated and sluggish, while the pulse and the respiration are greatly reduced in frequency; but unless things have gone too far for recovery to take place, such patients under suitable treatment regain their normal temperature within a few hours, and sometimes pass into a condition of reactive pyrexia.

INFLAMMATION

Historical review: Virchow: William Addison, Waller and Cohnheim: Goodsir—Direct observation of inflammation in animals—Redness, swelling, pain, heat, pyrexia—Exudation: membranous, catarrhal, purulent, diphtheritic, gangrenous inflammation—Repair—Causes of Inflammation: irritants and predisposition—Chronic inflammation.

No one, at whatever period of the world's history, could have applied himself to the study of disease, without seeing that the hot, painful, reddened state of the eye which follows the entrance of a particle of dust within the lids, is typical of a morbid change that is of frequent occurrence in every part of the body accessible to observation. Hence it is not at all surprising that before the end of the second century we find, in the writings of Galen, inflammation (φλεγμονή) already characterised by its four cardinal symptoms, which for so many generations since have been known as *calor*, *rubor*, *tumor*, *dolor*. To use the words of the late Prof. Hughes Bennet, inflammation has been in all ages "the pivot upon which medical philosophy has revolved." But, for that very reason, there is little use in our now attempting to recapitulate the different views and theories with regard to it which have been proposed. It is scarcely too much to say that only the most recent advances of pathology and histology have rendered possible a real understanding of inflammation.

Very soon after the promulgation of Schwann's doctrine, in 1839, that nucleated cells constitute a principal element of animal structures, pathologists discovered the presence of these bodies in inflamed parts; but at first it was supposed that they developed themselves in the interstices of the tissues by molecular aggregation, in accordance with the revived opinion that this was a common mode of origin for cells in general. In his 'Cellular Pathology,' however, published in 1858, Virchow laid down as a dogma, *omnis cellula e cellulâ*. And upon this foundation he erected a complete theoretical system, assuming that the cells of the connective tissues throughout the body undergo multiplication or "proliferation," so as to generate the "exudation-cells" or "pus-cells" of inflammation.

Even before Virchow's views were made known, certain observations had already been recorded in this country, which, although they at first attracted little notice, nevertheless involved conceptions as to the real nature of cells that have since proved to be more accurate than his. But the first shock to the edifice which Virchow had so carefully erected was given by one of his pupils, Recklinghausen. He showed, in 1863, that pus-cells, instead of being rounded bladders, each with its cell-wall and its nucleus, are during life solid masses of gelatinous substance, which are constantly changing their form,—now throwing out delicate processes, and now drawing them in again,—and that they actually possess the power of moving from place to place. Of course this discovery at once deprived Virchow's proliferation-theory of the impregnable position which it had seemed to hold as a necessary consequence of the law *omnis cellula e cellulâ*. Four years later, in 1867, Cohnheim, who

also was a pupil of Virchow, pointed out that in the mesentery of the frog, when it becomes inflamed under exposure to the air, the white corpuscles of the blood can, with the aid of the microscope, be distinctly seen to make their way through the walls of the smaller vessels, so as to become pus-cells. This very fact, however, had been demonstrated as far back as 1846 by the late Dr Augustus Waller, then in practice at Kensington. His investigations were made on the tongue of the frog. Still earlier, in 1843, Dr William Addison, of Great Malvern, had insisted on the transformation of the colourless corpuscles of the blood into pus-cells, and described the former not only as "lining the vessels" in the web of the frog when inflamed, but also as "lying among the fibres forming their walls and exterior to their boundary." The resuscitation and general acceptance of views which for so long a time were neglected, should make us cautious in rejecting any statements that are based upon direct observation, however much they may clash with our preconceived opinions.

In those countries in which Cohnheim's experiment is not forbidden by law, it is performed in the following manner :—A male frog, which has been rendered quiet by curare, has an incision made into the abdominal cavity, large enough to allow a loop of intestine to be drawn out, with its mesentery. It is then placed upon its back on a broad glass plate, which has been fitted with a thin glass disc for the convenient reception of the mesentery, and with a semicircle of cork to which the intestine is to be pinned. Under the microscope, the circulation can now be seen to go on naturally. But the contact of air appears to act as an irritant, and inflammation soon sets in. In from fifteen to twenty minutes the arteries are observed slowly to dilate, until at the end of an hour or two their diameter is nearly twice as great as before. A little later, a similar change begins in the veins. At first the blood-stream perhaps flows more quickly than before, but very soon its rapidity begins to diminish, so that the oval red blood-discs can be distinctly seen in the arteries, at least during diastole. A striking change is now noticeable in the veins. Leucocytes begin to fall out of the middle of the current and loiter against the sides of the vessel, rolling over and over, and sticking from time to time, when their soft substance yields to the pressure of the fluid upon them so that they become pear-shaped. Before long, the channel of the veins becomes lined by a thick layer of these bodies, which lie at rest, while the central current of red discs goes on as before. In the capillaries, too, the leucocytes are arrested here and there; but between them there are seen many red discs also adhering to the walls. The most wonderful thing of all is that outside the contour of these vessels there gradually appear a number of minute rounded bodies, each of which is connected with a leucocyte within. Thus the leucocytes may be said to have assumed a dumb-bell form. By an imperceptible process, the extra-vascular part keeps growing bigger, and the intra-vascular part smaller, until at length the latter altogether disappears; whereupon the leucocyte resumes a more or less globular shape, and now lies close to the vein, but outside it. The result is, that large numbers of leucocytes are soon collected not only in the immediate neighbourhood of the vessels, but in all the interstices of the surrounding tissues. At the same time a quantity of fluid exudes, which reaches the free surface of the mesentery and coagulates there, forming a membranous layer, itself thickly set with leucocytes.*

* The important fact of the white corpuscles adhering to the sides of the vessels in the web of the frog's foot had been observed and figured by Dr C. J. B. Williams, in his 'Prin-

The separation of the leucocytes from the red discs flowing through the mid-channel of the veins, and their collecting against the walls of these vessels, are direct physical results of the slowing of the blood-current. The process of *emigration* must be referred to the power of spontaneous locomotion possessed by the leucocytes themselves. It seems, however, to depend to a certain extent upon their being under pressure from the blood, for Cohnheim has observed that it instantly ceases when the flow of blood through the main artery of the part is arrested. But, on the other hand, it is not due solely to blood-pressure, for this is actually less than under normal conditions where no emigration seems to go on, at least in the case of tissues which are not in an embryonic state.

My colleague, Mr Laidlow Purves, and Prof. Julius Arnold of Heidelberg, have maintained that the points at which leucocytes emerge from the vessels are always situated in the meeting lines between the endothelial cells. But Cohnheim argues that at any rate actual orifices or stomata can hardly be present, or they would give exit also to an unaltered liquor sanguinis, whereas the fluid which exudes during inflammation is of different chemical composition, and contains less solid matter in solution. It has been suggested that emigration may be considered to take place without any breach in the vascular walls, if they and the leucocytes be regarded as colloid substances, which become temporarily continuous with one another; and I am one of those who think that this view is fairly illustrated by the experiments of Dr Norris, who has shown that if a film of soapsuds be stretched across an iron ring, a moistened glass rod, or even a spherical soap-bubble, can be passed into it on one side and brought out on the other, leaving it unbroken.

Before the time of Virchow, a favourite topic of speculation had been whether parts into which no vessels penetrate could be said to undergo inflammation. And one of the merits of his system was that it placed the question on its right footing by showing that the distinction between vascular and non-vascular tissues was after all only one of degree, and that a minute islet of liver-substance ought to be regarded as being outside the blood-current, no less than the cornea or the cartilage of a joint. Nor did Cohnheim's investigations modify this conclusion, but rather carried it a step further; for he actually proved that leucocytes could penetrate into the interior of the cornea from the blood. It had been shown by Von Recklinghausen that when finely divided vermilion was injected into a lymph-cavity in the frog, the leucocytes would take this substance into their interior, or "feed" upon it; and Cohnheim found that after injecting pigment into one of the aortæ many of the leucocytes which appeared in the cornea under inflammation were coloured, so that they had evidently been derived from the circulatory fluid. Still, however, it could be maintained that others of them were formed by proliferation. This view was, and is still, vigorously upheld by Stricker, who described the fixed corpuscles of the corneal tissue as breaking up into amœboid masses, containing bodies which he believed to be the germs of future pus-cells. But other observers afterwards pointed out that by more

ciples of Medicine' (1843). Mr Wharton Jones had detected the same phenomena and accurately described the dilatation of the vessels and stasis of the blood ('Guy's Hospital Reports,' New Series, vol. vii, 1850), and Mr (now Sir Joseph) Lister had confirmed and extended these observations by his own (also carried out on the web of the frog's foot) in the 'Philosophical Transactions' for 1858. One of the important points established by Lister was that the dilatation of the arteries depends on paralysis of the vaso-motor nerves. —Ed.

refined histological methods the stellate corpuscles could be seen in a cornea which was already full of leucocytes; and although it seems to be a fact that their neuclei do undergo division, this is probably an indication (as Dr Thin has suggested) of commencing disintegration rather than of germination. The observations which Cohnheim, writing in 1877, deemed most worthy of notice, as running counter to his own, were some which had been made by Böttcher. He cauterised the minutest possible spot in the centre of the frog's cornea, and showed that pus-cells were to be found in the neighbourhood of the injured part, whereas there was no marginal opacity such as would have indicated that they had come from the conjunctival vessels. Cohnheim's rejoinder was that they might have been derived from the fluid secretion which covers the free surface of the eyeball. And this, which at first seemed to me a very far-fetched solution of the difficulty, appears to have been since established by the experiment of placing a dead cornea, in which a puncture has been made, into a healthy conjunctival sac for a few hours; "it remains transparent," says Dr Sanderson in his Lumleian Lectures for 1882, "everywhere excepting in the neighbourhood of the injury," so that a breach of surface is evidently all that is necessary to enable leucocytes from the conjunctival fluid to make their way into corneal tissue. It has also been shown that a limited part of the cornea may be destroyed without any opacity of the adjacent tissue resulting, if the caustic employed be chloride of zinc, which does not disintegrate the protective epithelial covering.

To me it seems impossible to obtain an adequate conception of inflammation, without taking into account its relation to the physiological process of repair. By Dr Sanderson himself it is defined as the "physiological effect of the damage done to a tissue when it is injured." He, however, goes on to say that its "essential nature is fully expressed in the word *damage*," and I do not think that he anywhere suggests that a useful purpose is served by the emigration of leucocytes. Yet to call a process physiological is surely to imply that it is likely to be beneficial to the organism rather than the reverse. Pathologists have indeed been accustomed to describe the healing of wounds and the restoration of the substance of injured parts, as not only independent of, but even opposed to, the occurrence of inflammation in the same structures; and so long as the pus-cells found in an inflamed area were supposed to be formed by the breaking up of the fixed elements of the tissues in proliferation, it might well seem that such a process was altogether different from the suppuration which was described as constituting one factor of "repair by granulation." But now that the leucocytes are believed to be always derived from the blood, it is obvious that no such distinction can be drawn.

The solution of the difficulty seems to me to lie in the fact that when a part is at all severely injured, the removal of damaged tissue-elements is an essential preliminary to the work of reconstruction and repair. I have for some years past ventured to teach that the emigration of leucocytes in inflammation serves this especial purpose of clearing away such portions of an injured structure as are no longer fit to remain. We have seen that leucocytes are capable of taking up granules of vermilion; and it is well known that they also "feed" upon milk globules, or particles of myelin, when those objects are placed in their way. May we not assume that they can also remove damaged cells or fibres? I did not know, until recently, that this very doctrine was promulgated, forty years ago, by the late Prof. Goodsir. In his 'Anatomical and Pathological Observations'

(1845) speaking of "a rapidly extending ulcerated surface," he described "a peculiarly endowed cellular layer, which takes up progressively the place of the subjacent textures;" and a little further on he attributes the separation of a dead portion of bone to the "cells of the newly-formed cellular mass, contained in the Haversian canals . . . . taking it up as nourishment, and substituting themselves in its stead."* The same explanation is obviously applicable to the detachment of a slough in the softer tissues, and perhaps even to the formation of an abscess-cavity, and to the thinning of the roof of an abscess which is about to "point." We may also have recourse to it when we find that, in certain parts of a solid organ, a dense infiltration of leucocytes is attended with disappearance of the normal structure, although there is no obvious gap or breach of continuity; this, for example, may often be observed round the Malpighian capsules of the kidney, when it is in a state of inflammation.

Such a hypothesis seems to me far more satisfactory than the view of Cohnheim, who appears to place the inflammatory process on a merely physical basis; ascribing it to a molecular change in the walls of the blood-vessels, wrought directly by the irritant which sets up the inflammation. In support of this view he cites the experiments of Winiwarter, who found that the vessels of an inflamed part allowed a solution of gelatin to transude through them under a lower pressure than under normal conditions. But I think that the change in question is only a part of the general softening of all the tissues, which has long been known to constitute one of the phenomena of inflammation. An acutely inflamed bone is, as Paget points out, so soft that a knife will easily penetrate it; the ligaments of an inflamed joint yield, allowing the bones to be displaced and distorted; and we are accustomed, in the dead-house, to regard a diminished resistance of the texture of a lung as one of the best rough tests of it having been the seat of acute pneumonia.

Before we quit the study of inflammation in cold-blooded animals, I have to mention some further phenomena which belong to its more severe forms. When croton-oil is smeared over the tongue of a frog, the superficial capillaries pass into a condition which has long been known as *stasis*. It consists in complete arrest of the circulation through them, with coagulation of the blood which they contain; and it corresponds with the condition which in larger vessels is called *thrombosis;* in fact, the two are often associated together. But a still better way of observing severe inflammation is to apply caustic to a circumscribed spot on the tongue of the frog. This produces what may be described as a series of concentric zones. In the middle all the tissues are killed; or, in other words, an *eschar* is formed. Next comes an area of absolute stasis, in which, according to Cohnheim, the vessels alone are destroyed. Further outwards, the blood-stream is almost stagnant, and the capillaries are choked with red discs, which escape from them in large numbers. Beyond this again the emigration of leucocytes is the principal change. Last comes a zone in which the vessels are merely dilated, while the circulation through them is somewhat retarded. However, it is not clear to me that stasis has been proved always to depend upon the occurrence of alterations in the vascular walls, exceeding in intensity those in the other tissues. At any rate, no such conclusion can fairly be

* See pp. 404, 406 of the second volume of Goodsir's Memoirs, republished by his successor Prof. Turner, of Edinburgh, in 1868. Also Prof. Redfern's papers on ulceration in articular cartilage, 'Edin. Monthly Journal,' Sept., 1851.—Ed.

drawn from the well-known experiments made by Ryneck, of Gratz, in 1870. He showed that stasis could be set up by irritation in the web of a frog, after defibrinated mammalian blood, or even milk, had been substituted for the natural circulating fluid of the animal; but that, on the other hand, when a solution of chromic acid or sulphate of copper had once been passed through the blood-vessels, even for a few moments, no such effect could be produced. But these observations, although they disproved the now obsolete doctrine that inflammation is dependent upon a change in the blood, appear to have little or no bearing upon the questions which are under discussion at the present time.

In warm-blooded animals the attempt to watch the inflammatory process directly with the microscope was at first found to be attended with considerable difficulties. But these were gradually overcome. Mr Wharton Jones had observed in the bat's wing that the vessels when inflamed dilated, that the blood-stream slackened, and that the corpuscles crowded together to the sides of the vessel ('Med.-Chir. Trans.,' 1853). Mr (now Sir James) Paget, in his 'Lectures on Surgical Pathology' published in the same year, also used the bat's wing, and observed the very short period of initial contraction, the subsequent dilatation, and other phenomena as afterwards described, excepting the emigration of leucocytes. In 1870 Dr Sanderson and Professor Stricker, of Vienna, contrived an admirable method of studying the circulation in the mesentery of the guinea-pig, which was demonstrated at the meeting of the British Association in that year at Liverpool, and this method was afterwards applied to the observation of inflammation. Lastly, Professor Thoma published in 'Virchow's Archiv' for 1878 a series of microscopical observations on inflammation in the mesentery or the omentum of dogs, cats, rabbits, and guinea-pigs. The only difference between his results and those described above seems to be that he could not satisfy himself that the calibre of the arteries became wider. Emigration of corpuscles and exudation of coagulable fluid occur in precisely the same way as in frogs.

The phenomena of inflammation.—I may now proceed to trace out in detail such explanations of the four cardinal symptoms of inflammation as accord best with our present views; and in doing so I shall follow Cohnheim closely.

1. *Rubor.*—The redness of inflammation depends partly upon the dilatation of the veins and other blood-vessels of the affected area*, partly upon the accumulation of blood-discs in its capillaries. If the separate vessels are visible, the redness is spoken of as "injection;" if they cannot be seen, it is said to be "diffused." The tint is somewhat purple, which seems to be due not only to the preponderance of dilated veins, but also to an augmented deoxidation of the blood in the capillaries, in consequence of the retardation of the current through them. When stasis occurs, this also causes redness; and at all but the earliest stages hæmorrhages also play an important part in the production of rubor. They often give to it a punctiform character, and they may be further distinguished by not fading beneath the pressure of the finger. On the other hand, the presence of any considerable quantity of exudation tends to conceal the red colour, even where it does not actually compress and empty the vessels: thus, at advanced periods of the morbid process, the affected tissues often become grey or even yellow. When non-

* According to Prof. Thoma, in mammals the veins only dilate, not the arteries also, as in frogs ('Virchow's Archiv,' 1878).

vascular structures become inflamed, the redness of course shows itself in the vascular parts around, from which they derive their nutriment; for instance, in the conjunctiva at the margin of the cornea, and in the synovial membrane about articular cartilages. After death, so much of an inflammatory redness disappears as was due to fluid blood in the interior of the vessels.

2. *Tumor.*—This may, to some extent, be due to vascular turgescence, but the chief cause of it is exudation. Lassar, Cohnheim's assistant, showed some years ago that the stream which flows away through the lymphatics of an inflamed part is greatly augmented. For example, he irritated the paw of a dog, sometimes by immersing it for a short time in hot water, and sometimes by rubbing in croton-oil; and he found that the lymphatics in the limb above became greatly dilated, and that when he introduced a cannula into one of them the fluid which escaped through it was increased to several times its normal quantity. Evidently, therefore, the swelling corresponds, not with the whole amount of exudation, but with the excess of it, as compared with what is taken up again. Cohnheim even holds that inflammation may run its course, and be attended with exudation and with the emigration of leucocytes, and yet lead to no swelling, provided that reabsorption goes on at a proportionately rapid rate. He suggests, for instance, that this may be the case with the eruptions of scarlet fever, and other exanthemata, in some of which the subsequent occurrence of desquamation of the cuticle shows that there was more than a mere hyperæmia. In describing erysipelas I shall mention what is perhaps a more striking example of the same thing.

In distributing itself through the inflamed structures, exudation follows the lines of least resistance. In connective tissue, and in the stroma of a parenchymatous organ, it fills the natural interstices. Wherever there is a gap or interspace of any size it accumulates. It covers the free surfaces of serous membranes. Mucous membranes throw it off, to mix with their natural secretions; this, for example, is one reason why the urine becomes albuminous in nephritis. In the skin, it penetrates through the deeper soft layers of the cuticle, and it often raises the horny layer to form small collections of fluid, which are termed vesicles and pustules.

3. *Dolor.*—The pain of inflammation is usually, and no doubt correctly, attributed to the compression of the sensory nerves by distended vessels and afterwards by exudation. Thus it often has a throbbing character, being momentarily intensified by each pulsation of the heart. And it is generally accompanied by "tenderness;" or (in other words) it is increased by pressure from without. Doubtless a general relation can be traced between the amount of pain induced by inflammation in different parts and the abundance of their sensory nerves; but, on the other hand, it is well known that some tissues, such as ligaments and bones, may become exceedingly painful when inflamed, although they are not naturally very sensitive. The determining factor in such cases appears to be the degree of resistance which is offered by the structure inflamed to distension and swelling.

4. *Calor.*—That those parts which are most accessible to observation become hotter than natural when inflamed, is matter of common knowledge; anyone may satisfy himself of it by placing one hand over an acutely swollen joint or a recently formed abscess, and the other upon the corresponding spot on the opposite side of the body. But contradictory statements have been made as to the relation between the temperature of an inflamed part and that of the blood and of the deep internal organs.

In 1860 Mr Simon made a series of observations with a small thermo-electric apparatus, so shaped that it could be thrust like a pin into the soft tissues, or even into the interior of large vessels; and he found not only that the arterial blood flowing towards a severely injured limb in a dog was always less warm than the venous blood flowing away from it, but that the inflamed tissues themselves were warmer than either kind of blood. But he did not attempt to measure the amounts of difference of temperature which he believed that he detected; and if (as is possible) they were very small, there may be the less difficulty in supposing that there was some source of error in his experiments. However, it appears to be certain that he was wrong in concluding that inflammation is attended with a special local production of heat. For Jacobson, of Königsberg, who afterwards investigated this question with more accurate apparatus, found ('Virchow's Arch.,' 1870) that the temperature of the skin and of the muscles down to the bone never, even under conditions of the most intense inflammation, reaches that which exists in the rectum, the vagina, or the peritoneal cavity, at the same time. The difference was generally as much as 2° or 3·5° Fahr. And as for the relation between the temperature of one inflamed part and that of the opposite side of the body, the difference was always less in proportion as the distance from the surface was greater. Thus, whereas a rabbit's ear when inflamed was warmer than the other ear by 7° or 8° Fahr. the temperature of the deep muscles of a limb under the same circumstances seldom exceeded that of the same structures on the opposite side by more than 1°. It would follow that the internal organs under inflammation continue to be of a temperature approximately the same as that of the blood. Jacobson showed by direct experiment that in pleurisy the affected cavity was either of the same temperature as, or even slightly colder than, the other one. More recently, in December, 1879, M. Peter brought before the Paris Academy of Medicine a series of observations made upon patients affected with chronic peritonitis, and drew from them the conclusion that there was a very considerable local production of heat independently of the general bodily temperature. But what he really showed was nothing more than that the temperature of the abdominal wall may in such cases exceed that of the axilla by as much as 3·5° Fahr. Unfortunately he omitted to place a thermometer in the rectum, and, therefore, I think it may be fairly contended that in his cases the temperature of the inflamed part may after all have been no higher than that of the blood.

If Jacobson's observations are correct, the real increase of temperature in the skin and other superficial structures when inflamed may yet receive a very simple explanation. For the blood-current is to be regarded as a great warming apparatus, which carries to the tissues all over the body heat that is generated chiefly in the liver and other glands, and in the muscles. If there be no additional local production of heat, the temperature of each part must be the resultant of four varying conditions: (1) the temperature of the arterial blood supplied to it; (2) the proportion between the space occupied by its blood-vessels and that occupied by its extravascular material; (3) the rapidity with which its vessels are traversed by the blood; (4) the greater or less extent to which heat is dissipated from it by conduction, by evaporation, or otherwise.

Now, I think it is obvious that none of these conditions is liable to so great fluctuations as the second and third. We have already seen that the vessels of an inflamed part are dilated. That the flow of blood through an

inflamed part is much augmented has long been suspected. Sir William Lawrence is said to have roughly demonstrated this long ago by bleeding from both arms simultaneously a patient whose hand and forearm on one side were inflamed. But Cohnheim has recently established the same fact by careful experiments. Having set up inflammation in the paw of a dog, he measured the amount of blood which afterwards escaped through a cannula from the principal vein of the limb; and he found that it was sometimes more than twice as much as flowed from a corresponding vein on the opposite side in the same length of time. The only exceptions were, when extensive gangrene set in, and when there was very profuse suppuration. Any such increased velocity in the blood-current of an inflamed part must necessarily raise its temperature.*

The second of the conditions above mentioned explains why sometimes an inflamed part is not apparently hot. I have already cited Cohnheim's observation that when profuse suppuration was going on in a dog's paw there was sometimes no excess of blood flowing through its vessels. And it must also be borne in mind that the presence of inflammatory exudation in any considerable quantity tends to lower the temperature, since it is just so much added to those extravascular materials which have to be warmed by the blood-current. No wonder, therefore, that in cases in which the inflammatory process runs its course slowly, one occasionally meets with what is termed "cold abscesses." But it has not, I believe, been proved that they can develop from beginning to end without any elevation of temperature.

Exudation.—The changes of inflammatory exudation present many differences, according to circumstances which are as yet but imperfectly known.

In some cases, usually when a serous membrane is the seat of inflammation, the exudation assumes the form of a solid fibrillated material—"plastic lymph," as it used to be called. This is no doubt the same substance as the fibrin of coagulated blood, and is formed in the same manner. Where this, with the leucocytes contained in its meshes, seems, as in a dry pleurisy, to constitute the entire exudation, it must be formed very rapidly, for it appears as a thin uniform layer upon the sloping surface instead of accumulating in the more dependent parts of the serous cavity. But perhaps the whole of the exudation never does solidify; a fluid residue may have been removed by the active absorbing process which we have seen to be at work.

As Cohnheim remarks, we should *a priori* expect that inflammatory exudation would always contain fibrinogen, since that substance is one of the constituents of the blood-plasma. And, as white blood-corpuscles are certainly seldom absent, the question arises why fibrin, instead of being constantly present, is found principally in inflammation of certain structures —the serous and synovial membranes and the alveoli of the lungs.

In the first place, it is to be observed that so far from the composition

* No doubt if superficial vessels are involved in the inflammatory process, more heat is at the same time dissipated, and this tends to prevent the surface temperature from approaching that of the internal organs. But a point of considerable importance is that such an increased dissipation of heat actually makes the surface feel hotter to one's hand than it would otherwise feel. I have alluded to this in describing the relation between heat of skin as measured by the thermometer and as estimated by the perceptions of another person: *supra*, p. 46.

of inflammatory exudation throughout the body being uniform, almost every part yields a material which possesses characters of its own.

One peculiarity of *mucous membranes* is that when they are but slightly inflamed, as in catarrh from exposure to cold, they throw off an abundance of more or less altered epithelial cells. In the lungs or in the kidneys such "catarrhal products" are often seen in immense numbers; not only do they accumulate in and fill up the pulmonary alveoli or the renal tubes, but a great many of them are also carried away in the sputum or in the urine. The relation between catarrhal inflammation and inflammation in general constitutes a question of the greatest importance, especially in reference to tubercle, as we shall see hereafter: it has, however, been almost ignored by those who have recently written upon the subject. I doubt if we can at present form any other idea of it than that the excessive formation of epithelial cells is a result of the greatly augmented supply of pabulum offered to them in the inflammatory exudation. A tissue which in normal circumstances is constantly undergoing renewal may fairly be supposed to be ready to grow still more actively when such a stimulus is supplied to it.

On the other hand, when a *serous membrane* is affected with an inflammation of but moderate severity, it pours out a transparent fluid which accumulates in its cavity. There may be a thin layer of fibrin upon the free surfaces of the membrane; or shreds and flocculent masses may be floating in the fluid. But sometimes not a trace of fibrin is to be seen; fibrinogen is present, but for some reason remains uncoagulated. Virchow many years ago pointed out that fluid effusion, after its removal from the pleura by paracentesis, would often throw down a coagulum of fibrin, and that if this were removed, a fresh one might form in the course of the following day, and so on for several days in succession.

In the more intense inflammations of serous membranes the exudation is purulent; and this, as a rule, possesses no coagulating power. Intermediate conditions, however, are frequent, in which the surfaces are coated with fibrin, while the cavity contains liquid pus; and in the pericardium I believe that pus unmixed with plastic lymph is seldom, if ever, seen. But this is an exception, for in the case of all other serous membranes it may happen that every part of the surface retains its natural smoothness and lustre notwithstanding that pus is present in large amount.

The quantity of pus which collects in a large serous cavity, such as the pleura, is often enormous; and since it may be formed very rapidly the question has sometimes been raised whether the blood could possibly have yielded a sufficient number of leucocytes. But, as Cohnheim remarks, this difficulty in reality applies far less to the emigration theory than to that of pus-formation by proliferation. It has been shown that while inflammation is going on, the blood throughout the body contains an excess of leucocytes; and, indeed, the well-known hyperinotic condition of it may be said to be nothing more than a rough illustration of the same fact. One can easily suppose that the lymph-glands and the spleen throw into the circulating fluid a largely increased supply of leucocytes to make up for the drain that is going on. At the same time it appears very probable that pus-cells may themselves multiply by fission. Dr Thin has figured a dumbbell-shaped leucocyte which seemed to be dividing into two, from the inflamed cornea of a rabbit; and similar appearances have been observed in cold-blooded animals by Stricker, Klein, and Ranvier.* I have often thought that since

* It is important to notice that the peculiar appearance of the nucleus of a pus-cell—

leucocytes evidently must have nutriment to enable them to multiply, the absence or scarcity of fibrinogen in pus may perhaps be accounted for by its having been used up by them.

We have seen that the rarity of plastic lymph upon the free surfaces of mucous membranes is far from being a solitary breach of a general law for inflammatory exudations throughout the body. And the exceptional frequency with which a fibrinous layer is found lining the higher air-passages may be due to the same unknown causes which render the alveoli of the lungs also capable of a "croupous" as well as of a "catarrhal" inflammation. In connection with this question certain points have recently been raised which deserve careful consideration. Cohnheim has drawn attention to the fact that in the interstices of the tissues generally, exudation seldom coagulates; and he has shown that this probably depends not upon the absence of fibrin-factors, but rather upon a controlling influence which living structures exert over coagulation in their immediate vicinity. It would follow that whenever we find solid exudation in the connective tissue of any part of the body or within the stroma of an organ, we may be sure that the vitality of the part was endangered, if not lost, before coagulation took place. Now, I am about to point out that there is an important class of inflammations in which the exudation of fibrin is thus associated with a local death of the inflamed structures. And Wertheim has lately asserted that a destruction of the epithelium of the trachea is a necessary antecedent to the development of a false membrane in the trachea, both in animals under experiment and in the human subject during disease. So far as I know, the suggestion has not yet been made that the endothelium of a serous cavity must die before a layer of plastic lymph can be formed upon it. Yet surely inflammatory exudation is far less removed from the influence of living structures when it is contained in the pleural or peritoneal cavities than when it is free upon a mucous surface.

The inflammations to which I have just referred as constantly combining fibrinous exudation with a loss of vitality in the affected tissues are those which morbid anatomists now call "diphtheritic." The term is an unfortunate one, since it suggests an impression that they are connected with the disease Diphtheria, whereas this is by no means generally the case. Still, I think we cannot avoid using it, for I know of no alternative name, and the condition itself is one which demands recognition. It is that in which a free surface, usually a mucous membrane, becomes coated with an adherent layer which in part at least consists of the upper strata of the mucous membrane itself, infiltrated with a fibrinous material. The distinction between a *croupous* and *diphtheritic* false membrane is not merely, as was formerly taught, that the one is easily separable while the other is firmly attached. It is chiefly in the air-passages, where there is a thick basement membrane, that even croupous membranes lie loose; upon the fauces and elsewhere they often adhere closely, if their fibrillation is dense and strong. A surface affected with diphtheritic inflammation looks dry, and is of a slate grey or pale yellowish colour unless it has been accidentally stained by a coloured fluid, such as bile. It feels rough and granular, and a still more marked character is its tough elastic hardness. In the dead body, if an incision is made into a diphtheritic membrane, the dry grey appearance is found to penetrate to some

the replacement of the single nucleus of a blood-leucocyte by two, three, or even five irregular granules—is an indication not of multiplication by fission, but of impending disintegration and death. Cohnheim, indeed, says that pus-cells must be alive, for they are capable of amœboid movements; but I suppose that this is only when the pus is of very recent formation.

depth, and to cease somewhat abruptly. Under the microscope is to be seen a granular, more or less distinctly fibrillated substance, containing in its meshes altered epithelial cells, and leucocytes which have lost their nuclei. In the more superficial part of such a false membrane the fibrin is often developed into thick and glistening fibres; in the deeper layers, where it is infiltrating the interstices of a pre-existing tissue, it has no room to show even fibrillation, and it looks granular. As I have already shown, so much of the substance of the part as is involved in the diphtheritic process has already lost its vitality, and the only way in which the disease can end (unless it destroys the life of the patient) is by the separation and shedding of all the dead tissues, with the false membrane of which they form a part. This detachment is effected by *ulceration*. From the first there was an inflammatory action of more or less intensity in the living structures beneath. But now leucocytes collect in large numbers along the boundary line, and I believe that the connecting material is directly eaten away and absorbed by them. Perhaps the best specimens of the shedding of an extensive diphtheritic membrane are afforded by the urinary bladder. This organ is exceedingly liable to diphtheritic inflammation, which often involves the whole thickness of its mucous coat: and it is no uncommon thing for the *mucosa* to be cast off almost entire. A year or two ago I made an autopsy in the case of a woman from whose bladder during life a piece of considerable size had been removed. All the rest of the vesical mucous membrane lay loose, as a thick ash-grey substance, with markings on one surface corresponding with those of the hypertrophied muscular trabeculæ.

It is obvious that recovery from a diphtheritic inflammation, when possible at all, involves the regeneration of the tissue which has undergone destruction, or at least its replacement by cicatricial tissue. After the separation of a croupous membrane there is nothing to be renewed unless it is the epithelium.

Not only is diphtheritic inflammation characterised by loss of vitality of the affected tissues, but also by their putrefaction; as is shown by their dirty grey colour, and by the fœtid odour which they give off. Indeed, this form of inflammation is but one member of a group, which may properly receive the name of "putrid inflammations," and in many of which large masses of tissue undergo death—*mortification* or *gangrene*.

Gangrenous inflammation.—It is customary to describe several forms of gangrene, and to refer them to different causes. But I think that the more one studies these supposed varieties the more difficult is it to regard the distinctions between them as fundamental. Of course, it is true that a part may be directly killed in various ways without the intervention of an inflammatory change in it; as, for instance, by escharotics or by certain degrees of either heat or cold, according to the length of time during which they are in operation. But in every case, I believe that a less intense application of the destroying agent would set up a violent inflammation, which itself would end in gangrene. So, again, when the death of a part is due to a failure of its blood supply, no doubt it does not always first undergo inflammation throughout its whole substance. But here, also, it seems impossible to draw a hard and fast line. Surgeons are now familiar with the fact that in many instances senile gangrene of the foot, which used to be attributed merely to obstruction of the arteries, arises from slight injury to a toenail, and begins as an inflammatory process. And even when a limb undergoes mortification as the direct result of embolism of its main artery there is always at the

margin of the gangrenous part an active inflammation, which leads to a further death of the tissues. Indeed, Cohnheim has shown that the effects of a temporary ligature applied round the ear of a rabbit depend entirely upon the duration of the constriction. After a certain length of time the blood fails to penetrate into the vessels when the ligature is removed, and the part dies; but in other experiments, which were terminated at earlier periods, the result was only an intense inflammatory swelling of the ear with hæmorrhages into it. Lastly, in those forms of gangrene which seem to be determined by the occurrence of pressure—whether directly, by its lowering the vitality of the tissues, or indirectly, by its cutting off the blood-stream through them—there are always further conditions: a previous enfeeblement of the circulation, and some slight local injury which under normal conditions would have produced no appreciable ill-effects. Thus, bedsores occur chiefly in those cases in which there is also a marked tendency to hypostatic congestion of every part that happens to be dependent; and I believe that the gangrene is always preceded by an inflammation that can be directly attributed to such causes as irritation of the skin by excreta, or bruising of it during even gentle washing and drying, or friction of it against folds in the sheets. Thus, if we exclude what may be termed the immediate or instantaneous occurrence of gangrene, I believe that all forms of it may be said to arise out of an inflammatory process.

We have seen that putrefaction is an essential part of gangrene. But in different cases there are great variations in the rate at which chemical changes in a dead tissue advance, and in the degree of fœtor produced by them. In the "senile" form, the affected parts dry up and shrivel into a hard material like that of a mummy; and "dry" gangrene was formerly described as the characteristic effect of obstruction of the arterial blood supply. But it is now known that the desiccation depends upon the escape of fluid from the surface in consequence of detachment of the epidermis. And it is only when mortification spreads very slowly indeed through the more massive segments of a limb that they can "mummify." The gangrene which ensues upon embolism of a large artery, is, I believe, always more or less moist. But it often happens that deep structures remain soft, while superficial ones dry up into a horny mass, which is then termed an *eschar*. The colour of gangrenous parts is generally greenish or purplish black, but sometimes grey; it is due to chemical changes in pigment which has escaped from the blood-discs and has diffused itself through the tissues. The horrible odours so often emitted are attributed to the formation of ammonia, sulphuretted hydrogen, volatile fatty acids, &c. Gases frequently collect to such an extent as to render mortified tissues emphysematous, so that they crackle or "crepitate" when they are touched. In other cases they are saturated with a dark, thick, red fluid, which perhaps also raises the cuticle into bullæ. That structures which have undergone mortification are completely devoid of sensibility and of any other vital function, is a matter of course. They also feel cold, since their supply of warmth is cut off with their supply of blood; but it is to be noted that very superficial patches of cutaneous gangrene sometimes receive so much heat by conduction from the subjacent textures, that their temperature scarcely appears lower than that of the skin in their neighbourhood.

Gangrene is often from the first "circumscribed," or limited to a certain part, and even when it spreads from one spot to another, its progress—if indeed the patient's life is saved—is at last always arrested. A "line of

demarcation," as it is termed, is then formed. Here active inflammatory changes develop themselves, an abundant exudation of leucocytes takes place, and the connection between the living and the dead tissues is gradually eaten away, until the latter become completely detached and are cast off.

Hitherto we have been regarding the different characters of inflammatory exudation as consequences either of special tendencies which we find to be manifested by the several tissues, or of the greater or less intensity of the irritation by which the inflammatory process was set up. There is, however, another side to this question. Some years ago Sir James Paget examined the fluid contained in blisters raised by the application of cantharides to the skin, in thirty patients of St Bartholomew's Hospital. In some cases it formed a firm, elastic, fibrinous coagulum, in others it was purulent. The former condition was observed in persons who were in sound health, the latter in those who were suffering from advanced phthisis or a similar disease. And, with a little practice, he found himself able to form a fair opinion, from the contents of a blister, as to the degree to which the person was cachectic, and as to the extent to which inflammation in him would tend to be "adhesive" or "suppurative." Again, in those who are suffering from Bright's disease of the kidneys it is well known that the exudation produced by even slight inflammations is apt to be very profuse and watery. Lassar found, by direct experiment, that when hydræmia was induced by the free injection of a saline solution into the veins of a dog, in whose paw inflammation had previously been set up, the lymph which escaped from the lymphatics of the limb became much increased, while the amount of solid matters in it was as greatly lowered. Moreover, patients affected with dropsy from renal disease are especially liable to gangrenous forms of inflammation, and the same thing may be said of those who labour under diabetes.

Events of inflammation.—Up to this point we have been dealing with the advance of the inflammatory process. But, except when it is cut short by the death of the patient, it always sooner or later enters upon a further stage, one of *subsidence* and *repair*.

If the inflammation is but slight, and if its exciting cause does not continue in operation, it may end directly in what is termed *resolution*. The circulation through the vessels of the affected parts then gradually resumes its natural condition; any leucocytes which may have escaped into the tissues pass away through the lymphatic channels; and any solid exudation that may have formed undergoes conversion into a fatty emulsion, which is likewise carried off in the lymph-stream.

On the other hand, if pus-cells have accumulated in large quantities in a serous cavity, or in the sac of an abscess, and are not removed by a natural or artificial opening, they die there and break up into granules. The fluid belonging to them is then absorbed, while the solid matters remain as an opaque yellowish mass. This process is known as *caseation;* it is one to which I shall frequently have occasion to allude. Such cheesy residues are supposed by many pathologists to be incapable of undergoing further absorption; but, for my own part, I think that this is very doubtful. They often become the seat of deposits of earthy salts, which give to them the consistence of mortar, or may render them as hard as bone. The tissues which surround them always present formative changes such as I am about to describe.

These, however, must sometimes be regarded as the result of a fresh irritation set up by the dead and caseating matters, which are then said to act as "foreign bodies."

In all other cases, the *formation of new tissues* constitutes a direct and essential part of the subsidence of inflammation. Thus, when a slough or an eschar becomes detached, or when an abscess discharges its pus, a hollow space is left, which has to be filled up by a process of growth. And even where the morbid change does not go beyond the exudation of plastic lymph this is often to some extent developed into a permanent structure. The steps by which such varieties are brought about were studied with remarkable success by Ziegler in 1876. He embedded in the subcutaneous tissues of dogs a series of minute flat chambers, each consisting of a pair of glass plates, with a capillary space between them, cemented together at the corners, but open at the sides. These he removed after an interval of several days or even of some weeks, and he was fortunate enough to find not only that leucocytes had penetrated into the space between the plates, but sometimes that tissue elements and even blood-vessels had developed there. The earliest change consisted in the formation of large granular cells with vesicular nuclei. These he terms "epethelioid" cells on account of their appearance. They are generally round, but sometimes oval or even irregular in shape. Their size is very variable, but sometimes they are larger than the epithelial cells of the tongue. They evidently arise out of leucocytes, which appear to grow at the expense of other leucocytes, whose protoplasm they appropriate or absorb. Indeed, some go on developing until they become what are termed *giant-cells*—irregular masses of protoplasm—each containing a large number of nuclei. That these also are formed by the growth of a single cell, which destroys several others, there is no reason to doubt. In support of this view Ziegler lays stress on the fact that they are constantly found surrounded by an empty space. At first, he supposed that all the nuclei of a giant-cell were developed by fission from the nucleus of the leucocyte in which it had its origin; but he has since admitted that the nuclei of the absorbed cells may perhaps persist in it. Lastly, another feature of some of Ziegler's preparations is a *reticulum*, forming sharply outlined septa round the individual cells. This appears to indicate a tendency towards tissue production, but there is no reason to suppose that it is a permanent structure.

The really important agents in the formation of connective tissue seem to be these so-called "epithelioid" cells. They become pear-shaped or spindle-shaped, and give off bundles of delicate fibrils. These fibrils assume parallel directions, and ultimately their extremities unite with those of fibrils derived from other cells. But there is one condition which appears to be essential to such a result; namely, the development of blood-vessels in the exudation. They seem to arise partly as buds and loops from the sides of already existing capillaries, partly out of rows of cells, placed end to end, so as to open into one another, and form hollow tubes.

When much suppuration occurred round Ziegler's glass chambers, no formative changes were discernible in them. This corresponds with the well-known fact that only fibrinous effusions into a serous cavity are capable of organisation. But it is particularly to be observed that in speaking of "plastic lymph" as undergoing development into connective tissue and vessels one means, not the fibrin itself, but the cells that are included in it. The "fibrillated" structure of the lymph has nothing whatever to do with the processes that subsequently go on in it.

In human pathology it is chiefly by the study of granulations that one has an opportunity of observing formative changes originate in leucocytes: but Cohnheim speaks of large round or elliptical cells or even cells with many nuclei, as being generally discernible in small numbers in the pus which is discharged from a wound or an abscess of not less than a week's standing.

The connective tissue which is developed out of inflammatory exudation is sometimes of an enormous thickness, as in the pleura. It very slowly reaches its permanent condition. At first it contains no elastic fibres; according to Paget, they may appear as long as twelve months after its first formation. Lymphatic channels in it have been particularly described by Van der Kolk; and nerve-fibres are said to have been discovered. Bone is often formed where the part inflamed is one that naturally contains bone. In the cardiac valves, amorphous calcareous matter may be deposited in large quantity. Inflammatory adhesions and cicatrices have a strong tendency to contract, and this may give rise to very serious consequences if vital organs are concerned. But, on the other hand, adhesions sometimes after a long time relax and soften, even if they do not altogether disappear. Paget cites the following observation of Bichat's: a man had made from twelve to fifteen attempts at suicide, at different times, by stabbing himself in the abdomen. In the situation of the more recent wounds it was found that the intestines adhered to the parietes; but the adhesions corresponding with the older ones were reduced to narrow bands, or had even become divided and were hanging free. In cicatrices also a marked "loosening" may at length take place. These facts, and others, like the ultimate subsidence of sclerodermia (*v.* infra, vol. ii), deserve to be borne in mind in reference to analogous changes in internal organs, which we are too apt to regard as necessarily permanent.

Hitherto I have said nothing about the development of epidermis or of *epithelium* during the subsidence of inflammation, although it is a necessary step in the process, not only after ulceration of the skin or of a mucous membrane, but in many other circumstances. On this question, the most modern doctrine is one to which I hesitate to give my assent. That the cuticle which ultimately covers a granulating wound always arises in contact with pre-existing epidermis is well known; Reverdin's practice of skin-grafting is an interesting illustration of it. But this fact by no means necessarily involves the truth of the doctrine that epithelial cells are always actually developed out of cells of the same kind; it would be quite explicable upon such a theory as that which Rindfleisch propounds under the name of "epithelial infection," the existing epithelial cells being supposed to influence the adjacent granulation tissue, so as to lead its cells to grow into epithelium. However, on the basis of the differentiation of the cellular elements of the embryo into three layers, it is now taught by most pathologists that it is impossible for leucocytes, which belong to the middle layer, to form epithelial cells, these being constituents of the upper and lower ones. We shall see further on that in relation to malignant tumours this question is one of the highest importance.

Ætiology.—With regard to the causes of inflammation in general little need be said in the present chapter; they will be discussed hereafter in detail. But it must be pointed out, that the analogy of the pathological processes concerned, compels us to classify as inflammatory a large number

of affections in which we cannot directly recognise the operation of any irritant upon the vessels or the tissues, so that we have to fall back upon theoretical conceptions of a more or less unsatisfactory kind. Concerning the mode of origin of the large and important class of internal inflammations due to exposure to cold we know absolutely nothing. That cold often excites pleurisy, pneumonia, myelitis, nephritis, and a host of so-called catarrhal affections, is certain. But I am inclined to think that there is no general principle to which their ætiology can be referred, and that the relation between these several diseases and their cause is different for each one of them. A point of importance is that there are some organs and tissues which, so far as we know, are not capable of inflammation as the result of cold. This is the case, for example, with the brain; and, I believe, it will be found to be so with the peritoneum likewise if one is sufficiently careful to exclude all those forms of peritonitis which start from the uterus, or from some other neighbouring structure.*

There can be no doubt, however, that many inflammations depend only in part upon the external cause that excites them; the larger share of their ætiology belongs to what may be fairly termed a "predisposing" morbid condition of the tissue itself or of its blood-vessels. And it would seem that this has an important bearing upon the way in which some inflammations spread from point to point, and even from tissue to tissue, without decrease in their severity. When an irritant is applied to any part of the body its effects may extend for some little distance beyond, but their intensity rapidly diminishes—unless, indeed, the resulting morbid process belongs to a separate group of affections, those which are "septic" or "infective." But many inflammations not obviously traceable to an external cause diffuse themselves very widely. For instance, we may find pleurisy complicated with pericarditis or peritonitis, pneumonia leading to meningitis or to nephritis, and so on. In one remarkable case, in which I made an autopsy, the patient, a boy, was admitted into hospital under Dr Taylor for what at first appeared to be meningitis, but a day or two later the symptoms seemed rather to be those of peritonitis. After death the oldest lesion was found to be a chronic pericarditis, probably of three or four weeks' standing; but in addition there was acute pleurisy, diffused acute peritonitis, and equally universal acute meningitis. In no part of the body was there any trace of tubercles, or even of caseation. In such cases we have no reason to suppose that any specific infective agency has been at work. To what can we attribute the spread of the disease, if not to the circumstance that its main cause was an unsound state of the affected tissues altogether antecedent to irritation of them?

We have seen that the characters of the morbid process lead us to identify as inflammatory a number of affections which are by no means obviously traceable to irritation of any kind. They also enable us to draw a similar conclusion in another large group of cases, notwithstanding the absence of most or even all of the four "cardinal" symptoms. The inflammations in question are what are termed *chronic*. No doubt Cohnheim

* The same remark applies to the pericardium, and beside the brain, to the liver, spleen, adrenals, testes and ovaries. Of the mucous membranes, those of the entire respiratory tract, including the conjunctiva, frontal sinuses, larynx and bronchi are most prone to inflammation "from cold;" next the fauces and intestines; rarely the bladder; and the mouth, œsophagus and stomach are exempt.—Ed.

is right in saying that the reason why they are so often unattended with heat, or redness, or pain, is that their extent is so limited; and we may perhaps follow him in assuming that their prolonged duration is chiefly due to the fact that the newly-formed vessels and tissues act as causes of fresh irritation. It has often been said that there is great difficulty in determining what affections should and what should not be classed as chronic inflammations. But I think that this has mainly arisen from the fact that until recently pathologists were strongly biassed towards regarding the changes in question as either "degenerations" or "hypertrophies." Let me cite as examples *arteritis deformans*, with its terminations in atheroma and in calcification, the fibroid thickenings of cardiac valves, the secondary sclerosis of the spinal cord and of other nervous tissues. The inflammatory nature of all these affections is now generally allowed. As regards hypertrophies, however, it must be admitted that there is in reality no boundary line between them and chronic inflammation, at least so far as the connective and the epithelial tissues are concerned.

I shall not in this work attempt to lay down rules for the *treatment* of inflammation in general. The affections to which alone such rules would apply almost all belong to the domain of surgery rather than to that of medicine, so that I should be repeating to no good purpose what will be found in full detail in surgical text-books.

PYÆMIA

Theories of pyæmia—Metastasis—Phlebitis—Septicæmia—Embolism—Infection—Microphytes—Practical application—External or traumatic and internal pyæmia—Channels of infection—Local distribution of secondary abscesses—Idiopathic pyæmia—Slighter forms—Diagnosis: especially from enteric fever and tuberculosis—Prognosis—Treatment.

It is remarkable that so little attention was formerly paid to the malignant process which often rendered slight wounds and injuries mortal, and which up to the present day was the great danger to be feared after the most skilful surgical operations. The very name is recent, and the disease was quite unknown, while tetanus, the far rarer sequel of wounds and injuries, was familiar. Probably one reason for this was that the immediate effect of wounds in producing what was called "irritative fever" was confounded with the later appearance of pyæmia, and that this was not distinguished from the still later "hectic fever" which accompanied prolonged suppuration. Another reason may have been that in the hotter climates with which the physicians of ancient Greece, and of Italy in the sixteenth and seventeenth centuries, were familiar, cases of tetanus are more common than in the north of Europe. Lastly, whatever other miseries followed an ancient or mediæval battle, the wounded were at least free from the dangers which made the crowded wards of a city hospital, until recent times, a hotbed of pyæmia.

Pyæmia (wound-fever, surgical infection) is happily of much diminished practical interest to the modern surgeon; but its pathological interest is as great as ever, and it enters so far into certain forms of internal disease that a treatise on the principles of medicine is incomplete without some notice of this general morbid process.

Theory of pyæmia.—When an unhealthy-looking wound was accompanied by fever, with wasting, sweats, and more or less jaundiced tint of skin, the earlier pathologists supposed that an "ill-habit of body" prevented the wound from healing; and when abscesses were discovered after death in the lungs, liver, and other organs, they were ascribed to *metastasis* or transference of "disease" or "inflammation" from the wound to the internal organs. Even the strongest humoralists seem not to have supposed that the ill-habit of body was due to poisonous humours absorbed from the wound.

The first attempt to ascertain the exact nature of the process of pyæmia was made by the great French anatomist, Cruveilhier. He believed it to depend upon *suppurative phlebitis*,* inflammation of the lining membrane of the veins which were in communication with the suppurating wound. Hence, for many years surgeons dreaded injury or even ligature of veins.

It was one of the achievements of Virchow that he discovered the true meaning of what was called phlegmonous, suppurative, or infective phle-

* Hunter had long before (1784) distinguished adhesive from suppurative phlebitis.

bitis. He showed that what was supposed to be a mass of pus-corpuscles, the produce of inflammation in a vein, was really a discoloured clot composed of leucocytes and fibrin. He showed that fragments of this clot might be detached and carried by the still open veins to the right side of the heart and thence to the lungs, or might be carried from the lungs to the systemic capillaries. This remarkable process of *embolism*, by which detached particles of clot, carried from the original *thrombus* by the blood-stream, are lodged in the next capillaries, had been independently discovered by the late Dr Kirkes, of St Bartholomew's Hospital. It was extended by the great pathologist of Berlin to explain not only the mechanical effects of a plug of fibrin acting like seeds or other indifferent foreign bodies experimentally introduced into the blood-vessels of animals, but also the more dangerous results when the emboli are derived from aseptic or putrid thrombus, and act no longer as mere mechanical obstructions to the blood-vessel in which they lodged, but as centres of infection to the surrounding tissues. The mechanical effect of emboli upon the hydraulics of the circulation will come under consideration when embolism of the cerebral arteries is discussed. It is of practical importance in this case, and also when it occurs in the retina, in the kidney, or in the spleen; and it is most important of all when it affects the lesser circulation and obstructs a main branch of the pulmonary artery. When the clot is septic, two pathological processes are combined, the mechanical effects of the embolus and the infective power of the poisonous embolus. This latter condition was distinguished by Virchow as *septicæmia.**

Previously, the effects of pyæmia had been ascribed (as the name implied) to actual contamination of the blood by pus. But when pus-corpuscles were ascertained to be only, as Virchow believed, young cells like white blood-corpuscles, or rather, as we have seen in the last chapter they are now proved to be, white blood-corpuscles out of place, it was clear that the mere presence of these elements in the blood could no longer explain the process of pyæmia. Moreover, as Virchow had proved, the white cells found in the thrombus were not pus (for they had never left their habitation), but leucocytes aggregated by coagulation and not by inflammation. Accordingly, various terms such as septicæmia, ichoræmia, and sapræmia (since applied in a special sense) were invented or adopted from earlier pathology to express the element of "blood-poisoning" which complicated that of embolism.

In this as in other departments of pathology, direct experiment upon the lower animals was at once the herald and the guide of speculation,—speculation based on the exact facts of minute anatomy, and controlled by clinical experience. It was found, first, that minute fragments of healthy blood-clot, introduced into the circulation, acted like seeds or any other non-irritant foreign bodies as mechanical obstructions, leading to anæmia, to hæmorrhage, and other local results, but not to suppuration or to fever. Secondly, it was found that pure *pus laudibile* when injected into a vein, either mingled with the blood, the corpuscles gradually breaking up and no effect following, or if in large quantity formed an ordinary non-infective thrombus. Thus again, "pyæmia" was proved not to be what the name signified, pus in the blood. Cruveilhier had himself ascertained that globules of mercury when injected acted as irritants and produced inflammation and abscess around the point where they stuck in the capillaries. Thirdly, it was found that when ichorous,

* The refutation of the hypothesis that suppurative phlebitis spread by continuity from the wound to the heart, was due to Arnott and Dance, who wrote before 1830. See Virchow's 'Ges. Abh.,' p. 637.

putrid, unhealthy pus, carefully filtered so as to get rid of all fragments of solid matter, or even dialysed so as to leave corpuscles and most of the albumen and globulin behind, was injected into a vein, the result was increased temperature, disturbance of the nervous system, diarrhœa and congestion of the lungs; but neither during life nor on dissection after death were "metastatic" abscesses discovered, nor lobular pneumonia nor embolic "wedges."

Thus the result of observation and experiment up to a recent date was that pyæmia or wound fever was not due to "metastasis" of suppuration from the seat of injury to internal organs; that it was not due to suppurative, still less to ordinary adhesive phlebitis; that it was not due to the entrance of pus into the blood, nor to mere embolism, nor to poisoning of the blood with chemical compounds in solution, such alkaline products as have lately been recognised as *ptomaines;* but that it was made up of two pathological processes, embolism and septicæmia. An infective or septic thrombus gave rise to infective emboli, and each of these reproduced the parent suppuration, not by a mere deposit of pus, but by acting as an irritant and exciting around it as a focus true inflammation which ended in the formation of pus. Every "pyæmic" abscess of the lungs is preceded by lobular pneumonia, every such abscess of the liver or kidneys by corresponding inflammation. The "secondary" abscesses resemble the primary suppuration as the secondary "deposits" of cancer resemble the primary tumour.

But all these observations and experiments were, in the words of Bacon, "light-bearing" rather than "fruit-bearing." Knowledge of the process of pyæmia had come, but power to prevent it lingered. The mortality from pyæmia after operations in well-ventilated, cleanly hospitals was grievous, but in such as the old Hôtel Dieu of Paris, twenty years ago, and the old hospital at Halle, it was terrific. In order to avoid this dreaded scourge, the most elaborate dressings were devised, as complicated, as cumbersome, and as useless as the armour of the fifteenth century; breasts were removed by *cauterization à flèches*, wedges of chloride of zinc were thrust into the tissues and allowed to eat away the organ, tumours were grubbed out with the fingers, and even limbs were amputated by an *écraseur*. Meantime, thoughtful surgeons noted that pyæmia, so fearful under all the appliances of great cities and the dexterity of the most skilful operators, was far less frequent in country places, in open tents and in cottage hospitals.

At length the explanation came. It was not access of air, nor of oxygen, nor of pus, nor of ptomaines, but it was access of certain lowly-organised plants which made the utmost skill and the most elaborate precautions unavailing, and which had almost led some distinguished surgeons to abjure operations altogether. A brief account of these microphytes was given in the chapter on Contagion. They have been divided by their physiological effects, and rather for convenience than for scientific distinction, into those which are fermentative, those which secrete pigment, those which give rise to specific diseases, and those which produce putrefaction. The members of the last group, the septic bacteria and micrococci, are those which cause pyæmia. When they are precluded from gaining access to a wound, no infective thrombi are formed, no septicæmia occurs, no fever results, and no secondary abscesses are produced.

The recognition and investigation of these perilous microscopic organisms was the work of many observers, microscopists and botanists, physiologists and pathologists. But the application of this knowledge to the practical end of preventing half of the mischief they cause, was due to the scientific

training and philosophical insight, the skilful ingenuity and untiring efforts towards perfection of methods, which enabled our illustrious surgeon, Lister, to effect a beneficent revolution in practice.

Pyæmia then, though the term may be retained when its original significance has been disproved, is a somewhat complex process. It combines (1) Septicæmia or absorption of chemical soluble products, themselves probably the result of the action of septic bacteria; (2) Thrombosis; and embolism or transference of minute clots from the veins to the lungs and thence to the systemic capillaries; (4) Infective bacteria, mostly micrococci, surviving their transference in the blood-stream and propagating in the spot to which they are conveyed; (5) Local inflammation of a putrid, infective, and virulent character, which leads to (6) Necrosis and abscess.

Probably the local determination of a pyæmic focus is often determined by the size of the capillaries of the part; those of the lung are comparatively large, and transmit objects which will block the far more minute vessels of the joints. But beside this, Drs Wilks and Moxon ('Path. Anat.,' p. 626) argue with much force, and from large experience, that local stagnation (as in the back of the lung when a patient is in bed, in the cerebral sinuses or the large veins of the prostate, and in the recesses of the valves of veins) will of necessity cause an accumulation of infective particles, and thus determine the local points of their earliest and greatest activity.

Internal pyæmia.—In the great majority of cases pyæmia results from an external wound, accidental or operative, and so far it belongs to what is called surgical pathology rather than to that of internal medicine. But not unfrequently we meet with cases where the origin of the pyæmic process is internal and not traumatic. It is always a breach of surface. But it is not always a mechanical breach in the skin or accessible mucous membranes.

One variety of internal pyæmia, and the most important by its frequency, is when infection takes place from the *uterus* after delivery. The septic peritonitis and other pyæmic symptoms which follow the formation of septic thrombi in the great uterine sinuses, constitutes one form of the dreaded puerperal fever; and here also antiseptic precautions have been introduced. Another form of internal pyæmia is when the origin of the process is in *bones*, most often in the petrosal bone of the skull; such cases will be described under diseases of the brain. A third depends on suppuration of the *urinary tract;* but this is most frequently the result of stone in the bladder or of stricture, diseases which admit of mechanical treatment; so that urinary pyæmia, or as it has been called, "erysipelatous," "diphtheritic," or "infective" inflammation of the urethra, bladder and kidney, falls within the arbitrary limits of surgical pathology. Again, when the presence of a *foreign body* in the alimentary canal excites suppuration, a local or a general infection of the blood may result.

In most of these cases the primary seat of inflammation is more or less directly in communication with the exterior of the body, and therefore liable to contamination from bacteria on the surface. But in another remarkable group of cases the original focus of infection is in the interior of the vascular system. These cases of ulcerative, infective, or malignant *endocarditis* are in the fullest sense internal pyæmia, and here, as was first shown by Professor Heiberg, of Christiania ('Virchow's Archiv,' lvi, 407), the primary ulcer is characterised by the presence of septic micrococci. This remarkable variety of disease will be described under diseases of the heart in the second volume

of the present work. How microphytes gain entrance to the interior of the cardiac chambers is most difficult to understand, but not more so than their access to the bones in cases of acute Osteomyelitis.

Transference.—The virulent, putrid or septic, bacterial inflammations which accompany pyæmia appear to spread in three ways:

(1) By continuity: chiefly seen in the case of mucous surfaces, as when a gonorrhœal urethritis extends to the bladder and then along the ureter to the pelvis of the kidney; or when scarlatinal angina passes along the Eustachian tube and infects the middle ear; or when septic metritis travels up the Fallopian tubes to the peritoneum.

(2) By lymphatic channels: as when an unhealthy sore on the finger sets up inflammation of the lymphatics of the arm and causes a glandular abscess in the axilla. By a somewhat different route, but probably still by means of the large lymphatic channels which unite the areolæ of connective tissue with each other and with the great serous lymphatic sacs of the body, an acute suppurative pleurisy spreads to the pericardium, or infective parametritis to the peritoneum, or bacterial peritonitis to the pleura.

(3) By veins: in this process alone of the three, septicæmia and conveyance of septic organisms is combined with thrombosis and embolism. It is the commonest and most fatal of all. It may infect the veins of the extremities after amputation, the sinuses of the dura mater from caries of the bones of the skull, the portal system of veins from dysentery, and the systemic capillaries from ulcerative endocarditis.

Apart from these forms of local pyæmia, which can be explained by the anatomical relations of the parts involved, certain remarkable facts have been observed which appear to point to the great principle that, when morbid influences affect the whole body in common, each tissue and each organ has its own greater or less proclivity to disease, and each has, to some extent, an individual power of modifying the general process when it is itself affected. We see this law in the way in which the organs and tissues are affected by starvation, by overfeeding, by lead-poisoning, by alcohol, by the diseases which, like poisons, affect not the blood only but blood and bone and flesh —tubercle, syphilis and cancer, lardaceous and fatty degeneration, syphilis, scarlatina, and malaria. Every organ and tissue, every living cell in the body must be acted on by the same modifying agent, but each reacts differently and some do not react at all.

So with pyæmia. Percival Pott in the last century noticed that abscesses in the liver were particularly apt to follow injuries to the skull. The serous membranes suffer most in puerperal septicæmia. The lungs and liver are more prone to pyæmic suppuration than is the brain. The pancreas and mamma and testis almost always escape. Arterial pyæmia affects the spleen and kidneys and brain and liver more often than other organs. Infection starting from a bone is most apt to produce secondary abscesses in the heart and in the kidneys, as Wilks and Moxon discovered.

Idiopathic pyæmia.—Ulcerative endocarditis, portal pyæmia, urinary pyæmia, and cerebral pyæmia, with the results of infective caries of the vertebræ and pelvis, are the most frequent kinds of internal or, as it may be styled, "medical" pyæmia. Occasionally it results from an empyema or some internal abscess, from typhlitis, or from sloughing ulceration of the fauces, the rectum, or the skin.

But the most puzzling cases to account for are those which we are forced to call Idiopathic Pyæmia. It occasionally happens that on a post-mortem examination unmistakeable signs of general infective embolism are found, lobular pneumonia with pulmonary abscesses, ecchymoses of the pleura and pericardium, staining of the inner surface of the aorta, and multiple abscesses in the liver or kidneys or spleen, or suppuration in the joints; and yet it is impossible to discover the source of the infection. There is no external injury or wound, the urethra is found healthy, there are no anal fissures or inflamed piles, no chronic ulcer of the fauces or pharynx or nasal fossæ, no necrosis of the long bones, or caries of the vertebræ or ilium or internal ear. In one case I saw the late Dr Moxon, after laboriously searching these and other parts, extract every tooth from its socket to make sure that there was no alveolar abscess or caries; yet all was in vain. Sometimes we are rewarded by discovering the source of the mischief in unexpected situations,—a pin or other foreign body in the appendix cæci, caries of the ethmoid bone or of the sacro-iliac joint, pyosalpinx as the result of gonorrhœal inflammation of the vagina. But in a few cases we are compelled at the end of the search to confess our failure and to call the case one of idiopathic or spontaneous pyæmia.

Whatever doubts may still be felt as to the possible origin *de novo* of infective diseases, such as enteric fever, scarcely any pathologist will admit that bacteria appear in the blood spontaneously. They must, either as minute micrococci or possibly as still more excessively minute bacterial spores, gain an entrance by some undiscovered breach of surface. But we need be less scrupulous after due search has been made in vain for a source of infection in admitting the rare occurrence of what we may provisionally call idiopathic pyæmia, inasmuch as we are quite unable to explain the origin of the infective bacteria (*Micrococcus endocardiacus*) which are found in cases of infective ulceration of the cardiac valves.

Slighter forms of pyæmia.—It is probable that under this head, using pyæmia as inclusive of septicæmia, should be classed certain cases, common in surgical practice, and not unknown in the medical wards of a hospital, where a patient, suffering from some internal suppuration which has been relieved either naturally or by incision, is from time to time attacked by more or less marked rigors, rise of temperature, quick pulse, loss of appetite and sometimes profuse sweats. We can usually trace this to the flow of pus being interrupted, and when free exit is restored the symptoms disappear without further inconvenience.

Such accidents we see in the course of empyema which has been opened, of purulent pyelitis, of suppurating hydatid of the liver, and of otorrhœa from caries of the petrosal bone. In many instances an abscess has been opened under strict antiseptic precautions, and there is no reason to suppose the entrance of impure air or secretions afterwards. The symptoms referred to are certainly connected with retention of pus and disappear when free discharge is established. They are quite different from the mere effects of pain when healthy secretions are retained, or when an over-full cyst or an inflamed testis produce general disturbance by pressure, which disappears as soon as the tension is relieved. Hence, these symptoms cannot be referred to the mechanical effects of pressure upon nerves. They seem more likely to be caused by the increased pressure leading on hydrostatic principles to entrance of chemical products of inflammation, or possibly of septic organisms themselves, into the adjacent lymph-spaces or blood-vessels.

Diagnosis.—The symptoms of pyæmia as it occurs after a surgical operation are happily less familiar to the present generation of medical students than before the introduction of antiseptic surgery. Still, however, they occasionally occur and are well recognised. The difficulty arises when no wound or injury is found to account for the symptoms.

These cases of internal or non-traumatic pyæmia present themselves to the physician under the form of Fever, and pyrexia is the only constant symptom. It may be accompanied by local pains, by tenderness, or by œdema, which are of the utmost value in leading to the source of mischief. But these are often absent. Headache, delirium or stupor, muscular twitchings, diarrhœa, profuse sweats, even a jaundiced tint, are none of them constant symptoms, and all may be present in cases of typhus, of enterica, or of tuberculosis. Pain and swelling of the joints may also be present and help to simulate rheumatic fever. In cases of ulcerative endocarditis the cardiac murmurs with pyrexia sometimes make this last resemblance extremely close, and pericarditis, pleurisy, and synovitis may rather increase than diminish the difficulty of diagnosis, while the fact of previous attacks of rheumatism are far from making the more serious alternative unlikely. This difficulty will be discussed in the chapter on rheumatism in the second volume of this work. The age of the patient, the persistence of local synovitis, the presence of jaundice, and signs of embolism in the brain, spleen, kidneys, or retina, may more or less conclusively decide the diagnosis, but in some cases it is impossible to decide until time has developed the course of the disease. Moreover, rheumatism occurring repeatedly with concomitant lesions of the valves may at last appear in combination with a fresh and infective form of endocarditis.

The most characteristic single symptom of pyæmia is pyrexia, which is of a remittent type, rarely falling below normal, but rising suddenly and irregularly, to fall again with equal abruptness. It has none of the regular daily variations and steady general rise, culmination and subsidence which we observe in typhus, enterica, and the exanthems. It has not the evening rise of hectic fever, which in many respects it resembles, nor the frequent subnormal temperatures, alternating with irregular elevation, characteristic of some forms of acute tuberculosis. Hyperpyrexia is more common than in enteric fever or in phthisis. The resemblance to a regular form of remittent malarial fever is sometimes remarkably close and deceptive.

When a patient is found suffering from fever not due to external inflammation, we first ascertain the absence of internal acute disease, and especially of pneumonia and of phthisis. We may then have to await the appearance of a characteristic exanthem, as the rash of scarlatina, the mulberry eruption of typhus, or the erythematous blush of erysipelas, which is often delayed for a day or two after the temperature has risen. If the first week of fever has passed without these appearances, and if there are no physical signs of pneumonia—which sometimes only appear after two or three days of pyrexia—we then reconsider the possibility of enteric fever and of acute tuberculosis, and also the question of internal pyæmia.

Enterica usually begins gradually, the temperature rises gradually, and the state of the tongue and of the bowels makes diagnosis easy even before the rose rash appears. But occasionally it begins more abruptly, the temperature rises high in the first week, the rash is absent or has passed unnoticed before the patient was seen, the bowels are constipated, and diagnosis may then become extremely difficult.

Acute tuberculosis can usually be traced to previous tubercular disease

of the lungs or lymph-glands or abdomen, of which the signs remain in evidence, or there are caseous testes, or symptoms of "strumous" pyelitis. Or we may have evidence of meningitis, or we may discover one or more tubercles in the choroid. The lungs are almost always the seat of disseminated tubercles, and this may be recognised, not so much by physical signs, which are often limited to a little rhombus now and then, but by the high ratio of respirations to pulse and temperature, by slight duskiness or marked cyanosis, by the alæ nasi working in aid of the chest, and by dyspnœa unaccounted for by examination of the chest.

When combined with tubercular enteritis in a child, it may be almost impossible to distinguish acute tuberculosis from enteric fever, until the subsequent course of the disease makes it clear; and I have once, at least, under these circumstances misjudged the case until the autopsy showed that what was thought to be tuberculosis was really typhoid fever.

When we suspect pyæmia, our first investigation is directed to the ear for a purulent discharge, to any of the bones or joints which may be tender or œdematous, to the genito-urinary organs, and to the heart. Symptoms of pyæmia, with a cardiac murmur, point almost certainly to ulcerative endocarditis; this is the more probable if previous valvular disease has existed, and particularly if the febrile symptoms have come on during convalescence from pneumonia, as well stated by Dr Osler in his lectures before the College of Physicians ('Brit. Med. Journ.,' March 7th, 1885), and the signs of embolism in other organs confirm this diagnosis. In the rare case of this disease affecting the right side of the heart, of which I have lately had an example under my care, the lungs are the first organs to be affected, and the diagnosis is not less easy. When infective emboli lodge in the spleen it becomes enlarged, partly by the formation of fibrinous wedges *(infarcta)* which speedily break up into abscess-like masses of detritus with blood-discs and micrococci, partly by the febrile intumescence of the whole organ.

The splenic dulness being increased and the viscus being felt below the ribs on inspiration, does not in itself distinguish the case of pyæmia from one of enteric fever or of ague, but the "typhoid spleen" and the "ague-cake" are not usually sensitive, while the pyæmic spleen is tender to the touch, probably by reason of acute local peritonitis. Hæmaturia, hemiplegia, and, above all, the presence of icterus would confirm the diagnosis.

When otorrhœa, especially fetid otorrhœa, with a perforated tympanic membrane, is discovered, we may often trace tenderness or œdema over the mastoid process or down the neck. The infection travels by the internal jugular vein to the heart, and then affects the lungs, setting up suppurative lobular pneumonia and acute pleurisy.

On the whole, it may be said that in most cases internal pyæmia has an origin which with care and patience can be discovered during life; that enterica is the most variable of all specific fevers in its course and the most frequently unrecognised; and in doubtful cases we may remember that "common things most commonly occur," or, to state a converse truism, that we rarely meet with rare diseases.

The following recent example of internal (idiopathic?) idiopathic pyæmia will serve to show the difficulty of diagnosis, or at least the fact that a wrong diagnosis may be made.

A girl, twenty years of age, who had been confined four months earlier, was admitted into one of my wards on December 6th, 1886. She was in high fever, with more or less constant delirium and severe headache, and

photophobia. She had, we were told, complained only that morning, had left home without eating her breakfast, and was found moaning and almost insensible in a corner of the room where she had gone to sell bottles to the hospital patients. The pupils were contracted, there was broncho-pneumonia on both sides, and the temperature rose high, notwithstanding cold affusion and ice-bags. She had, we afterwards learned, complained of headache three or four days before the attack began, but otherwise appeared to be in her usual health. Her mother and a brother had died of consumption. She was in a very dirty condition and her scalp was covered with scabs from *impetigo pedicularis*. When the temperature was 104·2°, the respirations were 44, and the pulse 120. Next day she was more dull and lay curled up in bed. There was a faint apex-systolic murmur. On the third day a pleuritic rub was heard and the respirations rose to 54, with temperature 104·8°, and pulse 140. The next day the fever rose to its highest point (105·8°) and was once more reduced by ice; albumen appeared in the urine, and she died after three and a half days' illness. I supposed that she was suffering from acute tubercular meningitis with similar disease of the lungs. The brain, however, was found normal. There was recent plastic pleurisy and a vomica in the apex of the right lung, but no miliary tubercles in the lungs. There was no ulcerative endocarditis, but a little recent lymph on the mitral curtains. One small abscess was found on the surface of the liver. The spleen was swollen but contained no embolic foci. Both kidneys contained numerous small abscesses as in ascending pyelitis, but the pelves were normal, as were the ureter and bladder. The vagina, uterus, and Fallopian tubes were healthy; one ovary contained a serous cyst. The skull and internal ear, the sinuses, the vertebræ, the sternum and all the large joints were searched, and all the internal organs; but nothing further was found except one small ulcer in the duodenum. The portal vein was perfectly normal.

Prognosis.—As a rule this is hopeless, and the exceptions are very few. But there are cases, to which Sir James Paget has particularly drawn attention, of chronic pyæmia where the fever is never high, internal organs are spared, and the joints are successively attacked by suppuration. These cases sometimes recover, and it appears certain that some forms of bacterial infection can be dealt with successfully by the living leucocytes and tissues when the number of microphytes admitted is not too large.* So that we may say that, even with our present means of treatment, pyæmia is not an absolutely fatal disease.

Treatment.—The indications are first, to ascertain, if possible, the source of infection, and give free exit to the septic materials. An empyema laid freely open and drained, a mastoid bone trephined and the pus given exit, a sequestrum of bone cut down upon, or a joint freely incised, may not only prevent infection, but, there is reason to believe, may check a pyæmic process already begun from becoming general. Secondly, the free exhibition of stimulants, quinine, and as much concentrated food as can be taken. Quinine in large doses is believed by many surgeons to have a "specific" as well as a "tonic" effect in pyæmia. It is possible that it may check the multiplication of microphytes; and we may one day find in perchloride of mercury, or some more harmless and energetic parasiticide, a far more efficient means of dealing with even internal pyæmia than we at present possess.

* See an interesting paper by Mr Watson Cheyne, in the 'British Medical Journal' for 1886.

TUBERCLE

Anatomy and histology of tubercle: views of Laennec, Bayle, and Louis: of Virchow, Schüppel, and Ziegler—Tubercle a product of modified inflammation—Infiltrating tubercle—Caseous and fibroid transformations of tubercle—Koch's tubercle-bacillus—Result of experiments on animals: inoculation of tubercle: Perlsucht: infection by the mouth and by the lungs—Ætiology of tubercle: spread of tubercle in the body—Course of acute tuberculosis—Varieties of chronic tuberculosis: phthisis pulmonum, laryngis et ilei; tubercular inflammation of serous membranes: genito-renal tuberculosis; Addison's disease; tuberculosis of the lymph-glands and spleen; tubercular disease of joints; multiple tubercle generally—Concluding historical retrospect.

We find in the deadhouse that certain peculiar diseases, which affect in turn almost every organ, agree in presenting minute rounded bodies which are called *tubercles*, or *tubercular granulations*. But the relation of these bodies to the diseases in question has throughout the present century been a matter of never-ending discussion.

The importance of the controversy may be judged from the fact that it concerns the most frequent of all fatal maladies, pulmonary phthisis. And in proof of the extent to which opinions have differed, the words of Laennec in 1819 may be contrasted with those of Niemeyer in 1867: the former taught that "the existence of tubercles in the lungs is the cause, and constitutes the true anatomical character of Consumption;" the latter, that in the majority of cases tubercles, if found in the lungs after death, "have been of recent origin and have complicated the disease when it was already in an advanced stage." My own view is, and has always been, entirely in accord with that of the older writer.

Anatomy of tubercle.—One great stumbling-block in the way of a right understanding of the nature of tubercles has been an imperfect observation of the changes in appearance which they present in different stages of their formation. In this respect Laennec was far more accurate than many who have succeeded him. He described them as first "having the appearance of small semi-transparent grains, greyish or colourless, varying from the size of a millet seed to that of a hemp seed. . . . Afterwards they gradually increase in size, and as they do so they become yellowish and opaque, beginning in the centre. . . . Several unite together to form larger masses, pale yellow, opaque, and of the consistence of very firm cheese. . . . At length they soften and finally liquefy; this change also begins in the centre, and progressively approaches the circumference." The grey semi-transparent granules were termed by Laennec "miliary tubercle;" to the yellow cheesy masses he applied the unfortunate name of "crude or immature tubercle."

Louis, whom Laennec's premature death left the leading pathologist of Paris, limited the application of the word tubercle to the yellow opaque stage

of it; his method of statement being that the grey semi-transparent granules "undergo conversion into tuberculous matter." By subsequent writers the early grey stage was ignored altogether. Perhaps they were influenced by the misleading term "crude" tubercle, applied to the yellow stage of it. At any rate, they taught that the yellow material was deposited as such from the blood.

Virchow, therefore, effected a most important reform of pathological doctrine when he showed that a yellow cheesy substance, identical with "crude" tubercle, may arise out of decaying matters of various kinds, from ordinary inflammatory exudations up to sarcomatous and carcinomatous growths: in other words that *caseation* is merely a mode of retrograde metamorphosis. Nevertheless, I think that Virchow greatly underrated the extent to which cheesy masses in the lungs and in other organs are really of tuberculous origin. And one or two of the writers who have followed him almost go so far as to deny that tubercles ever caseate at all.

A proof that grey and yellow tubercles may be stages of the same morbid product is, as Louis long ago pointed out, that the successive changes described by Laennec may often be observed to occupy definite positions in the lung. Towards the base there are grey semi-transparent tubercles; higher up they are of opaline aspect and yellowish in their interior; higher still they are yellowish white throughout their entire substance. He might have added that they become larger and approach one another more closely from below upwards. Now, since it is certain that the upper lobe of the lung is almost always the earliest seat of tubercles, and that they gradually spread downwards through the organ, the conclusion seems indisputable that they are first grey and afterwards become yellow. It is, however, also a fact that tubercles often undergo caseation before they are large enough to be visible with the naked eye. Both in the pia mater and in the liver I have repeatedly found tubercles of microscopic size, which were already opaque not only in the centre, but in the greater part of their substance. It is therefore quite possible that no grey tubercles may be discoverable in a diseased organ, even when tuberculosis has been rapidly destroying it. Again, another fact, which is still more important, is that tubercles, instead of caseating, often undergo a different change, which leaves them permanently grey. The earliest observation of tubercles in this condition dates further back than the writings of Laennec himself. A few years before the publication of his great work in 1827, another French physician, Bayle, had divided phthisis into different species, only the first of which was termed by him "tubercular." To the second species he gave the name of "Granular Phthisis," and described the lungs as "stuffed with transparent shining granulations, of cartilaginous nature and consistence, never opaque, and without any tendency to soften." Now, it is a curious circumstance that those pathologists who (as we have seen) came to regard a yellow cheesy condition as typical of tubercle, habitually spoke of *recently-formed* grey tubercles as the "grey granulations of Bayle:" yet a perusal of the four cases of granular phthisis, related in detail by that writer, shows clearly that in three of them at any rate, the disease was of a *chronic* kind. Such a degree of hardness as he attributes to his granulations is, indeed, inconsistent with their having reached only an early stage of their development; and he also expressly mentions that they are sometimes pigmented or speckled with brilliant black dots and lines, which will be shown elsewhere to be characteristic of *chronic* tubercular affections of the lungs and bronchial glands. In fact the grey granulations of Bayle are really

tubercles which, instead of caseating, have undergone a fibroid change, which makes them persistently hard and semi-transparent.

Miliary tubercles, then, may be of three kinds :

1. *Soft grey granulations*, always of recent formation, and essentially transitory in their characters.

2. *Yellow granulations*, of either recent or old formation, tending, if they undergo further change, to soften or liquefy.

3. *Hard grey granulations*, always chronic, and liable to no change, except pigmentation.

Histological characters of tubercle, according to different authorities.—It may well be supposed that of late years histologists have had much to say with regard to the nature of the tubercular process. But, for my own part, I venture to think that their views on this subject are fundamentally erroneous. Their object has been to discover some microscopical characters which should serve to distinguish tubercular lesions from all others. Thus Lebert many years ago thought that he had found a specific "*tubercle-corpuscle*" in the yellow cheesy material which was at that time taken for the typical form of the morbid product. Since Virchow taught that this material is already in a state of decay and degeneration, the search has been actively prosecuted in tubercles still grey and recent. Virchow himself held that their histology is that of a form of tumour, namely, *lymphoma*. According to him, individual tubercles are comparable with the Malpighian bodies of the spleen. And, as he believed that they are apt to arise where no such lymphoid structures are naturally present, he regarded them as "hetero-plastic" new growths. Subsequent writers further developed this view by insisting on the existence of a reticulated stroma, such as belongs to other lymphomata. It was also suggested that the true seat of tubercles was perhaps the interior of the lymph-channels throughout the body, and that their cells might probably be formed by a proliferation of the lymphatic endothelium. On the other hand, Dr Sanderson endeavoured, in 1868, to demonstrate in those parts in which tubercles occur, the normal presence of lymphoid tissue, an overgrowth of which might lead to their formation, and thus render them "homœoplastic."

But about that time the attention of histologists became drawn to another element of tubercle, which has played a principal part in all the various theories that have since been advanced. That very large cells are sometimes to be found in a tubercle has long been known ; Virchow himself had spoken of cells containing twelve nuclei, or even more. Langhans and Schüppel, however, now insisted on the frequent presence of such bodies, and applied to them the name *Riesenzellen* or giant-cells, which had previously been invented by Virchow for similar elements occurring in certain sarcomatous tumours. According to Dr Hamilton, whose papers in the 'Practitioner' for 1880 are illustrated by beautiful drawings, giant-cells occur with the utmost regularity in all tubercles. They are from ten to thirty or forty times larger than the lymphoid cells, which are also present. They are sometimes placed in the centre of a tubercle, sometimes laterally. When a giant-cell is young, it seems to consist of a large mass of granular protoplasm, sometimes with many nuclei in it, sometimes without them. As it grows older, the peripheral part of it (the "periblast") becomes organised, and constitutes an almost fibrous, mantle-like sheath, in which great numbers of round or oval nuclei may be perceived, and from which ultimately processes arise in continuity with the reticulated stroma of the rest of the tubercle. Further on, Dr Hamilton speaks of a tubercle as made up of one or more "giant-cell systems." He

imagines that even the "lymphoid" and "epithelioid" cells which are found in the tubercle have originally been connective-tissue nuclei, which have become detached and thrown off into the meshes of the reticulum. But I must confess that, without attempting to challenge the accuracy of Dr Hamilton's observations, I cannot help thinking that there is much that is artificial in his descriptions. Histologists, indeed, are by no means agreed as to the origin of the giant-cells of tubercle. Dr Hamilton's view is that each of them arises by the progressive growth of a single large connective-tissue element. But Schüppel himself attributed them to the coalescence of leucocytes in the interior of capillary vessels. And both Dr Klein and Prof. Julius Arnold have recently stated that they may be produced by the fusion together of a number of epithelial cells, of which the nuclei persist. If such opinions are correct, it is of course impossible that giant-cells can possess the significance in regard to the development of tubercles which Dr Hamilton and others would assign to them. I suspect that in this instance, as in so many others, a striking name has conveyed a false impression as to the dignity and importance of the thing. And, after all, it must be remembered that giant-cells are very far from being as conspicuous or as easily demonstrable elements of a tubercle as they are of a myeloid sarcomatous tumour.

Again, the conception of giant-cells as the essential and distinguishing elements of tubercles has of late years been rudely shaken from another side. I have already quoted at p. 63 the experiments of Ziegler, who has shown, what had been to some extent recognised before him, that giant-cells are formed in ordinary inflammatory exudation, during the course of its "organisation" or development into tissue. I did not then mention that Ziegler's researches were undertaken with the special object of throwing light upon the structure of tubercle, and upon the relations between tuberculosis and inflammation. Such, however, was the case. Moreover, nodules, having the histological characters of tubercles, but of microscopic size, had already been found by other observers in places where their presence certainly would not have been expected. I do not now refer to Köster's discovery of tubercles (sometimes visible even by the naked eye) in the granulating tissue projecting into the interior of diseased joints; nor am I alluding to Friedländer's statement that similar bodies are constantly present in lupus and in the walls of scrofulous ulcers of the skin: for all of these affections are believed to bear a more or less close relation to tuberculosis. But it is impossible to say the same of certain other lesions in each of which Friedländer or Köster or Cohnheim has found microscopic tubercles. Among them are a shallow ulcer of the uterus in an old woman who died of apoplexy, the stroma of a cancerous tumour, the floor of a phagedænic chancre, and bands of adhesion in the pleura or in the peritoneum. Friedländer, indeed, maintains that all such cases are examples of "a local tuberculosis," comparable with a scrofulous testis, or even with pulmonary phthisis, which does not happen to lead to a general outbreak of tubercles. But it seems to me that this is to take a very strained and partial view of the facts.

Probable connection of tubercle with inflammatory exudation.—The general conclusion which I wish to draw from all these observations with regard to the histology of tubercle is, that there is nothing in them to prevent our accepting the doctrine that *Tuberculosis is a modification of the inflammatory process*, if commended to us on other grounds. Some of the reasons which have led me for one to accept it will be stated further on. My present

object is to point out that it seems to afford the only possible solution of a difficulty which histologists regard as insuperable, but which I am sure cannot be really so. I have already referred to Niemeyer's rejection of the opinion that tubercles are an essential feature of pulmonary phthisis. Now, the fundamental fact upon which his teaching was based was the microscopical observation that in many specimens of phthisical lungs the morbid process which precedes the occurrence of caseation is not the development of a lymphoid tissue, but a filling of the alveoli with epithelial cells, or in other words, a "catarrhal pneumonia." He accordingly declared that in reality a chronic catarrhal pneumonia, ending in cheesy infiltration, constitutes the primary anatomical lesion in many cases of phthisis. His opinions have since been adopted by many other pathologists. Dr Hamilton, for example, asserts that dry yellow nodules, such as were described in the lungs by Laennec, are generally nothing but patches of catarrhal pneumonia; bodies having an aspect similar to that of a tubercle, but as large as millet seeds, usually (he says) prove to be groups of air-vesicles affected with catarrhal pneumonia. Now, when I come to the chronic form of pulmonary disease I shall bring forward the evidence which has always led me to maintain the unity of phthisis and its essentially tubercular character; and, indeed, this view seems to have been finally established by Koch's observations with regard to the bacillus of tubercle, to which I shall presently refer. But it is the greatest possible mistake to suppose that the cases in which catarrhal pneumonia must be separated from tuberculosis can be limited to those of ordinary phthisis, if the separation is to be made at all. On the contrary, the principal point on which Dr Wilson Fox insisted in opening the discussion on tubercle at the Pathological Society in 1873 was that, in the disease known as acute tuberculosis, occurring in children, granulations "composed of epithelial proliferation" are generally found in larger numbers than those made up of lymphatic cells. And in that very year a German observer, Hering, actually went so far as to declare that the ordinary fatal miliary tuberculosis of the lungs ought to have its name changed into that of acute disseminated catarrhal pneumonia. But this would, after all, leave the difficulty untouched; because, as Dr Fox pointed out, granulations made up of an interstitial small cell-growth are generally also present in such cases, and some granulations consist partly of one kind of elements, partly of the other. Moreover, it has recently been shown by Julius Arnold that precisely similar epithelial changes to those in the lungs occur in the liver and in the kidneys, when affected by acute tuberculosis; while Gaule has demonstrated a caseating catarrh of the seminal tubules in the so-called scrofulous disease of the testis. Now, if the tuberculous process be regarded as a modification of inflammation, it is, I think, possible to solve the difficulty without giving up the use of the term tubercle, and without ignoring the important distinctions that undoubtedly exist between ordinary inflammatory affections and those that are tuberculous. In speaking of catarrhal inflammation (*supra*, p. 61) I have already pointed out that its relation to the inflammatory process in general is still imperfectly understood. But at least this is certain, that the same kind of irritation which in most other tissues leads to an exudation of leucocytes, causes, when applied to epithelial tissues, an overgrowth of epithelial cells. Ziegler has thrown out the very plausible suggestion that a tubercle owes its rounded form and its definite size (varying only within rather narrow limits) to the fact that it is produced by the circumscribed action of an irritant of but slight intensity upon the spot which afterwards becomes its centre. And he

contrasts the effect of such an irritant with the far more powerful operation of a septic particle, such as gives rise to a miliary abscess. Now, it is, I think, easy to conceive that whereas the circumscribed irritation of a minute area of connective tissue shall result in the formation of a granulation made up mainly of leucocytes, the same kind of irritation applied to an epithelial area shall cause a granulation consisting of epithelial cells. The two bodies may differ completely in their histology, and yet we may be quite right in giving both of them the same name. This is what I meant when I said that the views of histologists with regard to the subject of tubercle seemed to me fundamentally erroneous.

Specific character of tubercular inflammation : infiltrating tubercle.—The conception of tubercle which I have unfolded in the last paragraph is one which I have now for several years past taught in my lectures on pathology. Koch's discovery of a tubercle-bacillus appears to me to supply exactly the "irritant of but slight intensity" that is needed for the theory. The slow growth of this organism, to which Koch himself draws attention, accounts for the fact that the morbid process which results from its action is so much less acute than infective inflammations in general, with which tuberculosis must henceforth be regarded as comparable. I cannot doubt that the other "Infections-Geschwülste" of Klebs and Cohnheim, syphilis, glanders, and leprosy, are also to be looked upon as modifications of inflammation, so far as concerns the pathological process which arises from the specific virus of each.

It is not the least of the advantages of this view of tubercle that it enables us to understand how an affection (whether of the lung or of any other organ) may fail to present the characteristic granulations, and yet be really tuberculous. Laennec long ago described what he termed a tuberculous "infiltration of the lungs," when considerable portions of the pulmonary tissue become solidified by a greyish and semi-transparent, or by a paler and yellowish white material; either without any previous development of distinct tubercles, or around tubercles already formed. Subsequent observers have very generally declared such lesions to be "pneumonic." But, for my own part, I am prepared to maintain that in their naked-eye characters these "infiltrations" differ altogether from anything that is seen in ordinary pneumonia. And it has always appeared to me a striking point that, even when the growing edge of a patch of pinkish grey infiltration appears quite homogeneous, one often finds that the less recent parts of it, where caseation is commencing, show yellow granulations corresponding exactly with tubercles in size and in general appearance. Now, it is easy to see how a uniform consolidation should result from the irritation set up by bacilli if they happen to lie very close to one another through the pulmonary tissue. Again, in speaking of tubercular meningitis I shall have to point out that in association with acute tuberculosis of other organs an affection of the membranes of the brain is not infrequent, of which the characters seem to be those of a simple inflammation, since no tubercles are discoverable. In such cases, the presence of recently formed tubercles elsewhere appears to me to render it almost certain that the meningitis must be essentially of the same nature.

Caseation of tubercle.—Another peculiarity of tubercular lesions, which is, I think, scarcely less characteristic than the presence of the granulations themselves, is their tendency to caseate. It appears to me that pathologists since Virchow have generally been far too ready to assume that caseation is of frequent occurrence in affections that are merely inflammatory. It is of course indisputable that the pus contained in a serous sac or in a large abscess

may, and often does, dry up into a cheesy mass. The same thing is observed in the crypts of the tonsils, in the cæcal appendix, and in other pouches of mucous membrane, with which, perhaps, the alveoli of the lungs may fairly be compared. It is also seen in the walls of arteries when they become atheromatous. But it appears to me to be a very important circumstance that in every one of these instances the inflammatory exudation which caseates is more or less beyond the range of the blood-vessels. Is it a fact that simple inflammation ever leads to caseation in the substance of a vascular solid organ —in the liver, or in the spleen, or even in a lymph-gland—unless a definite abscess has been formed? Such an occurrence is commonly spoken of as if it were the most natural thing possible. But I think it is difficult to find instances of it that will bear hostile criticism. It seems to me that in a healthy person inflammation generally runs its course so as to leave no *débris* behind it, except, indeed, when an abscess forms and fails to discharge itself. As regards the spleen and the lymph-glands, I shall have to discuss this question again when I come to speak of the diseases of those organs. In the solitary follicles of the intestine it used to be supposed that caseation was often met with, quite independently of the tuberculous process, but my own observations have taught me that it is scarcely ever seen unless other parts of the body contain tubercular lesions. One of the most typical features of tuberculosis seems to me to be the presence of spreading ulcers in cavities, with yellow walls of definite thickness, the substance of which regularly undergoes caseation almost as soon as it is formed. This process, wherever found, whether as a pulmonary vomica, or in the liver, the kidney, the prostate, or the testicle, may, I believe, be safely set down as tuberculosis.

Those pathologists who have recognised that caseation is especially apt to occur in tuberculous lesions have commonly attributed it to the absence of blood-vessels in tubercles. Thus Virchow's comparison between tubercles and the Malpighian bodies of the spleen was objected to, and with justice, on the ground that the latter contain capillaries. Every museum of morbid anatomy contains injected specimens of phthisical lungs, from which it appears that not only isolated tubercles, but also patches of caseous infiltration, are absolutely non-vascular. No new vessels seem to be formed in them, and the pre-existing vessels of the pulmonary tissue undergo obliteration. The further process of softening and liquefaction, by which vomicæ are formed, appears to be essentially of a chemical nature.

Fibroid transformation.—But in many cases, as I have already mentioned, tubercles, instead of caseating, undergo fibroid transformation by their cellular elements developing into fibrous tissue. What determines them to take the one course rather than the other is as yet uncertain; but it would seem that caseation is less frequent in proportion as the age of the patient is older. In organs other than the lungs, comparatively little is known about "fibroid" tubercles. But Schüppel has demonstrated their occurrence in lymph-glands, and has shown that the reticulated stroma grows at the expense of the cells, and is converted into a transparent, almost homogeneous, indistinctly fibrillated material. And Rindfleisch has recorded a remarkable specimen of fibrous tubercles in the great omentum. The occurrence of a similar change in tubercular infiltration is, I believe, the origin of the affection which is known as fibroid phthisis. Even in such cases, there seems still to be deficient vascularisation; for Rindfleisch has insisted on the impossibility of forcing injection into the interior of indurated masses in the lungs, and contrasts their state with the abundant supply of vessels to the newly-formed

connective tissue in cirrhosis of the liver and in granular diseases of the kidney. No writer has laid more stress on the fibrous transformation of tubercles than Dr Hamilton, who, indeed, declares that the natural ultimate destiny of these bodies is to become little fibrous tumours, and that many of the so-called cirrhoses of organs, both in children and in adults, are in reality the remains of outbreaks of tubercle.

The tubercle-bacillus.—In the 'Berliner klinische Wochenschrift' for April 10th, 1882, Dr Koch made known the very important fact that he had discovered bacilli in the tubercular diseases of man and of animals,* and that he had succeeded in cultivating these bacilli upon the coagulated serum of blood, and had been able to set up tuberculosis in healthy animals by inoculating the cultivated products. The organisms themselves he described as in length equal to a quarter or half the diameter of a red blood-disc (3–5 μ), and in breadth one fifth to one sixth of their length. They are slightly curved and have rounded ends. When prepared by a particular method of double staining,† they present a blue colour, whereas nuclei and other tissue-elements appear brown. This not only facilitates their recognition, but also serves as a point of distinction between them and all other kinds of bacilli, except those of leprosy, which, indeed, resemble them very closely, and only differ in being still more slender and in having pointed ends. The bacilli of tubercle, while lying in the living tissues, produce spores which are oval in form, from two to four in number, and placed at equal distances in their length so as to give the bacilli a beaded appearance. When grown upon the coagulum of blood-serum, the organisms become aggregated together into flat, scale-like masses, hardly as large as poppy seeds, which can be lifted off entire, and which are so firm that it requires some force to break them up. Their development takes place very slowly, about ten days elapsing after infection of the coagulum before any change is discoverable. Another condition necessary to their growth is a uniform temperature of 86°—106° F. Hence they cannot grow in the open air in this climate.

Koch does not think that previous observers who have described micrococci,‡ or even rod-shaped bodies, in tubercle, had seen the same organisms which were discovered by him. They are not generally present in large numbers, except where the tuberculous process is recent and active; if it is slow, they are often to be found only within the giant-cells, perhaps one or two in each cell. They appear to possess no power of spontaneous movement.

The cases in which Koch found his bacilli were the following: (1) Eleven cases of miliary tuberculosis. In miliary tubercles of the lungs they were never absent, but in those which had caseated they often existed only at the edges. They were present, too, in miliary tubercles of the spleen, liver, and kidneys, and in great numbers in the grey tubercles of basal meningitis. And they also occurred in the cheesy bronchial glands from the same cases. (2) Twelve cases of caseous bronchitis and pneumonia. (3) One case of solitary tubercular tumour of the brain, the bacilli lying within giant-cells

* There is still some doubt as to the identity of Perlsucht in cattle (v. *infra*, p. 89) with tubercle in man. Klein finds that the tubercle-bacilli in man are much larger than those in the tubercular lungs of cattle.—Ed.

† Viz. by methylene blue and vesuvin. Ehrlich has since devised a more delicate method in which the bacilli appear magenta-red with fuchsin (hydrochlorate of rosanilin). Weigert, Heneage Gibbes, and many other histologists have described methods of double staining. See an account of them in Dr Crookshank's 'Introduction to Practical Bacteriology,' pp. 162-7.—Ed.

‡ Prof. Toussaint, of Montpellier; Prof. Klebs, of Prague. See Cheyne, 'Practitioner, April, 1888.

contained in the tissue immediately surrounding the cheesy mass. (4) Two cases of intestinal tuberculosis. The bacilli were present also in the corresponding mesenteric glands. (5) Two out of three cases of freshly extirpated scrofulous glands. (6) Two out of four cases of inflammation of joints.*

Previous experiments on the artificial production of tubercle.—Koch's discovery constitutes the final link in a chain of evidence which has been long accumulating, and which places tuberculosis among the specific diseases. In 1865 a French observer, M. Villemin, made known a fact which roused the greatest interest in the medical world; he found that in certain animals, particularly rabbits and guinea-pigs, tuberculosis could be set up by the inoculation of tuberculous matter from the human subject. His experiments were soon repeated in this country by Mr Simon, Dr Sanderson, Dr Wilson Fox, and others. Tubercles from the pia mater or from serous membranes, caseous substances from the lungs of phthisical patients, even the sputa yielded by such patients during life, were introduced beneath the skin of animals. These were afterwards killed, or were allowed to die of the disease which followed the operation and generally proved fatal in from six to ten weeks. It was then found that pus or a dry cheesy substance had been formed at the seat of inoculation, and that bands of induration extended away from it into the adjacent subcutaneous tissue. The lymph-glands in the neighbourhood were enlarged and caseous. Nodules of various sizes were present in the lungs, the liver, the spleen, and the peritoneum. These resembled tubercles both to the naked eye and in their histology. They consisted of a grey, semi-transparent material, which underwent caseation from the centre outwards. The infective character of the process was further shown by the fact that matter taken from the body of an animal after death was capable of setting up the same disease in other animals.

After a time, however, certain observations were made which seemed to alter completely the bearing of these experiments. Dr Sanderson found that in guinea-pigs an "artificial tuberculosis" arose after the inoculation of pus from the secondary abscesses of pyæmic patients, or even after the mere introduction of a clean seton of unbleached cotton. Dr Wilson Fox independently arrived at a like result by inoculating guinea-pigs with pieces of putrid muscle, or with vaccine lymph. In Germany, Cohnheim and Fränkel introduced into the peritoneal cavity of animals of the same species portions of new growths, pieces of healthy organs from the human body, or even bits of charpie or gutta-percha; and they succeeded in setting up a tubercular process not only in the serous cavity itself, but also in the lungs and in the liver. It even happened that guinea-pigs in which incisions were made, without any injection, perished afterwards with inspissated abscess at the seat of injury, and with miliary tubercles in their organs; so that it became necessary to give up experimenting upon such animals, and to have recourse to dogs, as not being liable to be made tuberculous by trifling wounds. But, after injecting into the jugular veins of these dogs pus from guinea-pigs which had themselves been inoculated with non-tuberculous materials, it was found that the dogs likewise became the victims of miliary tuberculosis.

These observations fitted in perfectly with a theory of tuberculosis which had been propounded a few years before the publication of Villemin's experiments, but which at the time had attracted little notice. In 1857, Buhl, of

* In the twenty-first volume of the 'St Bartholomew's Hospital Reports,' Dr Harris states that in twelve ancient specimens of tuberculous lungs, from forty to seventy years old, he succeeded in demonstrating the presence of Koch's tubercle microscopically.—Ed.

Munich, had suggested that in the human subject acute miliary tuberculosis was due to the absorption into the blood of caseous matters from non-encapsuled yellow tubercles, or from pulmonary cavities not surrounded on all sides by dense fibroid tissue. Indeed, a similar opinion had been expressed earlier still by Dittrich, of Erlangen. It afterwards became the fashion to regard tubercle as always a secondary product, the origin of which was sought for in "caseous foci," of which the formation was supposed to precede, in all cases, the development of tuberculous lesions. In ordinary cases of phthisis it was thought that a catarrhal pneumonia, with secondary more or less extensive caseation, was the earliest change, and that this might go on for a great length of time before any tubercles made their appearance, so that perhaps the disease might become tubercular only at its very end. Such a view was of course in entire accordance with the teachings of Niemeyer already referred to (pp. 79, 83). The acute tuberculosis of children was traced back, in a large number of instances, to a simple intestinal or bronchial catarrh; this was supposed to lead first to swelling of the corresponding abdominal or thoracic lymph-glands, and then to their caseation; and when once caseation had commenced the conditions for the development of tubercle were assumed to be present. Dr Hamilton has recently maintained this doctrine with great vigour; he imagines that in the process of caseation some material, probably a ferment, is elaborated, which acts as an irritant upon the tissues to which it is carried. Now, I have always taught that such a theory of tuberculosis is altogether inconsistent with the broad facts of human pathology, whatever might be its application to the "artificial tuberculosis" of animals. Nothing is more certain than that, in man, the inspissated pus of a common abscess, or the caseous matter of an atheromatous artery, or of a degenerating new growth or gumma, does *not* produce tuberculosis. The few cases that have been recorded as illustrations of such an occurrence serve, by their rarity, to tell against the conclusion they are intended to support. Thus Dr Murchison some years ago exhibited at the Pathological Society a specimen in which a simple ulcer of the duodenum (an affection generally entirely free from all trace of caseation) had been followed by tubercular disease in the lungs. Is there the smallest reason for supposing that this was anything more than an accidental coincidence? Dr Hamilton insists upon a case in which a woman died, thirty-three days after her delivery, of general miliary tuberculosis. She had been attacked by a rigor on the seventh day, and her symptoms were at first mainly those of peritonitis. In many places, says Dr Hamilton, more especially behind the uterus, the peritonitic fibrinous effusion had become caseous, and here and there softening had occurred. And he assumes that this was the starting-point of the tuberculosis. But it seems to be very doubtful, according to observations which we shall presently find to have been made by Cohnheim with regard to the rate of development of acute tuberculosis, whether it would have been possible for the process to have run its course within twenty-five to twenty-six days, even supposing that the tuberculous "ferment" became "elaborated" at the very moment when the peritonitis first began. And, after all, there is not the slightest difficulty in supposing that the woman's illness was, from the first, an acute tubercular peritonitis, forming, as so often happens, the clinically obvious part of a more widely diffused miliary tuberculosis. I must therefore repeat that cases of acute tuberculosis in man, attributable to infection from non-tubercular lesions, are so scarce that it is impossible to attach any significance to them.

Meantime, however, it has turned out that the experiments which were supposed to prove that tuberculosis could be set up in animals by the inoculation of non-tuberculous matters had been misinterpreted. Klebs showed that in all probability they were vitiated either by the accidental presence of the specific poison of tubercle at the time when the operation was performed, or by the wound becoming infected with it afterwards. Cohnheim afterwards frankly acknowledged that, having repeated his investigations both at Kiel and at Breslau, he has utterly failed to obtain the same results as before; and he is now among the warmest supporters of the specific nature of tubercle. On referring to Dr Wilson Fox's observations I find that all the animals which were inoculated by him, whether with tuberculous or with non-tuberculous substances, were placed in a single house, so that it was quite possible for them to have infected one another accidentally. Moreover, Dr Fox himself points out that tuberculosis was produced with far more certainty by the inoculation of tubercle than by the other methods which he employs.*

Cohnheim made a further advance, by showing that in rabbits and in guinea-pigs tuberculosis has a tolerably definite period of incubation: from fourteen to twenty-one days. When a minute fragment of tuberculous matter is introduced into the anterior chamber of a white rabbit's eye through an incision in the cornea, the slight reaction which arises after the operation quickly subsides, provided that the tubercle is perfectly fresh. The fragment can now be seen through the transparent cornea, and it diminishes in size day by day until it may altogether vanish. Then, at the end of the period of two or three weeks, an eruption of small transparent grey granulations appears on the iris. Afterwards they caseate, and a destructive inflammation of the whole eyeball often results. Ultimately the animal may die of a general miliary tuberculosis, in from five weeks to three months, or later; or the diffusion of tubercles may be limited to the lungs or to the peritoneal cavity; or, lastly, there may be no development of them in any organ outside the eyeball.

To complete my account of the experimental study of tuberculosis in the lower animals, I have to describe two sets of observations, of which the object has been to illustrate certain methods by which the specific virus of tubercle may be supposed to make its way into the human body.

(1) One of them relates to the disease of cows and oxen, which is known in Germany as *Perlsucht*, and which is now admitted by most observers to be identical with tuberculosis, although the morbid appearances, as seen by the naked eye, differ considerably from those with which we are familiar in man. It is a chronic affection, which may run on for some years without impairing the animal's health. In fact, milch cows, particularly older ones, afford almost the only instances in which any symptoms appear during life: these are chiefly cough and wasting. It appears that animals thus affected are often sold to the butcher, so that beside the risk of drinking their milk during the early part of their illness, there is the further risk (at least to the poor) of actually eating the diseased tissues, perhaps concealed in sausages. The frequency of Perlsucht may be judged from the fact that at

* At a meeting of the Pathological Society, on December 4th, 1883, Dr Fox stated that at his request the experiments referred to in the text had been recently repeated by Dr Dawson Williams. The details of this series of experiments were stated, with the precautions against accidental infection: the results were absolutely negative. Dr Fox mentioned some possible sources of fallacy in his first series of inoculations, one of which was that suggested above by Dr Fagge.—Ed.

Augsburg 2 per cent. of all cattle slaughtered were proved to be tuberculous, and of cows as many as 5 per cent. Its experimental propagation in other than bovine animals has been studied by Gerlach, Klebs, Orth, and others. Not only have pigs, lambs, rabbits, and guinea-pigs been successfully infected by the milk from diseased cows, but a like result has been attained by feeding animals with portions of the affected tissues removed after the cows were slaughtered, and also by inoculating them beneath the skin with such materials. Klebs found that even boiling the milk did not, in two experiments, render it inert. A further step in the investigation has been taken by Bollinger, who thinks that a broad distinction can be drawn between the Herbivora and the Carnivora as regards their liability to be affected with tuberculosis through the alimentary canal; he supposes that animals of the latter kind secrete a gastric juice which has a more active power of destroying the virus. Man, as an omnivorous creature, ought (like the pig) to occupy an intermediate position.

One is strongly tempted to find in these experiments an explanation of the frequency of mesenteric disease in children, in whom milk forms so important an article of diet. But it seems to be a difficulty that no one has yet noticed the epidemic prevalence of the disease in the inmates of a nursing establishment, or among the children of the customers of any particular dairyman. The only approach to such an observation that I have met with is one recorded in 1878 by Dr W. H. Spencer, of Clifton. More than twenty boys in an industrial school fell ill of what was supposed to be enteric fever. Most of them recovered, but four died; and in each fatal case the organs were found to be full of tubercles. It is, however, to be observed that in every instance caseation of bronchial glands was present, which could not have occurred within the duration of the illness of the boys, this being only three or four weeks.

Dr Creighton has recorded several cases in which he believes that post-mortem appearances found in the human subject justify the conclusion that the disease was derived from the cow. His point of view, however, is that Perlsucht, or bovine tuberculosis, is not identical with the tuberculosis which chiefly occurs in man, and that it will henceforth be possible to separate by certain characters cases having such an origin from those which may be regarded as proper to the human species. And he cites Gerlach as having stated that in some of the animals that were infected by him from the cow the "peculiarity of Perlsucht was unmistakable." In addition to there being grey translucent tubercles, like those which are seen in man and in apes, the characters on which Dr Creighton relies are the following:—The peritoneum and the pleura present round or oval nodules, of the size of lentils, sometimes pendulous, sometimes connected with one another by fibrous cords, sometimes flat and cohering together so as to resemble the duckweed on the surface of a pond. In the lungs there may be soft, whitish masses up to the size of a walnut, some round, others wedge-shaped and like infarctions. These organs may also have in their interior closed smooth-walled cavities, giving them somewhat the appearance of a crumpet. It is to be observed that most of the cases in which Dr Creighton found such lesions occurred not in children, but in adults of all ages. Now, I must confess that the prospect of our being able in future to distinguish from other cases of tuberculosis in man such as are of bovine origin, does not seem to me to be promising. But even if what Dr Creighton terms the "structural mimicry of infection" really goes so far as to give special characters to bovine cases, it

yet would not follow that the two diseases are fundamentally distinct. Koch found his bacillus in ten cases of Perlsucht, chiefly in the interior of giant-cells surrounding the calcified nodules in the lungs, but also in the bronchial and even in the mesenteric glands.

(2) The other set of experiments was made by Tappeiner, in order to determine whether tuberculosis could be induced in animals by the inhalation of the sputa of phthisical patients diffused in the air as a spray. His method was to employ six grammes of the sputa at a time, and the inhalations were continued for six hours a day during fourteen days in succession, the animals being placed for the purpose in a narrow wooden box. Dogs were used in this inquiry, and the earliest period at which tubercles were found in the lungs after the commencement of the inhalations was on the twenty-third day. In no single instance were the results negative.

Bearing of the above experiments on human pathology.—If we now turn to consider the bearing of these experimental observations upon tuberculosis, as it is seen in the human subject, we shall, I think, be driven to the conclusion that they are applicable only within very narrow limits.

There are two questions to be discussed, which must be carefully kept apart, although they are often confounded together. One concerns the ætiology of tubercular diseases, the other refers to the way in which acute and chronic tubercular lesions spread through the body.

(*a*) *As to the origin of tubercle.*—The view that infection from without is the most essential part of the *ætiology of tubercular diseases* seems to me to be altogether inconsistent with clinical observation. It is in regard to phthisis that the matter has been most fully worked out, and I must not anticipate the account of the causes of that disease which I shall have to give when I come to describe it. But I may briefly state that there is the clearest evidence that phthisis does not ordinarily spread from patients to other patients, or to relations, nurses, or medical men. On the other hand, it has been proved that its prevalence is greatly augmented by such conditions as overcrowding and defective ventilation, and also by exposure to cold and wet, and by dampness of soil. Overcrowding and defective ventilation may be supposed to act by lowering the general health. But it is difficult to see how exposure to cold and wet can induce the disease except by their liability to set up catarrhal affections of the pulmonary and bronchial tissues; and I am disposed to think that this must also be the *modus operandi* of dampness of soil. I believe, too, that the affections of the lungs which are due to the inhalation of dust are really tubercular, and not (as is generally taught) of a different nature. Again, hereditary transmission plays a very important part in the ætiology of phthisis.

It is not easy to frame a theory of the ætiology of tubercular diseases which shall accord with these facts. There are probably great differences in the *susceptibility* of different persons to the virus, or, perhaps more correctly speaking, in the *resistance* which they offer to it. And, notwithstanding the enormous mortality from phthisis and the affections allied to it, I cannot doubt that this resistance is far greater in the human subject than in the rabbit or in the guinea-pig. For one must remember that those who die of tubercular diseases have commonly lived for several years, during which time they have most likely been exposed to tubercular infection again and again. It is also necessary to admit that this resistance varies in the same individual at different periods, and that it is not absent even among those in whose

families phthisis is hereditary. Otherwise we should find that when one member of such a family was attacked by the disease it would quickly spread to other members. Now, the fact is, that even if several brothers and sisters die in succession, their illnesses are usually separated by considerable intervals of time. But the principal difficulty is to understand how an affection that is associated with the growth of a bacillus can bear any relation to causes which might be expected to set up a simple inflammatory process. One possible solution is that the microzyme may be so widely diffused that it is continually finding access to the body, but that it fails to implant itself there until the local resistance of the tissues happens to be lowered by the occurrence of inflammation in it. But this seems to be inconsistent with Koch's statement that the bacillus cannot grow unless the temperature is above 86° F. Cohnheim appears to think that the bacillus may remain latent in the body for an indefinite time until some accidental circumstance calls it forth into active growth. He even imagines that the hereditary transmission of phthisis means the direct transference of the tubercular virus, either in the spermatozoa or in the ovum, as virus is transmitted in the case of the *pébrine* of silkworms. But it seems to me that there is a third possibility, namely, that under certain unknown conditions the occurrence of what is at first a simple inflammatory process may cause microzymes already existing in the body to acquire specific properties that they did not previously possess, and to convert the simple inflammation into a tuberculous one. For this hypothesis there are abundant analogies among the acute infective inflammations of which an account has already been given.

(*b*) *As to the spread of acute tuberculosis.*—Let us now pass on to consider to what extent the recent experimental observations upon tubercle throw light upon the *mode of spreading of tubercular lesions within the human body.* It is, I think, impossible to deny that they clear up very many of the difficulties which surrounded this part of the subject.

An acute general miliary tuberculosis, with its myriads of minute centres of tissue-irritation, is hardly explicable in any other way than by the dissemination throughout the blood-stream of minute particles of the tubercular virus in the form of bacilli or of their spores. Buhl himself failed to find a caseous mass which, on his view, could have been the starting-point of general tuberculosis in no fewer than thirty out of three hundred cases; and this notwithstanding that he accepted as satisfactory such lesions as a small embolic plug in an organ, or a degenerating purulent exudation in a serous cavity. The probability now seems to be that the real starting-point is often a very recent patch of tubercles which happens to be so situated that an infection of the blood-stream is a necessary result of its presence. Thus, for instance, Weigert discovered, in many instances of acute tuberculosis, a caseous mass of tubercle, growing into the interior of a pulmonary vein, by extension from the pleura, or the bronchial glands, or the mediastinum. In quoting this observation, Cohnheim alludes to Ponfick's cases of tuberculous infiltration of the wall of the thoracic duct as another possible source of blood infection: this, however, is a very rare lesion.

But it must not be supposed that the growth and the distribution of tubercles, when they are ever so widely scattered throughout the body, are regulated solely by the way in which the bacilli are disseminated through the blood-stream. There seems to be a curious difference between the artificial tuberculosis of animals and the disease as it is seen in men with respect to the distribution of the tubercles. In the former affection they are

found beneath the pleural surface more abundantly than in the substance of the lung, and Dr Sanderson speaks of them as having sometimes the form of blunt cones. In both respects artificial tuberculosis resembles the two other great examples of tissue infection by the blood—pyæmia and malignant tumour. A third point of similarity between the three affections is that in all of them the liver forms a conspicuous seat of the secondary lesions. In artificial tuberculosis Dr Sanderson seems to have found tubercles in the liver more often than in the lungs, but Dr Wilson Fox observed little difference between the two organs in this respect. On the other hand, in the general miliary tuberculosis of man the liver is much less frequently and less obviously affected than the lung, although no doubt the microscope shows that hepatic tubercles are far more often present than used to be supposed. In the lung itself the tubercles are not more numerous beneath the pleural surface than elsewhere, and they do not possess the conical or wedge-like form. But, further, in many if not in most cases of acute tuberculosis, the distribution of tubercles in the lung is such as to show clearly that the proclivities of the affected tissues play a part in determining it. In the upper lobe the tubercles are more abundant, larger, and more advanced in their development than in the lower lobe. They often gradually diminish both in number and in size from the apex downwards. They may be quite caseous above, grey and semi-transparent below. In other words, even when the virus is disseminated by the blood-stream to all parts of the lungs at once, the result is an affection which bears some resemblance to an ordinary local phthisis so far as concerns the distribution of the tubercles. This fact seems to me to be very important, because it helps to explain what would be otherwise unintelligible—the frequent limitation of acute tuberculosis to a single organ or tissue. In tubercular meningitis, for example, it often happens that not a single tubercle exists anywhere except in the cerebro-spinal membranes; and yet one can hardly doubt that bacilli in immense numbers must have been distributed by the blood equally to all other parts of the body.*

(*c*) *As to the distribution of chronic tuberculosis.*—In chronic tubercular affections the mode of spreading seems to be by the gradual invasion of the lymph-channels from point to point. Among the most striking appearances are the lines of progressive thickening and caseation, with or without the formation of distinct tubercles, which run in the course of the sub-peritoneal lymphatics from the floors of intestinal ulcers. It does not often happen that the process of local infection fails to respect an anatomical boundary line between two widely different tissues; but in children I have two or three times seen a continuous cheesy mass, with a festooned growing border, extend from bronchial glands directly through the lung-substance. And once I observed a similar instance in an adult. A still more striking case seems to have been met with by Buhl. A child had caries, with caseation, of the lower dorsal vertebræ; the pleura became adherent, and the ulcerative process spread continuously into the base of the left lung. And Grancher (' Arch. de Phys.,' 1878) mentions a case of tubercular peritonitis, in which tubercles penetrated the diaphragm and infected the pleura covering the inferior surface of the lung, without adhesion having taken place. But in most instances of chronic tuberculosis tissue-proclivities seem to me to play a most important part in determining and in limiting the distribution of the tubercles. It is indeed difficult to see how the bacilli can fail to enter the blood-current in greater or less

* It is, however, possible that, in this very acute disease, infection is carried through the sub-arachnoid space by the movements of the cerebro-spinal fluid.

numbers in any of these cases; and if so, the resistance of the tissues in general can afford the only reason why lesions are not always found scattered irregularly through all parts of the body. Instead of this, the fact is, as I pointed out in the 'Pathological Transactions,' for 1874, that several distinct varieties of chronic tuberculosis can be recognised, each of which has peculiarities of its own, in regard to the organs and tissues attacked. And I would particularly remark that a parallel can generally be found for the distribution of each of these several varieties in the similar distribution of some other non-tuberculous disease; so that one is forcibly reminded of what Darwin calls the "correlated variation" of organs in their first development; as an instance of which he cites the curious fact that male cats which are entirely white, and have blue eyes, are generally deaf.

The following are these local varieties of chronic tuberculosis:

1. Pulmonary phthisis is very commonly accompanied by tuberculosis and ulceration of the air passages (especially of the larynx), and of the intestine (especially the lower end of the ileum). Some observers think that the affection of the lungs directly causes the lesions in question, the larynx being infected by the sputa which pass over it, and the ileum by the sputa which are swallowed. There is some experimental evidence in support of such a view. But it seems worthy of notice that in enteric fever likewise, ulceration of Peyer's patches and the solitary follicles goes with ulceration over the bases of the arytænoid cartilages. And also, in acute pneumonia, ulcers are apt to form at the same spots within the larynx.

2. Dr Wilks long ago pointed out that tubercle sometimes attacks the pleura, the peritoneum, and the pericardium, without affecting any of the solid viscera. I have already remarked (p. 68) that the same distribution is observed in some cases of simple inflammation.

3. Tuberculous disease which began in one kidney not only spreads to an extraordinary extent by continuity along the genito-urinary mucous membrane and the corresponding submucous tissue, but is also commonly attended with like affections of the opposite kidney, and (in the male) of one or both of the testes. I fail to see how this can be adequately accounted for by local infection. And if infection occurs by the blood-stream, tissue proclivities must surely play an important part in the result. Have we not an analogous fact in the occurrence of a "sympathetic" orchitis in a case of renal calculus? There is also a relation between tuberculous disease of the kidney and Pott's disease of the lumbar vertebræ at the same level: but in this instance direct extension probably occurs.

4. Addison's disease of the adrenals (the tuberculous nature of which affection I do not doubt) is sometimes associated with spinal caries at the same level, which, indeed, probably precedes the adrenal lesion in point of time. I have seen two examples of a still more remarkable connection between solitary tubercle of the brain and tubercle affecting the adrenals in the form of one or more scattered cheesy masses. Virchow has pointed out that gliomatous growths sometimes occur simultaneously in the brain and in the adrenals; and I believe that facts have confirmed this observation.

5. Tuberculous disease of the lymph-glands often spreads widely throughout the body, and this not merely by obvious continuity, for it may affect groups of glands widely distant from one another. It is also very generally attended with tuberculosis of the spleen, an organ which is not otherwise very apt to be the seat of tubercles, at least in chronic cases. A precisely similar association is seen in Hodgkin's disease.

6. Several of the joints are often affected by tuberculous disease in succession, without any corresponding affection of other structures. Here again one is reminded of the frequent limitation of pyæmia to joints—a peculiarly apt illustration of the effect of tissue proclivities in determining the localisation of lesions, even when they are due to blood-infection.

In conclusion, I must add that it seems to me an open question whether multiple tubercular affections in the same subject are not sometimes altogether independent of one another. I was once much struck by finding in an infant, six months old, two large masses of tubercular glands, entirely distinct from each other, one being in the chest, the other in the abdomen. And one not infrequently meets with cases in which the distribution of tubercular lesions is altogether capricious, without any one of them being obviously of older date than the rest. Thus I have seen phthisis associated with a tubercular affection of one testis and of the vesiculæ seminales in a man, and in a woman with a similar disease of the uterus and of an adrenal body. In a child who died with a tubercle in the cerebellum there was a mass of caseous glands in the abdomen. Similar instances might be multiplied indefinitely. But of course it is possible that all, or any of them, may have been due to chance infection of the blood-stream, the virus happening to be carried to a spot where it met with no resistance sufficient to interfere with its action.

Historical retrospect.—I have in the present chapter carefully abstained from entering into a historical inquiry into the successive views that have been advanced as to the nature of tuberculosis ; but it may perhaps now be interesting to ask whether any of the writers in the early part of this century expressed opinions approaching those which seem to accord best with our present state of knowledge. As regards the doctrine of the unity of phthisis, indeed, we need not look beyond Laennec. But his conceptions as to the relation between that disease and inflammation may almost be summed up in the statement that inflammation might occasionally be excited by the irritation caused by a numerous crop of tubercles. Nor can I agree with those who would assign a large share of credit to Reinhardt. It is true that he in 1850 identified grey tubercle and grey infiltration with inflammatory processes, but, on the other hand, he maintained that yellow or cheesy tuberculous matter never arose out of the grey, but was merely inspissated pus. Among English physicians, my distinguished teacher, Thomas Addison, is often spoken of as having been the first in 1845 to challenge the views of Laennec. But it seems to me that what he taught was that many cases of phthisis were pneumonic and non-tubercular, rather than that tubercles themselves were inflammatory products.* I find no one expressing himself so clearly on the latter point as Prof. Alison, of Edinburgh, who, in his 'Outlines of Pathology,' published in 1844, stated that "in certain constitutions, tubercles and all their consequences are direct effects of inflammatory action." Still more definite were the views of Dr C. J. B. Williams, who classified grey and yellow tubercles as cacoplastic and aplastic varieties of coagulable lymph, "differing from the normal plasma, not in kind, but in degree of vitality and capacity of organisation." Would the pathological knowledge of his day have admitted of a more exact statement of what seems now to be the truth ?

* "Inflammation constitutes the great instrument of destruction in every form of phthisis." "Pneumonia and inflammatory tubercle are identical" (1841). Addison's works, pp. 40, 64.—Ed.

TUMOURS

New growths—Innocent and malignant tumours—Virchow's characters of malignancy—Malignancy not an attribute of structure—Histological classification of tumours—Malignant osteoid tumour—Sarcoma: round, spindle, and giant cells; hæmorrhagic and alveolar sarcoma: melanotic sarcoma—Multiple sarcomata as a general disorder—Osteitis deformans—Lymphoma—Carcinoma—Heredity—Theory of new growths.

We have seen that, throughout the present century, theories with regard to Inflammation and to Tubercle have been incessantly changing, and have not seldom oscillated backwards and forwards. The doctrine of Tumours has been equally unstable, but with this difference: that the progress of investigation has opened up a succession of views, most of which had never before presented themselves to the minds of pathologists, and, indeed, could not have been understood at earlier stages of the science. The scope of the word Tumour has itself undergone limitation. Once it was applied in practice to all swellings which were not obviously inflammatory, so that it included hydroceles, retention-cysts of all kinds, hydatids, hypertrophies of the spleen, uniform enlargements of the liver, &c. Hence, when attention was first drawn to the fact that the more important Tumours consisted of solid tissues which were altogether of recent development, it seemed necessary to distinguish them by a special name; and they were accordingly termed *pseudo-plasms* or *neo-plasms*, or *new growths*. But, since tissue-formation likewise occurs in inflammation and in tubercle, such an attempt to introduce an accurate nomenclature involves great difficulty of definition. And it is now, I think, becoming the general practice to use the word Tumour in a sense which is sufficiently restricted.

One of the most conspicuous features of a Tumour is that it grows from a centre or centres, paying little or no regard to the configuration of the structures among which it lies. When its seat is a mucous membrane, it often projects from the surface, and may even hang by a more or less distinct pedicle, in which case it is spoken of as a *polypus*. In a solid organ it may form a rounded mass, which is called a *nodule* or a *tuber*. At a time when the life-history of the cystic entozoa was all but unknown, nothing was more natural than that even solid growths should be imagined to have a parasitic origin. This notion, however, has proved without foundation. Their life is that of the individual in whom they develop.

Benign and malignant growths.—It would be impossible to devote the slightest consideration to the study of Tumours without observing that whereas some of them may remain for years without affecting the health or causing any inconvenience, others rapidly destroy the patient who is unhappily affected with them. The distinction between "*innocent*" and "*malignant*" growths is therefore a most natural and obvious one. A little further observation would bring to light the fact that whereas many of the former kind are like the natural tissues of the body—fat, or cartilage, or

bone—many of the latter kind are unlike any healthy tissue. And the microscope accentuated this difference by showing that some soft, white malignant tumours, which by Laennec and others had been compared with brain-substance or marrow and called *encephaloid* or *medullary*, have in reality an intimate structure which bears no resemblance to normal tissues. Thus a fundamental division was made between *homologous* growths, generally innocent, and *heterologous* growths, generally malignant. And it was further supposed that, although there were several varieties of malignant growths, differing in character and appearance, yet that all these belonged to a single disease, which was termed Cancer. The *word* "cancer," or its equivalent *carcinoma*, was of much earlier origin; it dates, indeed, back to Celsus.* But hitherto its tendency to ulcerate and to eat away the natural structures had been regarded as the fundamental character of this affection rather than the presence in it of any new-formed structures. Thus, not only had the venereal "chancre" been confounded with it, but even the "cancrum oris" of children, which we now term noma. Henceforth these were finally separated.

The early pathological histologists, of whom Lebert may be regarded as the chief, saw no difficulty in supposing that the tissue of a heterologous growth might differ completely from all the normal tissues of the human body. They made it their aim to discover some particular specific element or "cancer-cell," the presence of which might constitute an infallible criterion, and solve the frequent difficulties which arose in the anatomical diagnosis of tumours.

But in 1847, and the years which followed, the genius of Virchow placed this question in an altogether new light. It had already been shown by Johannes Müller that the structure of every growth, however heterologous in appearance, always corresponded in its ultimate elements with some natural tissue; if not with any of the permanent tissues, at least with some one of those which exist during early embryonic life. Virchow carried the same idea into full detail. As he pointed out, the physiological type of the tailed and irregular cells which had been supposed to characterise cancer is to be found in the epithelium of certain mucous membranes; while the round- or spindle-shaped cells which really make up the substance of many malignant tumours correspond with those which are seen during the development of connective tissue, or (as Billroth has since taught) of muscle or of nerve-tissue. And, in general, he insisted on the principle that the structure of a

* Cancer is of course the Latin translation of καρκίνος, a crab; and the word was applied by the Greek physicians to any eating, gnawing sore. Both καρκίνος and καρκίνωμα occur in this sense in Hippocrates ('Aph.,' 1257, *et passim*) as well as in later writers. Celsus speaks of diseases, "cum quid intra se ipsum corruptum est, ut in *Cancro*," in close proximity to those which result "cum quid extrinsecus læsit ut in vulneribus" (lib. v, cap. xxvi, 1). A little further on (ibid., xxvi, 31) he says that a cancer ensues from too great inflammation, or too great heat or cold, or too tight binding of a sore, or because the patient is old or of an ill habit of body. "Omnis autem cancer non solum id corrumpit quod occupavit, sed etiam serpit." He goes on to say that the Greek physicians divided cancers into species for which there were no Latin equivalents: as erysipelas, gangrene, &c. He describes (ibid., xxxviii, 2) καρκίνωμα as chiefly affecting the upper parts of the body, the nose, ears, lips, and breasts of women.

Galen admits, however, of καρκίνος χωρὶς ἑλκώσεως ('De tumorum præter naturam,' c. 12), and Paulus Ægineta says, "Cancer tumor est inequalis—interdum sine ulceratione, quem Hippocrates latentem (κρυπτὸν) nominavit." This author derived the name from the distended veins of a cancerous breast resembling the legs of a crab (lib. vi, cap. 45), and so Galen; others from the difficulty of getting rid of it ('Expositiones vocum medicinalium,' 1564.) Our English word *canker* is an older form of cancer and more generally applied. It is used in the Authorised Version of the New Testament to translate the Greek γαγγραίνα (2 Tim. ii, 17).—Ed.

tumour in man must necessarily be human; so that, for instance, such a tumour might contain hairs but never feathers; whereas, in a bird a tumour might have feathers in it but not hairs. For Virchow, therefore, no growth was *heterologous* in the sense hitherto attached to that term. The distinction, as he drew it, was between tumours resembling in structure the tissues in which they were placed, and those differing from them. Thus, he said that the same growth, which in one situation would be homologous, would in another situation be heterologous. And he further pointed out the necessity of recognising that between certain tissues of the body there naturally exist close relations which are altogether wanting between them and other tissues, and that such relations have important bearings upon pathology. Thus, all the structures belonging to the connective-tissue series being physiologically related to one another the presence of bone, or of cartilage, or of fat in fibrous tissue, would not be inconsistent with homology, whereas that of epithelium would be. Such views have since undergone many further developments, to which I shall presently have to allude.

A still greater advance was made by Virchow in regard to the mode of development of tumours. It had before been generally held that cancer was something deposited from the blood. Chemists had made analyses in the hope of finding in it some peculiar principle for which the name "carcinomatin" had been invented in advance. Its cause was believed to be a "dyscrasia" or altered constitution of the blood. The fact that active tubercle and cancer are but seldom found in the bodies of the same individuals was supposed to support this notion, it being imagined that the blood, or, at least, the formative part of it, could hardly present two different dyscrasiæ at the same time. But wounds and injuries undergo repair in exactly the same way in those who are affected with cancer as in other persons: which could scarcely happen if the doctrine in question were true. Indeed, were that docrine pushed to an extreme, I suppose that all parts of the body ought to become cancerous simultaneously, or at least all such tissues as are undergoing constant nutritive changes, for which they depend upon exudation from the blood.

Virchow, in accordance with his dogma *omnis cellula e cellulâ*, maintained that the substance of a cancer was developed *in situ* by the growth of the cells and other structures of which it was composed. He was, indeed, mistaken in the view which he advanced that they arose by a proliferation of the connective-tissue corpuscles. But, after all, this was an insignificant detail in comparison with his rejection of the notion that cancerous and other tumours were merely "deposits."

One necessary consequence of Virchow's conception of Cancer was that the so-called cancerous cachexia, instead of preceding the formation of malignant growths within the body, must be a result of their actual presence. And, so far as I know, no one has since appealed to definite clinical experience in a contrary sense. No doubt one of the earliest signs of an internal tumour is sometimes the fact that the patient becomes cachectic, that his skin acquires a waxy yellow colour, and that his features look pinched and sharp. But in such cases the tumour really exists long before the indications of it have appeared. It wears down its victim by pain and distress of mind even if does not attain a large size or undergo ulceration and hæmorrhage. Lastly, it is certain that some innocent growths, when they are so placed as to cause severe pain and to give rise to much bleeding, are accompanied by well-marked cachexia. I may cite, for

example, certain uterine polypi, and even hæmorrhoids and non-malignant tumours of the rectum.

Again, Virchow defined the term *malignancy* far more clearly than previous writers. Hitherto the pain caused by a Cancer, and its tendency to destroy life, had been enumerated among the signs that it was malignant. He set these points altogether aside, and insisted that the following four characters are the real indications of malignancy.

1. The *local progress* of a growth, its tendency to increase by extending into the tissues around it. A point on which much stress has since been laid, is the fact that malignant tumours show little or no respect to the natural boundary lines marking off one kind of tissue from another. Thus I recently saw a case in which a cancer of the bladder, having doubtless started in the mucous membrane, ate its way through the whole substance of the organ, through both layers of the peritoneum, through the muscles and aponeuroses, and all the other structures of the abdominal wall, until it formed an immense ulcerated cavity, reaching from the pubes to the umbilicus.

There is, however, one exception to this power of overcoming the resistance offered by the different tissues, in the fact that malignant growths comparatively seldom penetrate the walls of the larger arteries, whereas they frequently extend into the interior of veins, even into the venæ cavæ. Mr de Morgan remarked that cicatricial structures also sometimes form a barrier to the spread of a cancer, so that when recurrence takes place after an operation the disease creeps along one side of the scar for a considerable distance, without passing over to the other side.

It is a point of some interest that cancers are capable of passing across a serous cavity without previous union between the two surfaces. Of this I observed a very remarkable instance some years ago. The fundus of the uterus being occupied by a malignant growth, the great omentum hung down into the pelvis, so as to lie in contact with it; these parts were not in the slightest degree adherent to one another, but the lower edge of the omentum contained a mass of the disease. It is not improbable that infection in such cases is the result of active amœboid movements on the part of the cells of the primary tumour; Waldeyer ('Virch. Arch.,' lv) has at any rate detected slow changes of form in cells from a cancer of the breast, and in those of a round-cell sarcoma of the axilla, when placed on a warm stage; they did not, however, observe them move from one spot to another.

2. Its *tendency to return in loco* after having been extirpated by the surgeon. This is one of the facts which used to be urged in favour of the constitutional origin of cancer. According to the modern view, it depends upon the circumstance that a malignant tumour always has outgrowths far beyond the area in which the tissues can be seen by the naked eye to be affected. But I ought in candour to add that I have not heard that surgeons who adopt the new theory are generally able to obviate the return of the disease by increasing, to whatever extent, the size of the mass which they excise. A point, however, which is worthy of notice is that in the case of double organs, like the breasts, the removal of the whole of one gland for a malignant growth is not followed by recurrence in the organ on the opposite side, as might certainly have been expected if the constitutional view were correct. Were such an instance ever to occur, one would probably be right in regarding it rather as an example of the independent development of a primary cancer in each breast; for this sometimes, though rarely, happens when no operation has been performed.

3. Its *spreading to lymph-glands.* As a rule, the glands first attacked are those which immediately receive the lymphatic vessels from the diseased part. But sometimes it seems to pass over certain peripheral glands, and to begin by affecting others, which lie nearer to the thoracic duct. Even glands which are altogether remote may become seats of the growth, but in such cases it probably has been brought to them by their blood-vessels. There can be little doubt that the radicles of lymphatics round the margins of a tumour take up from it something which is carried into the lymph-stream and deposited elsewhere. And there is no difficulty in supposing that this something consists of nuclei, or even of cells from the growth; for Virchow has shown that sometimes an entire lymphatic network—beneath the pulmonary pleura, for example, or the peritoneum—may become filled with such materials, so as to appear as if it had been artificially injected ('Kr. Geschw.,' p. 52, fig. 4). Indeed, as we shall presently see, Köster has endeavoured to prove that the "local progression" of cancer takes place along the lymph-spaces. Whether lymphatic channels exist in the interior of tumours is said by Cohnheim to be still open to question, although Van der Kolk long ago, as he believed, discovered them.

4. Its *becoming generalised*, i.e. *spreading to distant organs and tissues by infection of the blood, or, as it is termed, by "metastasis."*

With regard to this point a fundamental distinction must be laid down. It is not the mere multiplicity of tumours which indicates that they are malignant. Sebaceous cysts and fatty tumours are sometimes present in great number and yet are perfectly innocent. Perhaps there is no form of malignant disease in which so many nodules are found as in some cases of molluscum fibrosum. But all such innocent multiple tumours have their seat in some particular tissue, and remain limited to it. On the other hand, cancerous growths may appear in almost every tissue of the body at the same time.

Again, it is a most important feature of most cases in which death has been due to malignant tumours that the pathologist can easily recognise some one mass of the growth as having been the starting-point of all the others, either by its characters, or in consequence of what he knows as to the origin and mode of distribution of the particular kind of neoplasm. Even when such a primary growth is seated in an internal organ, and gave rise to no symptoms during life, it is almost always plainly distinguishable from the rest. The difference consists not so much in its greater size (though this may be sufficiently striking) as in its being of firmer consistence, which suggests that it developed more slowly; or an advanced state of degeneration, or extensive ulceration, may show that it is of earlier date. With regard to certain theoretical considerations which also assist in determining the question I shall have much to say further on. It is, as a matter of fact, exceedingly rare to find in the same case two malignant tumours, with equal claims to priority.

Another curious circumstance is that organs and tissues which are especially apt to be the seats of primary growths seldom have a tendency to be affected with secondary or "metastatic" growths of the same kind. For instance, a primary cancer is very often developed in one of the breasts, but in such cases we do not commonly find that the opposite breast contains any of the secondary cancers when they appear; nor are they met with in the cervix uteri, another locality from which cancer frequently starts.*

* The most common seats of primary cancer are the lower lip, the tongue, pharynx and larynx, the œsophagus, stomach, colon and rectum, the mamma, uterus, penis, and testis,

Again, in many cases the distribution of secondary nodules or tubera is so limited as clearly to indicate in what way they were derived from the primary growth. For example, a malignant tumour in the stomach or in the intestine may give rise to secondary nodules in no other organ except the liver, showing that something was carried by the blood of the portal vein which served to propagate the disease; or, the primary growth being in one of the limbs, the secondary growths may occur only in the lungs, so that the infection obviously took place through the systemic veins and the pulmonary artery. I have already remarked that malignant tumours often penetrate into the interior of veins; there is, therefore, no difficulty in understanding how nuclei, or cells, or even pieces of the growth, may get washed away by the blood-current, and carried to distant parts. Cancerous thrombi form soft colourless masses, parts of which are blended inseparably with the coats of the vessel, while other parts, often much elongated and lobulated, project into its channel, either perfectly smooth on the surface, or coated with a layer of fibrin. Some time ago I saw an instance in which, the primary growth being in the liver, and the hepatic veins extensively involved, the lower border of one lung contained a whitish-yellow wedge-shaped mass, exactly like an infarctus due to embolism from ordinary thrombosis: Dr Pye-Smith, however, under whose charge the case had been during life, found that it was really a secondary nodule of the tumour. Dr Moxon mentions in vol. xviii of the 'Guy's Hospital Reports' that he had twice seen cancer growing in clots which lay within the cavity of the right ventricle at its apex. He does not state that the growth had been brought there by the blood of the systemic veins, but I believe that this was the case. In 1871 Dr Payne showed to the Pathological Society a heart in which both the right auricular appendix and the apex of the left ventricle contained similar cancerous thrombi projecting from between the muscular trabeculæ. In that instance it must be supposed that the infecting agent traversed the pulmonary capillaries to get from one side of the heart to the other.

I must not omit to mention that the distribution of secondary growths is often different from what would have been expected on anatomical grounds. Thus, I examined a case of epithelioma of the œsophagus, in which the lungs were healthy, but a secondary nodule existed in one kidney. Frequently the metastatic formation of cancer is limited to some particular tissue throughout the body, without any obvious relation to the seat of the primary growth. Thus, in a woman with cancer of the breast almost every bone may become cancerous, while all viscera remain entirely free from the disease.

To complete the description of the ways in which malignant growths multiply in the body, a fifth point should perhaps be added to those enumerated by Virchow, namely, the possibility of portions of a tumour becoming *transplanted* when set free upon a mucous surface by ulceration or otherwise. In 1868 Dr Moxon showed to the Pathological Society a specimen in which he thought that an epithelioma of the œsophagus extending into the trachea had in this way infected the lungs. Secondary nodules were seated in the interior of the lower lobes, and not beneath the pleura, as is usually the case. They occupied the centres of lobules, and small tubes could be traced up to them. He also mentions that Mr Simon had exhibited specimens in which

and in these regions secondary cancers are almost unknown. On the other hand, the lymph-glands, liver, lungs, bones, and serous membranes, so frequently attacked by secondary cancer, are very rarely the starting-points of the disease. Nor is this a matter of structure, for the whole epithelial surface of the small intestine is all but exempt from cancer; and some glands, as the parotid, are equally so. With sarcomata no such rule holds.—Ed.

"cancer germs" had appeared to take root in the bladder after descending the ureter from the kidney. But I think that all supposed facts of this kind should be cautiously weighed before they are accepted; for there can be no doubt that the particles detached from the face of a malignant growth are, as a rule, dead and already in a state of decay. It has been imagined that a woman with cancer of the uterus may affect the penis of her husband, but Cohnheim maintains that this never occurs. I believe that no experimental proof has yet been offered of the possibility of inoculating any kind of tumour from one animal to another. There has sometimes been a slight apparent commencement of growth at the spot inoculated, but it has always before long undergone reabsorption.

Malignant behaviour and cancerous structure.—It must be particularly noticed that the above characters which Virchow laid down as distinguishing malignant from innocent new growths, so far from being peculiar to any special kind of tumour, belong also to other pathological processes, such as inflammation and tubercle. These exhibit "local progression," and they sometimes pay no more respect to the natural boundaries of the tissues than does cancer itself. The question of their "recurrence after removal" is seldom or never raised. But each of them "spreads to lymphatic glands." And inflammation, at any rate, assuming the form of Pyæmia, undergoes "generalisation" or "metastasis" through the blood. Surely, then, it is more probable that malignancy should belong to several kinds of new growths, perhaps in different degrees, than that it should be a fixed property of one particular species, isolating it from all the rest. Now, that is precisely the doctrine which Virchow taught, and is perhaps the most important part of his views. Other observers independently pointed out the impossibility of drawing an absolute line of distinction. Sir James Paget described, in 1853, under the name of "*recurrent fibroid*," a growth characterised by an inveterate tendency to return *in situ* after removal, without infecting distant structures. Dr Wilks, in his 'Lectures on Pathology,' published in 1859, made a separate group of "semi-malignant" tumours including, besides the "recurrent fibroid" of soft structures, the "osteo-sarcoma" of bone. And he specially recorded instances in which "myeloid" or "enchondromatous" growths, which were then generally regarded as innocent, appeared metastatically in the lungs. But by these writers cancer was still left as an independent affection, consisting of several varieties, of which the chief were Scirrhus, or hard cancer; Encephaloid medullary, or soft cancer; Epithelioma, Melanosis, Osteoid cancer; and Colloid, or gelatinous cancer. These forms of "cancer" were completely redistributed by Virchow, according to their histological characters. He showed that most instances of "encephaloid cancer," "melanosis," and "osteoid cancer," really belonged to the *connective-tissue* series of growths, in common with Paget's "recurrent fibroid" tumour and many others. Scirrhus he associated with epithelioma, both forms being characterised by cells of an *epithelial* type. Colloid cancer is now known not to be an originally special kind of growth, but the result of a peculiar degeneration.

Virchow's theoretical views have since been substantially adopted by all pathologists. But there are still some physicians and surgeons who think that in clinical practice it is advisable to use the word cancer for malignant tumours in general, without regard to their histology. In this I cannot agree with them. Histological characters appear to me to indicate the true relations of tumours, and therefore to be fundamental characters in their case,

although I hold a different opinion in the case of tubercle. Nor does it seem that any practical advantage is to be gained by using "cancer" as a term of prognosis. We shall presently find that among new growths affecting the interior of the body, the seat in some particular tissue, the age of the patient, and other circumstances, enable one very generally to determine with accuracy what is likely to be the pathological nature of the disease, before we see or handle the affected part. To ignore all these points —which apply to the *histological* classification of new growths—is deliberately to deprive oneself of some of the most important means of tracing out the real origin and course of the disease. In describing the various kinds of tumours I shall frequently have occasion to illustrate my meaning. But one example which has recently occurred to me is perhaps worthy of special mention. An old woman died in Guy's Hospital with cerebral symptoms, and I discovered three tumours in the brain or in its membranes. From their appearance I felt convinced that they must be secondary to a primary growth elsewhere. But I searched every part of the body in vain, until at length I happened to notice that the extreme lower end of the rectum had not been taken out with the rest of the intestine. This, when removed, was found to contain a large ulcer with prominent fungating edges, which evidently was the lesion for which I was looking, although it had given rise to no symptoms during life; even the administration of enemata had failed to lead to its discovery. A microscopic examination of the secondary cerebral tumours afterwards showed that these were "columnar epitheliomata." Now, if I had been able to examine them previously, my task would have been very much more easy, for I should have known almost exactly where to look for the primary affection which gave rise to them.

Classification.—It seems to be impossible to frame a satisfactory classification of tumours, or, indeed, of any group of diseases. But since I must follow some order in my description, I shall adopt the following arrangement, which is very like one proposed by Lücke, in Pitha and Billroth's 'Handbuch d. Chirurgie:'

I. Cystic Tumours.

II. New growths proper.

A. Of connective-tissue type.

(α) Normal or fully developed.

1. *Fibroma.*
2. *Lipoma.*
3. *Enchondroma.*
4. *Osteoma.*

(β) Embryonic.

5. *Myxoma.*
6. *Sarcoma.*

B. Of the type of lymph-glands.

7. *Lymphoma.*

C. Of epithelial type.

8. *Papilloma.*
9. *Adenoma.*
10. *Carcinoma.*

D. Of the type of higher tissues.

11. *Angioma.*
12. *Neuroma.*

Not all of these, however, require to be described, since I only propose to discuss such tumours as fall frequently under the notice of physicians; and, in this chapter, such as are found in various parts at once, so that they cannot fitly be left to a later part of this work. Thus, I shall altogether omit cystic tumours and those conforming to the type of the "higher tissues," and shall only say a few words of others.

New growths of connective-tissue type.—1. *Fibroma.*—This is made up of fibrous tissue, which may be either arranged in loose meshes, or felted, sometimes very tightly. In the former case its substance is soft and succulent; in the latter it may be exceedingly hard, so as to creak when cut through. I shall have occasion to mention the occurrence of fibromata in various organs, as giving rise to more or less serious symptoms by pressure, or in other ways. Here it may be worth while to observe that in the kidneys it is not uncommon for several small growths of this nature to be found, which appear as soft white masses, and which are apt to be taken for secondary tumours, when there is a growth in some other part, the malignancy of which seems doubtful. I have more than once been saved from a blunder only by the microscope; in all other respects these renal fibromata are unimportant.

English writers have, of late, been accustomed to separate, under the name of *Myoma*, a common variety of tumour containing much fibrous tissue, on account of the supposed presence in it of more or less numerous bands of smooth muscle. But all German pathologists seem now to be agreed that these growths, which are seen especially in the uterus and in the prostate, should be replaced among the fibromata; and Rindfleisch declares that the opinion that they contain muscular fibres is really a mistake.

2. *Lipoma.*—This is a tumour consisting of adipose tissue like the subcutaneous fat, but circumscribed, and generally enclosed in a well-marked capsule. When they occur in the interior of the body fatty tumours do not produce symptoms so as to come under medical observation unless they are so large as to press upon important organs, which is very seldom the case. But Dr Frederick Taylor ('Path. Trans.,' vol. xxvii) met with a case in which a lipoma grew in the post-pharyngeal space of a child, and caused death by suffocation. In the abdomen such growths have occasionally reached an immense size, so that they have been mistaken for ovarian tumours, and have been excised.

3. *Enchondroma.*—Tumours made up of cartilage are usually known by this name, although chondroma would seem to be the more correct form. Histologically they differ widely in different cases; the matrix may be either hyaline or fibrous, and the cells may present various characters. As primary growths they scarcely ever come under the notice of the physician. Virchow speaks of multiple enchondromata of the lungs, situated chiefly near their roots, and probably bearing some relation to the cartilages of the bronchia; but these, he says, are generally found quite accidentally in the post-mortem room.

4. *Osteoma.*—This is the technical name for growths which consist of osseous tissue, but it is not very often used. For when a tumour projects outwards from a bone it is called an exostosis, and when it extends inwards (as into a frontal sinus) it is sometimes called an enostosis; and most other bony growths contain soft structures as well, which are regarded as the more essential parts of them. I hardly know in what position an osteoma is likely to be observed in strictly medical practice unless it is as an intracranial exostosis, causing the symptoms of a cerebral tumour.

There is, however, a very remarkable kind of growth, for which the name *Malignant Osteoid Tumour* seems to me the most appropriate, and which is of much interest to the physician, because it not infrequently comes under his notice rather than that of the surgeon. By Sir James Paget it was called "Osteoid Cancer." Virchow termed it "Osteoid chondroma," a most unfortunate appellation, since (as he himself pointed out) the structure of the non-calcified part of the growth is not that of cartilage, but resembles rather that which belongs to periosteum. Moreover, as might have been anticipated, great confusion has arisen from its being supposed to be identical with an "ossifying enchondroma," which is a tumour really consisting of cartilage, the deeper and older parts of which have undergone conversion into bone. Malignant osteoid growths are made up of an exceedingly dense, firm, and tough material, which is of a pale greyish colour, and which cannot be teased out. Under the microscope it may appear more or less distinctly fibrous. Sir James Paget describes the fibres as "crisp and stiff," or as moderately broad, "with uneven thorny edges," and arranged in bundles "looking like faggots." Embedded in this substance are cells, which are rather small, of round or oval shape, without capsules. When calcification takes place, the salts may be deposited in hard granular masses, but in some parts more or less perfectly formed bone may be seen. The calcified growth has a peculiar dull white chalky, or mortar-like appearance. It cannot be cut, and must be sawn through, but it may be "rubbed or scraped into a fine dry powder." Malignant osteoid tumours are most frequent in young subjects. Of nineteen cases collected by Sir James Paget, five occurred in persons between ten and twenty years old, nine in those who were between twenty and thirty. They are generally attended with severe pain. A very favourite seat of them is the lower end of the femur. I have seen one affecting the humerus close to the shoulder. This kind of growth forms a very large, hard mass, surrounding the whole circumference of the bone, slightly if at all nodulated on the surface, and gradually sinking down to the level of the rest of the shaft. Thus it might seem to be outside the bone, but on section the medullary cavity and the cancellous tissue are found to be completely occupied by the opaque, hard substance above described. When a flat bone is attacked by it, the growth is said by Sir James Paget always to project from both surfaces. I have seen a remarkable case in which the two iliac bones were affected symmetrically, each having a large bossy prominence projecting from both the dorsum and the venter, which might have been felt during life. There may indeed be a large number of such tumours growing from different bones in the same patient, and the clinical aspect of the case may be that of a paraplegia from compression, or that of a cerebral tumour. Or, again, the main symptoms may be thoracic, from an immense mass lying at the root of the lungs, or from numerous nodules scattered in their substance and beneath the pleuræ. The secondary growths in lymph-glands and viscera may have an appearance identical with that of the tumours of the bones themselves. They may be calcified in almost their entire extent, only a very little dry, tough, white, fibrous-looking substance remaining round their edges. It was this circumstance which so surprised the older pathologists, who almost imagined that they had before them cases in which osseous tissue itself possessed the property of malignancy.

5. *Myxoma.*—The true relations of this form of tumour were first recognised by Virchow. Older synonyms for it are "collonema," "sarcoma gelatinosum," and "fibro-cellular tumour" (Paget), but in practice it was

probably often confounded with "colloid." It consists of a semi-translucent material, sometimes so soft as to quiver like a jelly, of a bluish or yellowish tint, emitting a sticky fluid when squeezed or scraped. This comes from the intercellular stroma, and owes its viscidity to the presence of mucin. The addition of acetic acid to a thin slice of the growth renders it white and opaque from precipitation of the mucin.

The cells are generally scattered at considerable intervals. They may be round, but they are often stellate, with long processes ramifying in the stroma. Virchow pointed out that this structure is identical with that of the mucous tissue, which in earlier fœtal life occupies the place of the subcutaneous fat, which at birth is found in the umbilical cord, and which constitutes the vitreous body of the eye. Myxomata are seen chiefly by the surgeon, but the physician may meet with them in various situations, as, for example, in the brain. Sometimes they are found in large numbers upon the nerve-trunks, constituting a part of the tumours which were formerly incorrectly grouped together under the name of *neuromata*.

6. *Sarcoma.*—I have already remarked that Virchow referred to the *connective-tissue* series of growths as consisting of embryonic cells belonging to that order of tissues, certain tumours which present a high degree of malignancy, especially those known as "Encephaloid" and "Melanotic" cancers. He grouped them with other tumours of innocent character, or which at most exhibit a tendency to return *in loco* after extirpation, and he adopted for them all the name of sarcoma. His views with regard to them have since been universally accepted; in this country the more readily because no confusion could arise. For the word Sarcoma, although it dates back to Galen,* and had been employed by many subsequent writers down to the time of Abernethy, had fortunately fallen into complete disuse except in the compound form of osteo-sarcoma.

A point on which Virchow laid some stress as distinguishing sarcomata from growths of which the cells were epithelial, was the absence in the latter of any intercellular substance or matrix. And this still holds good in so far, that where the cells of a tumour are embedded in a structureless or granular material, the conclusion may be drawn that they are not epithelial; but the converse is not universally true, for the elements of certain spindle-cell and other sarcomata are often tightly packed together without anything between them. Chemically the matrix of a sarcoma generally contains, according to Virchow ('Kr. Geschw.,' ii, 217), albumen, casein, or mucin, as well as gelatin.

The cells of a sarcoma may be of three kinds:

a. Round-cells.—These vary in character in different cases. Sometimes they are undistinguishable from leucocytes. In other instances they are much larger, and they are then often exceedingly delicate, so that the addition of water to a microscopical specimen may cause the disappearance of all but the nuclei, which then seem to be free.

b. Spindle-cells.—These are the bodies which by French writers had been called *fibro-plastic*, and which had in England been recognised by Paget and others as characterising the class of tumours which he termed "Recurrent Fibroid." They present many differences of size and appearance, but they all agree in having an elongated shape, tapering gradually into a point at each end, or having their ends prolonged as delicate sinuous fibres. Their nuclei are always elliptical, and sometimes narrow and oatshaped. They

* Σάρκωμα ἐστὶ σαρκὸς ἐν τοῖς μυκτῆρσι παρὰ φύσιν αὔξησις, ἐστι δε σαρκώμα τι καὶ ὁ πολύπους.—'Galeni de Deform. medendis.'

are commonly arranged side by side in bundles, or, as it is termed, are "fasciculated."

c. Giant-cells.—Under this name are now described certain bodies, which, however, are rather irregular masses of protoplasm than cells, and which contain round or oval nuclei to the number of ten, twenty, thirty, or even more. From similar elements being found in the medulla of bones, especially during fœtal life, as Kölliker and Robin pointed out, they were until recently termed "myeloid cells," or by French writers *myéloplaxes*. Indeed, the growths in which they occur, and which are comparatively infrequent, were by Paget, Wilks, and others described as a special kind of tumour. Virchow, however, insisted on the fact that such growths always likewise contain spindle-cells, and that therefore their true place is among the sarcomata.

But different kinds of cells by no means constitute the whole of the formed elements of a sarcoma. It also contains blood-vessels, which are sometimes so large and receive so abundant a supply of arterial blood, that it may pulsate, so as to be mistaken for an aneurysm. The walls of the finer ramifications of these vessels are usually themselves of embryonic character, consisting of spindle-cells applied to one another in such a way as to enclose a blood-channel between them. It is, therefore, not surprising that they are apt to allow blood to escape into the substance of the growth, where it may coagulate, so that on section the mass looks like a mere clot. Such tumours, of course, increase in size with extraordinary rapidity; and they were formerly known as *fungus hæmatodes*, a name now happily almost forgotten. In other instances, there are so few cells between the vessels that there may be the greatest difficulty in recognising the presence of any new growth. Thus, in 1877, I made an autopsy in the case of a lad, aged fifteen, who had been under Mr Cooper Forster's care for a fracture of the femur, which seemed clearly to have been caused by external violence. Repair did not take place, an incision led merely to the escape of blood; and amputation was followed by the death of the patient. I found the injured bones and muscles soaked in blood, and at first I had no suspicion of there being any tumour elements. But presently I noticed that the blood-stained appearance of the muscles was limited definitely by convex margins; and the microscope showed masses of delicate spindle-cell tissue in them. In the lungs there were five or six scattered secondary nodules, the size of marbles, and reddish in colour, projecting above the level of the pleural surface. These, however, felt quite soft; and when cut into, each of them collapsed, leaving a cavity surrounded only by a very narrow margin of indefinite-looking tissue.*

Again, instead of being embedded in a merely granular material, the cells of a sarcoma may be supported by a formed stroma, consisting of connective tissue. This, however, is hardly to be regarded as equivalent to the ordinary intercellular substance, for the fibres have no doubt been developed out of a corresponding number of the cells, and have taken their place. The growth is in fact a mixed one, a *fibro-sarcoma*. Sometimes, as Billroth especially has pointed out, there is a regular alveolar structure, exactly like that which characterises the carcinomata. Of late years, since it has been thought that cancers proper can arise only where there is epithelium, such "alveolar sarcomata" have acquired great importance, as enabling pathologists to account for apparent exceptions to this rule.

Still greater complexity is caused by the combination, in the same tumour, of a variety of other structures belonging to the connective-tissue series, such

* For further details of this case see the 'Guy's Hospital Reports' for 1880, p. 17.

as cartilage, mucous tissue, bone or fat. It is a question whether the occurrence of certain rare cases in which a growth, ordinarily innocent, undergoes metastasis to distant organs must not be explained by the presence in it of more or less numerous embryonic cells, the diffusion of which by the blood-stream causes the infection. According to this view all such tumours are "mixed," and should be designated accordingly. I have myself met with an instance in which fibromata of the uterus had led to the formation in the lungs of similar growths in which the appearances regarded by almost all pathologists as indicative of the presence of smooth muscular fibres were as conspicuous as in the uterine tumours themselves. And in another case, that of an infant fifteen months old at the time of death, there were in the liver, secondary to a mixed sarcoma of the neck, a number of dense flat button-like nodules, which consisted almost entirely of well-developed fibrous tissue, although at the margins some spindle-cell elements were with a little difficulty discovered.

Another circumstance which greatly modifies the appearance of sarcomatous growths is the occurrence in them of degenerative changes, of which caseation is the chief.

It is important to inquire whether, without having recourse to the microscope, one can distinguish the different varieties of sarcomata from one another. And I think the reply must be that this is often impossible. Different specimens vary greatly in consistence and in colour; but there is no constant relation between their characters in these respects and their histological structure. Still, I am under the impression that the softest and whitest among them—those which would be termed *par excellence*, "encephaloid" or "medullary"—consist rather of round- than of spindle-cells. The statement is commonly made that encephaloid tumours are almost always sarcomatous, as if exceptions to the rule sometimes occur. But I think that tumours of epithelial type never have so homogeneous, white, glistening an appearance as is presented by many sarcomata; and that the only cases in which they are equally soft are those in which they grow into the interior of a free space, as into the enlarged pelvis of a kidney, or into the channel of a vein, where they meet with scarcely any resistance. On the other hand, the giant-cell sarcomata are often recognisable without difficulty at the first glance: they are blotched, or uniformly tinted with a dull crimson, contrasting here and there with a greyish hue.

In many instances the primary seat of a sarcoma gives a good presumption as to its histological characters. Thus a growth which starts in the submucous tissue of the alimentary canal, or in one of the solid viscera, is but little likely to be made up mainly of spindle-cells. The fasciculated sarcomata belong especially to the periosteum and to the fasciæ. Giant-cell sarcomata, on the other hand, arise usually in the cancellous substance of bones, and spread out the compact tissue over them. They also often appear in the form of an *epulis*, a growth projecting into the mouth from the alveolar processes of the jaws.

Melanotic Sarcoma.—Of all the sarcomata that which is most definitely localised in its origin is the *pigmented* or "*melanotic*." This constitutes the larger part of what was formerly known as "Melanotic Cancer." Rindfleisch, indeed, seems to imply that even when a pigmented growth possesses an alveolar structure it is yet likely to be a sarcoma, belonging to Billroth's "alveolar" form. But most writers allow that true carcinomata are sometimes melanotic, and I have myself examined one specimen (from the skin of the neck) in which the cells lining the walls of the alveoli were distinctly columnar in

form, and arranged side by side, so that their epithelial character could not be doubted. Still, one may for practical purposes continue to describe pigmented growths under the sarcomata.

As a rule, they start from some part of the body which normally contains pigment, generally from the skin or from the choroid coat of the eye, sometimes, according to Virchow, from an adrenal body or a vesicula seminalis.

A curious fact, which was illustrated by a case in my own knowledge, is that the rectum sometimes becomes the seat of a primary melanotic tumour. And white or grey horses, which have a deficiency of cutaneous pigment, are extremely liable to be affected with a pigmented growth situated about the anus or upon the tail, although this is said to be of a comparatively innocent character. Many cases of Melanotic Sarcoma in the human subject are developed from pigmentary moles, which may have existed from birth, and have at any rate remained for years without causing the slightest inconvenience to the patient. Such a mole may begin to grow, or it may itch, provoking the fingers to scratch it; and then, within a very few months, a fatal diffusion of secondary growths from it may occur. Some observers, including Mr Hutchinson, even think that the development of multiple melanotic sarcomata throughout the body may, in the absence of any more obvious starting-point, be accounted for by the discovery upon one of the toes or elsewhere of a flat pigment mark, which may yet show no sign of having taken on active growth. But I should for my own part be disposed to place such cases rather under the head of a general diffused sarcomatosis.

It is to be observed that the distribution of pigment in melanotic tumours is exceedingly capricious. Whole nodules may be uniformly black as ink, but perhaps others in the same case are purely white and medullary, and yet others may be of a grey colour, or streaked or marked with black patches and lines. Where there is no alveolar structure, melanotic sarcomata usually belong to the spindle-cell variety. In many instances they are extremely numerous, and they may be thickly scattered in regions and tissues which are comparatively seldom affected by new growths. Thus there may be many of them along the course of the intestine, with ulcerated surfaces projecting into its interior. And others may be found in the spleen, in the thyroid, in the muscular substance of the heart, and, in fact, in every part of the body.

Mixed with the tumours and nodules there are sometimes black spots or patches, which look as though they were mere deposits of pigment, without any new growth. I have observed this, for example, in the mucous membrane of the stomach, of the renal pelvis, and of the bladder; and I have seen parts of the lungs stained black in a way which seemed to me to be peculiar. Lücke, indeed, says that a careful examination always reveals the presence of cells in spots thus affected. But Thiersch relates a case of melanotic carcinoma of the skin in which at certain points nothing could be discovered but a diffused brown staining of papillæ, the texture of which was perfectly normal. And it is certain that pigment is often excreted in the urine in large quantities, altogether independently of the occurrence of melanotic growths in the kidneys or in the bladder. Two instances of this were brought by me under the notice of the Pathological Society in 1876. It had previously been described by Eiselt and by other foreign writers. Urine so affected may have an olive-green hue when voided; it becomes darker on exposure to the air, and the addition of nitric acid turns it quite

black. It may throw down a deposit consisting partly of minute granules, partly of rounded brown translucent bodies resembling nuclei, partly of casts of the uriniferous tubules, brown or black with pigment. The colouring matter, which is known as *melanin*, can be separated in a pulverulent form, and when suspended in water it remains unchanged for years. Certain of the tumour-masses may be colourless even in cases attended with melanuria. It therefore seems clear that the pigment is originally manufactured, not in the circulating blood, but in the substance of the growths, which are themselves black. It is probably derived from hæmatin, but no transitions in orange or reddish-brown tints are to be recognised. One can only suppose that it is formed in the tumour in such large quantity that a surplus becomes reabsorbed and is carried to the kidneys by excretion. The clinical recognition of melanuria may sometimes be of great help in the diagnosis of an obscure internal tumour, and it must not be confounded with the blackening of the urine which may appear when carbolic acid, creasote, or tar in any form, has been applied to the skin or swallowed.

Multiple sarcoma.—To the physician it is a point of great practical importance that the development of sarcomatous growths in the interior of the body is sometimes accompanied with symptoms such as by no means suggest the real nature of the case. In some instances, the chief thing of which the patient complains is pain, which may either be fixed in certain parts or widely diffused, and may vary in seat from time to time. More or less rapidly advancing emaciation and anæmia complete the clinical features of the disease, the pathology of which sometimes remains obscure to the very last. The following cases are examples.

In 1880, a man, aged forty-six, was under my care in Guy's Hospital, who had originally been admitted for what was regarded as a gouty affection of the right hand and of the left great toe. This had set in about a year before his death. At a later period no evidence of gout could be discovered. He continued, however, to complain of pains, which were called rheumatic, in his right arm, in both shoulders, in the loins, and (especially during defæcation) in the lower part of the spine. He also suffered severely from headache, which, it was thought, might perhaps have resulted from a previous injury. The urine was at one period albuminous, and the case was therefore regarded as one of Bright's disease of gouty origin. Subsequently the urine became normal, and some of those who saw the patient at that time suspected that he was exaggerating his symptoms. However, he grew more and more wasted and bloodless, and his pains became fixed in the iliac fossæ, especially on the right side. The most careful search was made for tumours, which it seemed could hardly escape detection in so emaciated a subject. A few weeks before death hæmaturia occurred, and this of course led to the suspicion of primary malignant disease of the kidney. At last he became unconscious, with rigid flexion of the right arm, and in this state he died. The autopsy showed that sarcomatous growths had occurred to an enormous extent, but that there was nowhere any tumour discoverable by manipulation. In each iliac fossa a layer of sarcomatous tissue was spread out beneath the periosteum, and there was a large quantity diffused over the surface and in the interior of many other bones. One tumour, which was removed, and sawn vertically through, had in its cancellous tissue many rounded masses as large as walnuts. On the under surface of the dura mater there were a number of button-like nodules indenting the brain. One kidney had two small tumours affecting the mucous lining of its pelvis. The

growth consisted of large cells of the most irregular form embedded in a fibrous matrix.

Another case is that of a boy, aged sixteen, who came under my observation when I was clinical clerk to Dr Rees in 1854. He said that he had been strong and robust until eleven weeks before his admission, when he began to suffer from pain, at first in the loins, and afterwards in the shoulders and limbs. There was great tenderness of the whole surface of the body. He had also experienced a sensation of tingling in the area of distribution of the ulnar nerve to each hand. He was a very delicate-looking lad, with a clear complexion, a pink flush on each cheek, a moist skin, a white tongue, and a pulse of 144. The first diagnosis was of subacute rheumatism; subsequently of chronic inflammation of the spinal membranes. He lay for about two months, during which time he became emaciated to the most extreme degree. Towards the last, masses of enlarged glands could be felt through the walls of his contracted abdomen. He continued to suffer excruciating pain on the slightest movement, and there was exquisite tenderness of the skin. One day he became insensible, and had a convulsive attack, after which he lived only three hours. A large mass of medullary growth was found in front of the spine, eroding the vertebræ, and involving the nerves as they emerged from the spinal canal.

Sometimes the growth of multiple sarcomata leads to a still more rapidly fatal illness. In vol. xxv of the 'Guy's Hospital Reports' I have recorded certain cases which were attended with purpura and with hæmorrhages from mucous surfaces. One of the most curious is that of a man, aged twenty-five, who came under Dr Pye-Smith in 1879 for what was supposed to be an attack of rheumatism. He said that on Whit Monday, June 2nd, he had got wet through; and from that time up to his admission, two months later, he had complained of pains in the left shoulder, in the chest, and in the hips. He had been confined to bed for nine days with profuse sweating; the temperature varied from 102° to 102·3°. The skin had an unpleasant sour smell, and a systolic apex murmur was detected on one occasion when he sat up. He went on well for twelve days, when a purpuric rash came out on the chest and the abdomen, hæmaturia occurred, and his eyelids and his scrotum became greatly swollen. Five days later, on August 14th, he died. At the autopsy the scattered spots of purpura still remained visible. Some of them were flat, but others were slightly raised and indurated, and a few had a central pale elevation with a narrow ring of purple discolouration around it. These proved to consist of small round or irregular cells infiltrating the little lobules of subcutaneous fat. In the kidneys there were several white or pinkish sarcomatous nodules. In the lowest two inches of the ileum there was what appeared to be the primary growth, a homogeneous-looking yellow mass, of considerable thickness, involving all the intestinal coats.

Another case occurred in the person of a cab-driver, aged thirty-eight, a patient under Dr Moxon in 1877. Up to five weeks before admission he had always, he said, been a strong man. He then "caught cold from being exposed to draughts." After this he suffered from pains in the back, legs, arms, and chest, chiefly in the joints, and flying from one joint to another. He was restless and hot at night; he had profuse sour sweats; his urine was high-coloured and became turbid on cooling. Five days before he came into hospital he was attacked with severe pain in the back, hæmaturia set in, and purpuric spots came out on the neck, the groins, and the legs. Afterwards he had epistaxis, his gums became sore, and bled. His tempe-

rature had been taken before admission, and was found to range from 100° upwards. On admission he was much blanched and extremely weak, with no perceptible pulse. The nose began to bleed almost immediately, and on the following morning he died. The kidneys were found to contain a large number of white tumours, and there was in the right vesicula seminalis a firm growth, to which they were apparently secondary.

A third case was that of a man, aged twenty-eight, admitted under the care of Dr Wilks in 1872. He said that for some weeks he had had bad headache and neuralgia, that his teeth had been loose and painful, and that his gums had been spongy and had bled. For ten days he had been suffering with severe pains in the elbows, the knees, and the shoulders, but these seemed to be diminishing in intensity. He was an anæmic man, with a brown tongue, foul breath, and teeth and gums caked with dried blood. His pulse was 128, his temperature 100·2°, his respirations 28. His gums and his nose continued to bleed, and eight days after his admission, raised purpuric spots appeared on the abdomen. On the following day he became unconscious and died. A whitish firm growth was found in the anterior mediastinum, probably affecting the thymus; and there was a large quantity of a similar material in the subserous tissue of the peritoneum and in the mesentery.

A fourth case, somewhat like the others, came under my observation in February, 1876. The patient, a bank clerk, aged twenty-five, said that he had got very cold about a fortnight before Christmas, 1875; for three days he was chilled through, and he was never well afterwards. He visited me in the morning, and I then found his temperature 101°. His mouth and his tongue were stained with blood, his gums were slightly spongy, he had purpuric spots on the legs. It appeared that he was not in the habit of eating any vegetables, and I supposed the disease to be scorbutus. But it soon became apparent that this was an error; he rapidly grew extremely anæmic and feeble, hæmorrhage from the bowels set in, the lymph-glands in various regions of the body became enormously large, and at the end of a few months he died. There was no autopsy.

As I have remarked in my paper on this subject, different views may be taken as to the relation between the symptoms which presented themselves in these various cases, and the underlying malignant disease. It may be that the purpura, the spongy state of the gums, the epistaxis, the fever, and the rheumatoid pains are mere results of a profound alteration of the blood, analogous to that which exists in Idiopathic Anæmia, in splenic Leuchæmia, or in Scorbutus. But another possible explanation of the purpura may be that a minute development of sarcomatous tissue, with vessels made up of embryonic cells, occurs at each spot which is the seat of an effusion of blood; or, perhaps, that sarcomatous cells, or nuclei, or even leucocytes in an abnormal condition, become lodged in the capillary walls, and produce softening of them after the manner of emboli.

In some other instances, scarcely less obscure at the bedside, the diffused development of sarcomatous growths is indicated by other symptoms beside those mentioned in the last few paragraphs. One is the formation of nodules in and beneath the skin, which can be felt and seen. Such nodules, indeed, were present in the case of the clerk, above cited; but I committed the mistake of supposing that they belonged to an accidental "*molluscum fibrosum.*" It would, I believe, be possible to guard against a similar error in the future by remembering the peculiar discoid shape of molluscous growths, which

generally have a flat under surface and a convex upper surface, uniting in a distinct edge or border. In 1876, Dr Moxon had a patient under his care in whom the chief symptom was wasting, until sarcomatous nodules appeared in and beneath the skin of the chest and of the limbs, and cleared up the diagnosis.*

Another symptom of the formation of multiple sarcomata, especially when they affect the bones, is a gradual enlargement of certain bones. Thus, in one of my cases the calvaria was found to be very thick, and its whole substance was homogeneous and opaque; the bodies of the vertebræ, the ribs, and one *os innominatum* were also found to have their cancellous tissue converted into a dense material. Similar changes were observed in two instances recorded by Dr Goodhart in vol. xxix of the 'Transactions' of the Pathological Society; and Dr Cayley relates in the same place the case of a man who came under Mr Nunn for chronic enlargement of the lower jaw; his left clavicle was also much thickened, and both tibiæ curved forwards. This patient died of malignant disease of the lung and of the liver. But the most remarkable instances are those which were brought by Sir James Paget under the notice of the Royal Medical and Chirurgical Society in 1876, under the name of "Osteitis deformans." In these the diffused osseous change seemed to precede the development of any local tumour by many years. Thus one case was that of a gentleman who, at the age of forty-six, became subject to aching pains in the lower limbs; the left tibia grew nodular, uneven, and broad; the skull gradually enlarged, so that its circumference increased in the course of twenty years or more, from $22\frac{1}{2}$ to $27\frac{3}{4}$ inches; the spine became curved and shortened and stiff. He died at last of a malignant tumour of the left radius. The calvaria was found to be of about four times the normal thickness; its texture was in parts finely porous, in parts compact. Another case, very similar in its character, ended after many years in the formation of a medullary tumour of one humerus. Probably there is an analogy between this simple affection of the bones, leading ultimately to fatal malignant growths, and the diffused ichthyosis of the tongue which terminates in cancer. To the pathologist it may probably continue to appear necessary to regard the successive stages of each disease as distinct. But from a clinical point of view, it is important to keep before one the significance of a thickened tibia or clavicle in an obscure case, that may possibly be one of sarcomatous growth; and it may be of great value in diagnosis to elicit the fact that a patient's hat has become too tight for him, or to observe an alteration in his attitude, the chin projecting forwards and downwards, the arms hanging, and the legs being shortened and bowed outwards, as in Paget's cases, and in one which had been recorded some years before by Wilks.

I have already remarked that the occurrence of a large number of melanotic sarcomata in the same patient sometimes appears to be independent of any infective process—that, in fact, they are multiple rather than malignant. The same thing may, I believe, be said of other forms of sarcoma as in the above cases, and likewise of the lymphomata, which are next to be described. Not only is it often impossible to discover any one growth which can be regarded as standing towards the rest in the relation of primary

* I have since seen a case which was believed to be of a similar kind, until at the autopsy the subcutaneous tumours were proved to be cysticerci. Their real nature might, however, have been recognised, if one had thought of it, by their peculiar shape and size; they were firm elliptical bodies, very like a French olive in outline, but somewhat smaller.

to secondary; but the tumours themselves may fail to present Virchow's first indication of malignancy, that of "local progression" without respect to the natural configuration of the parts affected. For instance, I have seen a sarcoma of an undescended testis, weighing eight pounds, which had on its outer side a detached piece, bearing the very same relation to it in position and in size which the epididymis has to the healthy organ. And in another case one ovary, although of normal shape, was of about twice the length of the opposite ovary, and its substance was opaque, white, and made up of rounded sarcomatous cells. Another point, which is perhaps worthy of notice, is that in young adults who die of sarcomatous growths, the thymus is often found persistent, if not rejuvenescent.

7. *New growths of the type of lymph-gland tissue.*—Among the primary forms of tumour, in Virchow's classification, is one to which he gave the name of *lymphoma*, and of which he defined the structure as being identical with that of a lymphatic gland or of a solitary follicle: the leuco-cytogenic or "adenoid tissue of His." It is made up of cells, which lie in the meshes of a stroma. The cells are leucocytes, and can be more or less readily removed by pencilling a thin section of the growth. The stroma, which then becomes visible, is characterised by being "reticulated;" that is to say, its fibres branch and unite together, many of the points of union being occupied by nucleated cells which are fairly compared with fixed connective-tissue corpuscles, triangular, or stellate in form.

The majority of the lymphomata enumerated by Virchow really lie beyond the definition of tumours, as we now understand it. He included among them not only tubercle, but even the transitory growth to which enteric fever gives rise in the follicles of the intestine; just as under the term "granulation-tumours," he described the various lesions which are produced by syphilis, leprosy, and glanders. In fact, he seems almost to have aimed at gathering together, for the purposes of his great work, all the more important structural changes which belong to the science of pathology. This extended sense of "lymphomas" as of "tumour" generally, is incompatible with the order which I have found it necessary to adopt, although, as I have already admitted, it would be far from easy to define the word tumour scientifically.

Even when the lymphomata have been reduced within very narrow limits, they still constitute a vague and unsatisfactory class of new growths. Dr Green, in his 'Introduction to Pathology,' says that they include three varieties: simple lymphoma, lymphadenoma, and leuchæmia. Two of the examples which he cites of "simple lymphoma," are cases of enlargement of one or more of the lymphatic glands in some particular region of the body, and three are cases of overgrowth of the intestinal follicles, forming polypi. But I think it would be very difficult to say how many of the cases in question may not really be of an irritative or inflammatory origin, or else may not belong to the early local stage which sometimes occurs in Hodgkin's disease. And as for "leuchæmia," I shall hereafter have to discuss whether the lymphoid growths found in that affection are not merely accidental, the result of the leucocytes with which the blood is overcharged being deposited in the interstices of the tissues. There remains, therefore, only the third of Dr Green's varieties, that which he terms "lymphadenoma." This word is one which has tended greatly to confuse the whole subject. By many French observers, including Cornil and Ranvier, it is used to include all

forms of tumour, of which the structure is identical with "adenoid tissue." On the other hand, few German pathologists employ it at all. And we should, I think, be strictly correct in stating that its true position in the nomenclature of disease is that of being a French equivalent for the German term lymphoma. Dr Green, however, like several other English writers, applies it in a more limited sense, to correspond with what I shall describe elsewhere as "Hodgkin's disease." In that morbid condition there are, indeed, growths which spring up in immense numbers, and sometimes with extreme rapidity, not only in the lymphatic glands and the spleen, but in almost every organ and tissue; and they may possess at least the first of Virchow's characters of malignancy, spreading from one part to another without regard to the natural boundary lines between them. But it is nevertheless impossible to reserve for them any such name as "lymphadenoma," or even to describe them under any one pathological designation. For, as we shall hereafter find, their histological characters vary very widely in different instances, so that we must regard "Hodgkin's disease" as a clinical name for a group of cases in which the same organs are affected, and of which the symptoms and course are similar, but not as anatomically denoting any one special kind of tumour.

This brings me to another term, "lymphosarcoma," which also needs to be explained. Like lymphadenoma, it has been employed in somewhat different senses by different writers. Some seem to apply it to any sarcomatous growth having its starting-point in lymphatic glands; they forget that the seat of a sarcoma is in itself a matter of indifference, and certainly does not require to be marked by any special name. On the other hand, Virchow is disposed to designate as lymphosarcomata all lymphomatous tumours of any size, of which the elements show no tendency to caseate, and which undergo progressive and sometimes very rapid enlargement; he would include under this head most instances of Hodgkin's disease, and all the lymphomatous tumours of the mediastinum. But it is certain that many of the cases in question, even where a large number of organs are involved, exhibit no histological characters other than those of a pure lymphoma. And it seems to me that the only logical course is to reserve the name of lymphosarcoma for such growths as appear to be transitional between a lymphoma and a sarcoma. Lymphoma will then denote a tumour composed of lymphatic, adenoid, or cytogenic tissue.

New growths of epithelial type.—As I have already remarked, we owe to Virchow the important doctrine that the "cancer-cell," which was believed by histologists to be heterologous, *i. e.* unlike any of the elements of the healthy body, is, after all, identical with the cells of epithelium. He explicitly pointed out this fact in a paper in the first volume of his 'Archiv,' published in 1847; and at the same time he indicated the region in which the most perfect representatives of cancer-cells are to be found, namely, upon the mucous surface of the ureters and of the bladder, especially in infants. He also made a striking comparison between the alveolar structure of a cancer and that of the lung in a case of lobular pneumonia, where the pulmonary vesicles are filled with corpuscular exudation which is just turning into pus. It is a remarkable fact that, at a much later date, pathologists, still ignorant of his teaching, actually committed the mistake of diagnosing malignant growths in the urinary passages or in the lungs, on microscopical evidence alone, in cases where no disease was present.

It is much to be regretted that Virchow did not carry out his usual method of nomenclature, and stamp with his authority the practice of applying the term "epithelioma" to all tumours of epithelial type. Unfortunately it was already in use, as a designation for a very limited group of cancers, characterised, however, not by the presence of ordinary columnar or spheroidal epithelium, but by that of horny epidermic cells. But I believe that this inappropriate limitation of it will hereafter be given up, and that "Epithelioma" will universally be employed, as many observers do even now, in the wider sense which seems naturally to belong to it.

Epithelial growths, however, are not all of them included under cancers. There are certain innocent forms of them, two of which respectively bear the names of *papilloma* and *adenoma*. These bear precisely the same relation to one another which an intestinal villus does to a Lieberkühn's tubule. The former consists of a solid protrusion outwards of mucous membrane or of skin, upon which epithelial cells are arranged; the latter is a hollow inversion of mucous membrane or of skin, with an epithelial lining. They each come under the notice of the surgeon rather than of the physician; but I may briefly mention certain instances of them.

8. *Papilloma.*—Of this we have examples in common cutaneous warts, and in the horny growths which are sometimes seen upon the face, neck, or chest. Another common variety occurs as warts on the genital organs, and a fourth as condylomata and mucous patches; these will be mentioned among diseases of the skin. Yet another form is sometimes met with in the larynx, especially at an early period of life. Lastly, there is an affection to which the vesical mucous membrane is liable, and which is known as "villous tumour" of the bladder, or sometimes (on account of the alarming and dangerous symptoms to which it gives rise) as "villous cancer." It consists in the formation of one or more tufts of long, soft, delicate-branched filaments, each containing a wide vascular loop, and covered with a series of layers of large epithelial cells. I believe that this kind of new growth is never found to spread to lymph-glands, nor to produce metastatic nodules in distant parts. But Billroth still continues to place it among the carcinomata, on the ground that masses of epithelial cells lie in meshes at the bases of the villi. I have myself observed one case in which the resemblance to the structure of a cancer was certainly very close.

9. *Adenoma.*—Of this form of tumour (which resembles true gland tissue, not that of a lymph-gland) an instance may be found in molluscum contagiosum. Other examples also are met with, corresponding some with the sebaceous, some with the sudoriparous glands. Upon mucous membranes the most common variety is one which constitutes a large number of so-called "polypi," growing from the surface of the intestine, of the cervix uteri, or of the nasal fossa. In the female breast adenoma or adenocele, the "chronic mammary tumour" of Sir Astley Cooper, very frequently occurs.

10. *Carcinoma.*—It is to be observed that the structure of the two kinds of new growths last described is by no means always identical with that of the part in which they arise; thus a nodule of molluscum differs considerably from the healthy skin in its histology, and a villous tumour of the bladder from the vesical mucous membrane. And I think that now it is impossible to maintain in its integrity the dictum of Waldeyer, which is cited by Lücke with approval, that the carcinomata can be absolutely distinguished from other epithelial growths by their being "atypical." Probably the real fact is that, as in so many other departments of pathology, no positive line of

demarcation exists. The carcinomata, indeed, may be said to combine in many cases the distinctive features of a papilloma with those of an adenoma, in so far that they consist partly of outgrowths covered with epithelial cells, partly of inversions lined with them. Thus a cancer of the skin often begins in a growth resembling a wart. Many cancers of the stomach, of the intestine, and of the gall-bladder present abundant villous processes. At the os uteri, nothing is more common than for a cancer to begin as what is termed a "cauliflower growth."

In briefly sketching the histology of the carcinomata, it will, I think, be the most convenient plan to begin with what may be termed "*simple or ordinary carcinoma*," the common type of cancerous growths affecting glandular organs and mucous surfaces in general. Afterwards I will describe separately two varieties which occur, the one in skin and mucous membranes lined by laminated pavement-epithelium, the other in mucous membranes lined by columnar epithelium; these may respectively be called "*keratoid carcinoma*," and "*columnar carcinoma*."

a. Ordinary carcinoma presents a great many varieties of structure according to its exact seat. But it is always made up of oval or rounded alveoli, which contain cells like those of epithelium. The walls of the alveoli usually consist of fibrous tissue, and they are more or less abundantly supplied with blood-vessels. In very soft, rapidly growing cancers, however, the interalveolar stroma may be composed of spindle-cells; or, in other words, its tissue may be sarcomatous. Otherwise the softness or hardness of a carcinoma depends chiefly upon the proportion between the amount of stroma and the size of the alveoli. In many instances the substance of the growth is as easy to break down as that of a healthy spleen, or even of a spleen from a case of fever; and then one finds that the alveoli are large and that their walls are made up mainly of blood-vessels, and have but little fibrous tissue in them. But sometimes it is so hard as to be spoken of as "stony;" the microscope then shows that it consists almost entirely of interlacing dense white bands, the alveoli being very small, and perhaps so few in number that their presence may easily be overlooked. It is to such growths that the old name of "scirrhus" is still applied. They are commonly very slow in their course, and they contract and shrink so that the general appearance is often that of a cicatricial relic, rather than of a tumour; and if they occupy the wall of a hollow canal (such as the intestine or the common bile-duct), they greatly narrow it. I shall hereafter have repeated occasion to insist upon the caution which is required in accepting statements as to the occurrence of fibrous non-malignant growths in such situations.

As to the extent to which the alveolar walls and stroma of a simple carcinoma are a new formation, there is still some uncertainty; and perhaps different cases differ in this respect. The analogy of keratoid carcinoma, which will be described further on, suggests that they may be in great part made up of pre-existing tissue-elements, modified in their arrangement by the pressure of the masses of epithelial cells which are in contact with them on all sides. On the other hand, it is certain that in growing cancers the alveolar walls generally contain numerous leucocytes, from which a new fibrous tissue may well be supposed to be developed. And, as Waldeyer remarks, when a carcinoma spreads in the interior of a thrombus within the channel of a vein, its alveoli can be nothing else than entirely new formations. I must confess, however, that in some cases of this kind the alveoli have appeared to me to be ill marked.

In thin sections of a carcinoma the alveoli look like closed spaces, but in reality they freely communicate with one another in different planes. Thus, when one gently squeezes the cut surface of such a growth, or when it is scraped with a knife, the cell-masses, with more or less of an albuminous fluid, escape readily, forming what has long been known as the "milky" or "cream-like" *cancer-juice*, or appearing as a soft, curdy, solid substance. The cell-masses themselves—the "*cancer-bodies*," as Waldeyer proposes to call them—may sometimes be withdrawn in continuity from two or more alveoli so as to have a branching or reticulated appearance. The cells of which they are made up present every variety of shape, "polymorphism" being indeed one of their characters. They have commonly very large, sharply defined, round or oval nuclei, and sometimes two or even more of them. The protoplasm is abundant, sometimes clear but often granular, and it has no definite cell wall.

b. Keratoid carcinoma.—This is the form of growth which is commonly known in England by the inappropriate name of "Epithelioma," originally given to it by Hannover, of Copenhagen, in 1852. Many French writers and the late Dr Hughes Bennett, following Lebert, term it "cancroid." What characterises it is the fact that some of its cells undergo a chemical change identical with that which constitutes *cornifaction* in the superficial layers of the epidermis. The horny cells are situated near the centre of the "cancer-bodies" in which they occur. They become fixed together, and the pressure to which they are subjected moulds them into globular masses, more or less laminated in structure, but often having a transparent centre, which looks like a single large cell, although, no doubt, this is not really its nature. These bodies were called by Lebert "epidermic globes." In this country they are usually, but not very suitably, designated "birds'-nest cells." It must be clearly understood that in themselves they afford no proof whatever of the presence of a malignant new growth. On the contrary, they may be found under various circumstances, as, for example (according to Thiersch), "in lupus, in the hyperæmic borders of cicatrices, in sebaceous tumours, and in the epithelial covering of the gums." Their real importance lies in the fact that when they are seen in a new growth they show it to be not merely a carcinoma, but one which has its origin either in the skin or in some mucous membrane having a laminated epithelium. This form of cancer is exceedingly frequent about the orifices of the body, especially the lips, the anus, and the vulva. The mucous membranes upon which it may occur are those of the mouth, tongue, pharynx, larynx, and œsophagus, of the lower part of the rectum, of the urethra and bladder, of the vagina and os uteri. In many of these situations, however, it is common to meet with growths which, although the presence of horny globes compel one to call them keratoid carcinomata, yet contain the globes in small numbers, and which in their general appearance and structure approximate very closely to the "simple" form of cancer. Such transitional varieties are met with in the bladder, and they are very common in the cervix uteri. In the œsophagus they have appeared to me to increase in relative frequency from the pharynx downwards, until at the cardiac orifice of the stomach a point is reached at which the liability to cornifaction in the interior of the cancer-bodies ceases altogether. Another proof of the close relation between the two forms of cancer is sometimes afforded by an examination of the structure of the secondary nodules in distant parts from a case in which the primary growth is keratoid. In some instances I have seen a nodule in the kidney or in the lung present horny

laminated globes (birds'-nest bodies) which were as well formed as those in the original seat of disease in the tongue or in the œsophagus. But in a recent case of keratoid cancer of the lip in which the cervical glands contained typical globes, I found that some large secondary masses in the liver, which were softening centrally into cavities, had more the structure of a simple carcinoma, the indications of cornifaction of the cells being very slight and imperfect. The keratoid form of cancer is, however, much more apt to infect the lymph-glands which correspond with its primary seat than to be carried to distant parts by the blood-stream.

In the more characteristic cases of keratoid carcinoma, the material which can be scraped or squeezed from the cut surface of the growth is firm and granular looking, so that it is very like the secretion of a sebaceous gland. It often comes out in the form of long worm-like masses.

Histologically keratoid carcinoma differs from common cancer in some other respects as well as in containing the "globes" or "birds' nests." Not infrequently some of the cells have prickle-edges, exactly as in certain layers of the normal epidermis. Another peculiarity is that the "cancer-bodies" are much more plainly seen to form continuous bud-like branching processes which grow more or less vertically downwards from the surface, penetrate the deeper structures and push them aside. Indeed, in its typical forms keratoid carcinoma may be said to present no definite alveoli. The substance that intervenes between the different cancer-bodies was all of it pre-existent, and it has undergone little or no change except that it is commonly infiltrated with leucocytes.

It seems now to have been clearly proved that the so-called "*Rodent ulcer*," which occurs chiefly on the face of persons advanced in life, and which is characterised clinically by its very slow course, is only a variety of keratoid carcinoma. And in all probability the same may also be said of the affection termed "*Cylindroma*" by Billroth, in which certain peculiar hyaline bodies are found. Each of these kinds of growth concerns the surgeon rather than the physician.

c. Columnar carcinoma.—In 1852 Bidder, of Dorpat, described in 'Müller's Archiv' a case in which the cells of a soft cancer of the pylorus were identical with those of "columnar" or "cylinder epithelium." Many other instances of the same kind have since been recorded, and it has commonly been called a "cylinder" or "columnar epithelioma," a name which of course must be changed into that of columnar carcinoma, if we are hereafter to use the term epithelioma in the wide sense given to it in the present chapter. In all probability the area of distribution of this form of tumour, as a primary growth, has now been completely mapped out. It includes the whole digestive tract, from the cardia downwards to within a short distance of the anus, the biliary passages, and the gall-bladder, and (according to Cornil and Ranvier) the nasal fossæ, the upper part of the uterine cavity, and perhaps the ovaries. Of course, secondary nodules may occur in various parts. I have found them in the liver and the brain, and other observers record them in the lungs and in bones. The appearance of a primary columnar carcinoma is commonly that of an irregular shallow ulcer with a soft, raised, slightly projecting border; but I am not aware that it could be identified by its naked-eye character alone. It may yield an abundant juice, full of the columnar cells, which are often still adherent to one another by their sides. A thin section generally shows that they are very regularly arranged round the borders of long-branching channels

or alveoli. But, as Cornil and Ranvier admit, some of them may be polymorphous. And I think that I have clearly seen transitions between this and other forms of cancer, not only in examining different primary growths, but sometimes also in comparing together primary and secondary growths from the same case, the tendency being for the latter to approximate to the common type of carcinoma.

Histogenesis of cancer.—The development of Carcinoma, generally, has of late years been investigated by many histologists with infinite pains, but the conclusion at which they have arrived is, after all, a matter of inference rather than of direct observation.

That leucocytes are unable of themselves to produce epithelium is almost established by what is known of the healing of wounds and ulcers from their margins, and by the success of Reverdin's practice of transplanting cuticle. One scarcely seems to need the corroboration afforded by the fact that tumours of epithelial type arise primarily only in those structures which contain epithelial elements. This fact, indeed, rests upon an experience which is not altogether unequivocal. German writers still cite a case of Virchow's, recorded in 1850, in which the tibia is said to have been affected by a primary "cancroid," the skin being healthy, and Otto Weber is quoted as having in 1859 related a case in which there was a similar lesion of the lower jaw. One may, however, fairly hesitate as to the value of such observations made at a time when their theoretical significance could hardly have been appreciated. Far greater weight would seem to belong to the fact that Waldeyer and other recent investigators have failed to meet with a single instance of the same kind. It would, indeed, be easy to account for the occasional occurrence of exceptions to the rule. Apart from the difficulty of discovering in all cases the growth which is really primary, it might be argued that it is possible for a lesion starting from the skin to extend downwards and to spread into the deeper structures, so as to appear to have begun in them. Paget, for instance, speaks of having "seen two examples of primary epithelial cancer in lymphatic glands;" one was in a sweep, whose groin contained a large mass, notwithstanding that the penis and the scrotum appeared to be unaffected. But it may be doubted whether the microscope might not have thrown fresh light upon the matter. Thiersch records a case in which a nodule seemed for a time to be subcutaneous, but really had its origin in the sweat-glands. And I may take this opportunity of observing that some carcinomata seem definitely to arise from the cells belonging to the glands of the skin, or of a mucous membrane, rather than from those which cover the surface. Further, it is conceivable, as was suggested by Remak in 1854, that a fragment of germinal epithelium, embedded in other tissues from fœtal life, may sometimes afterwards develop into a cancerous tumour, instead of forming an innocent dermoid cyst. According to Thiersch it is no uncommon thing for cancer to arise in the wall of such a cyst after it has existed for a number of years; and Waldeyer cites an instance in which a similar change occurred in a congenital sacral tumour.

Even recently, however, some pathologists have maintained that although epithelial cells are formed only in contact with pre-existing epithelium they yet arise out of cells which are not themselves epithelial. Klebs speaks of an "epithelial infection," by which, for example, in a healing ulcer the rete mucosum of the skin at its margin is supposed to convert the cells of the adjacent granulations into epithelial cells. But, as Waldeyer remarks,

there are striking instances in which a cancer fails to impress its own characters even upon epithelium with which it happens to come into relation. Thus, whereas the secreting cells of the liver are directly concerned in the development of some of the primary carcinomata of that organ, they are pushed aside by secondary nodules, and take no part in their formation.

In 1867 Köster propounded the view that the cells of cancerous growths in the skin are formed from the endothelia of lymph-channels. This, however, has met with no support; Waldeyer says that he has repeatedly seen cancer-bodies lying in immediate contact with perfectly unaltered endothelium, and although he admits that they often penetrate into lymph-channels and occupy them, he maintains that it is far from being always the case. On the whole, if we are not to cling to the notion that the epithelial cells are solely developed by germination or fission from pre-existing epithelium, it would seem to be more easy to accept the view that they arise out of ordinary leucocytes. For, as I have already had occasion to mention, it is a fact that the tissues in immediate contact with a primary cancer are very commonly thickly infiltrated with leucocytes; and, if they do not contribute to the new growth, their presence requires to be explained in some other way. It is, I think, not unlikely that they are the results of a reactive or inflammatory process, of which a more intense degree is seen as ulceration of the growth. Waldeyer points out that the cancer-cells themselves are altogether passive when ulceration occurs; they break down and escape with the discharge. But there can be little doubt that the putrid and offensive character of the ichor which exudes from an open cancer is due to the extension inwards of a septic contagium along the epithelial columns and through the alveoli, which, as we have seen, communicate with one another.

Caseous decay.—Carcinomata are liable to certain retrograde changes beginning within their substance. Like sarcomata and almost every other kind of hard growth, they are apt to undergo "caseation." Whether this is altogether spontaneous—an indication that the cancer-bodies have reached their natural term of life—or whether it is due to interruption of blood-supply, has not, so far as I know, been ascertained. But it often affects all but the narrowest possible growing margin of a tumour, especially of a secondary nodule. And not uncommonly, where a caseating tumour is seated in the interior of a solid organ, such as the liver, its centre softens down so as to produce a cavity filled with a straw-coloured albuminous liquid.

Colloid transformation.—But the most remarkable degeneration of cancers, and one which never occurs in sarcomata, is that known as "*colloid.*" Chemically there is, I believe, an exact analogy for it in the substance of the thyroid body when enlarged. Colloid material differs from mucus in not being precipitated or rendered opaque by acetic acid, and also in containing sulphur. Cancers which undergo this change sometimes occur in the breast, but scarcely anywhere else except within the abdomen. At the pylorus one can sometimes trace very clearly the gradual conversion of a simple carcinoma into colloid. Until the nature of the colloid degeneration was understood by pathologists, there was no greater puzzle. The abdomen was sometimes found filled with enormous gelatinous masses, in which the microscope showed no definite structure, but which yet penetrated organs and destroyed them in such a way as clearly to show that it possessed malignant characters. In 1847, Virchow's idea was that it was nothing else than a persistent condition of the gelatinous exudation out of which, at that time,

he imagined all cancers to develop themselves. Long afterwards it was described as a separate and a very anomalous variety of carcinoma. But of late the doctrine that it is the result of a peculiar degenerative process has met with general acceptance. There can be no doubt, however, that much new material is laid down from the blood in a growth which is undergoing this change, for its alveoli have so enormously increased in size that they are often visible to the naked eye. Indeed, formerly, when the alveolar structure of all carcinomata was not known, the terms "alveolar cancer" and "reticulated cancer" were often employed as synonymous with colloid.

The epithelial cells become swollen out into glistening, structureless globes; and it is only towards the centre of some of the alveoli that a few unaltered cells may still remain visible. Colloid growths have a yellow tint, which serves to distinguish them from the myxomata which formerly were confounded with them. Immense masses of cancer, having undergone this degeneration, sometimes fill the abdominal cavity, lying in great part loose and unattached to the serous membrane. In such cases it may be only after careful search that one discovers any portion of the growth in an unaltered state. Secondary tumours, even when apparently recent, reproduce the colloid structure.

Heredity.—Hitherto I have spoken of tumours as if they had only a local origin. But there is another side to their ætiology, which is commonly expressed in England by the statement that they are, or may be, *constitutional.*

The proof of this lies mainly in the fact that malignant growths undergo transmission by inheritance. The experience of Sir James Paget is very striking; in his hospital days he found that the proportion of cases in which a family tendency could be traced was one in six; subsequently in private practice, he made it one in four, and still more recently it has become one in three. It is true that these results might perhaps lose some of their significance, if we were to inquire as to the proportion of healthy persons, chosen at random, who would be found to have had one or more relations affected with cancer, supposing their family history to be scrutinised with sufficient care. But some of the cases recorded by Paget and by others are far too striking to be set down as mere coincidences. Thus a lady, who died with cancer of the stomach, had seven children and about thirty grandchildren, who grew up. Some of them were still living; but a daughter had already died with cancer of the stomach, two granddaughters with cancer of the uterus, a granddaughter with cancer of the breast, a grandson with cancer in the bladder, a grandson with cancer in the rectum, a grandson with cancer in the axillary lymph-glands.

A point of considerable importance is that where the disease is transmitted by inheritance it often appears in the descendants at a much earlier age than in the first patient. Thus, a young lady of twenty-four died of "epithelial cancer" of the pharynx; her mother had been attacked by cancer when between forty and fifty years old, her grandfather between sixty and seventy, a great-aunt at about forty, her great-grandmother at eighty or older. A schoolfellow of my own died of cancer of the rectum before he was forty years of age; his father and his grandfather were believed to have also suffered from cancer, but at advanced periods of life.

In the 'St Bartholomew's Hospital Reports' for 1866, Mr Morrant Baker analysed 103 of Paget's cases, and carefully investigated the question whether the appearance of cancer in successive members of the same family

could be attributed to the mere propagation of a *local* defect, or (in other words) of a tendency of some one organ to a special morbid change. But he found that it was hardly more common for the disease to occur at the same spot in two members of the same family in turn, than for its seat to be quite different.

A scarcely less important question is whether the inheritance is limited to a particular kind of new growth in each case. In regard to this Cohnheim observes that sometimes a mother suffers from adenoma of the breast, and her daughter afterwards from cancer of that organ. And among Paget's observations there are several in which it seems clear that carcinoma occurred in one member of a family and sarcoma in another. So again, Sir William Jenner mentions the case of a man who had cancer of the tongue, and whose child, twenty-two years before, had died at the age of two or three years with disseminated malignant growths, which one may presume to have been sarcomata.

But, in truth, there are grounds for stretching to the widest possible extent our conception of the inheritance of a tendency to tumour-formation. In the course of the discussion of the Pathological Society in 1874, Mr Hutchinson remarked that persons who had common warts in large numbers generally had had relatives affected with cancer. And I know that Dr Goodhart shares with me the impression, based upon our observations in the deadhouse at Guy's Hospital, that it is very common to find all sorts of innocent tumours in the bodies of those who have died of malignant growths. Moreover, Paget has insisted on the frequency with which liability to sebaceous cysts, or to certain multiple osseous tumours, is transmitted by inheritance.

Thus, it seems clear that the hereditary propagation of cancer is, after all, only part of the widely-spread occurrence of new growths of various kinds in certain families rather than in others. And the larger half of the ætiology has obviously still to be made out.

It seems to be certain that, to use Paget's words, the growth of cancer often quickly follows "deep anxiety, deferred hope, or disappointment," in such a way as to suggest that mental conditions may play a part in its causation.

The origin of Tumours.—According to Cohnheim, there is but one way in which it is conceivable that a new growth should arise in an adult, and that is by a portion of *embryonic tissue* having become arrested in its development during fœtal life, and having remained shut off until its dormant capacities have ultimately been roused into activity. Such an hypothesis was, I believe, first propounded by Virchow to account for the remarkable fact that enchondromata arise in connection with bones, but never from permanent cartilage; he supposed that a fragment of the original cartilaginous precursor of the bone might remain unossified, and might ultimately form the starting-point of a tumour. Cohnheim maintains that cancers are particularly apt to arise at spots which (as in the case of the orifices of the great mucous channels) are the seat of somewhat complicated processes in the development of the embryo, so that involutions of the external germinal layer may be supposed likely to occur there. The ultimate formation of the new growth he attributes to a failure of "physiological resistance" on the part of the normal tissues around. A somewhat similar notion was put forth by Thiersch in explanation of the liability of aged persons to keratoid carcinoma: he attri-

buted it to the atrophied and inelastic condition of the fibrous texture of the skin at advanced periods of life, whereby he supposed the extension downwards of the epidermis to be facilitated.

There can be no doubt that Cohnheim's theory is readily applicable to certain kinds of tumour. It is probably true so far as the dermoid cysts are concerned; and it may perhaps account for the frequency with which growths arise in the uterus or in the breast of unmarried women in the later years of life, since in such persons the organs in question no doubt contain germs which fail to receive their normal physiological stimulus. As regards the breast, indeed, Dr Creighton has worked out a similar idea with extreme care and in great detail.* Having studied fully the normal process of evolution or "unfolding," which the mamma undergoes when preparing for its secretory functions, he finds that the growth of tumours in it may be regarded as a modification of that process, under what he terms "spurious stimulation," occurring at a time when the organ is in a quiescent condition.

But it is obvious, though Cohnheim does not seem to think so, that such theories afford no explanation whatever of the mystery of tumour development. How a fragment of tissue, after lying dormant for years, can produce a carcinoma or a sarcoma still remains entirely unexplained.

Moreover, there are some facts which seem to me to show conclusively that the theory of dormant embryonic masses is at least not universally true. I refer to the production of tumours by *injuries*, and to their appearing in spots which have for a long time previously been the seats of various local and accidental lesions. As regards the traumatic origin of cancers, Cohnheim, indeed, takes up a most uncompromising attitude. He cites Boll's statement that in 12 per cent. of the cancer cases operated on by Langenbeck there had been an injury before the growth was developed; but he declares that such a mode of causation is nothing less than an impossibility. He believes that his experiments on animals have enabled him to study all the congestive or inflammatory processes which are capable of resulting from injuries, and that such observations are final. But the following cases related by Paget seem to me to prove the contrary.

A boy was accidentally wounded in one eye, which was sound. Within a few days, a medullary tumour grew from the eyeball; it was removed three weeks later, but it quickly recurred, and destroyed life. Another boy fell and struck his knee; swelling followed, which was at first supposed to be inflammatory; but it increased, and proved to be a large medullary growth round the lower end of the femur. A man at his work, slipped and strained or broke his fibula; the injury led to pain and swelling, and in the course of eight weeks there was found to be a growth of large size outside and within the shaft of the bone.

Many instances can be cited in which a malignant tumour appears to arise out of some *chronic local lesion*. Thus it is well known that a keratoid cancer sometimes appears in the cicatrix of a burn on the arm or on the hand, where such a growth is otherwise rarely seen. A similar affection of the tongue often follows leucoplacia, or white syphilitic patches, at the end of several years. In the common bile-duct, a carcinomatous growth is very frequently traceable to irritation of its mucous membrane by gall-stones. And I have seen more than one instance in which a malignant growth in the

* 'Contributions to the Physiology and Pathology of the Breast,' 8vo, London, 1878.

posterior wall of the urinary bladder has seemed to have been caused by the repeated introduction of catheters.*

I am not sure, however, whether most of these facts are so absolutely incompatible with Cohnheim's theory as he himself seems to suppose. One must admit that injury or irritation of a part gives rise to the formation of a new growth only in very exceptional cases; and it is surely not impossible that a dormant fragment of embryonic tissue (if its presence there may be assumed) should find in the injury, or in the irritation, the stimulus needed to arouse it into pernicious activity.

The cases in which it is most difficult to admit the embryonic hypothesis seem to be those of cancer in the scars of burns, and (on account of their frequency) those in which cancer of the tongue follows ichthyosis, or in which cancer of the bile-ducts is set up by gall-stones.

* To the same order of facts belong the frequency of cancer of the glans in cases of phymosis, of carcinoma mammæ being preceded by eczema of the nipple, of cancer of the lips in smokers, and generally of the predilection of malignant growths for the lips, fauces, œsophagus, pylorus, sigmoid flexure and anus, the parts of the alimentary canal where mechanical friction is great either from undigested food or from fæcal scybala.—Ed.

SPECIFIC INFECTIOUS DISEASES

AFFECTING THE WHOLE BODY AND ACCOMPANIED BY FEVER

Nosology—Bases of classification—General and local diseases—Specific Fevers.

In arranging the order of diseases in a systematic treatise we cannot attempt a natural, perfect, or scientific classification.

For, in the first place, diseases are not comparable things. One is defined as an anatomical change of structure; another as a pathological process; a third as the result of the action of a mineral poison, a mechanical injury, or a parasitic animal or plant; while a fourth is only a frequently recurring series of concomitant symptoms, of which we know neither the material conditions nor the probable cause (cf. *supra*, p. 3).

We might arrange diseases by the *regions* they affect; as those of the head, the breast, the belly, and the limbs; or by the *tissues* affected, as bones, joints, mucous membranes, skin; or by the *organs*, as brain, lungs, heart, liver. We should need a separate class of diseases which affect more than one organ, another of those which appear to affect every organ at once, and still there would remain diseases "of uncertain seat."

A *pathological* arrangement would group together diseases in which inflammation was the most important morbid process; those which consist of pyrexia without local inflammation; contagious and miasmatic diseases; degenerations of tissue; new growths—and so on as far as our knowledge of morbid physiology extends, whatever the region, tissue, or organ which these processes affected.

Or we might make an *analytical* arrangement, founded on the most definite and obvious symptoms; and consider successively all diseases which are characterised by a raised temperature, by dropsy, by dyspnœa, by jaundice, by the presence of albumen in the urine.

Lastly, an *œtiological* classification would put in one group those diseases which depend upon the invasion of animal or vegetable parasites, in another those which are produced by lead, mercury, or other poisons, in a third and fourth dietetic and climatic diseases.

But even if our knowledge of every "disease" were as complete as that of alcoholic cirrhosis of the liver, syphilitic orchitis, aneurysm, scabies, or anthrax—if we had completed the task of Morgagni, and had discovered the seat and the cause of every malady to which the human race is liable—we should still find that no single principle of classification would give satisfactory results.

The object of medicine is to prevent, to predict, and to cure. These objects are sometimes best served by knowledge of morbid anatomy, sometimes by knowledge of processes, and sometimes by knowledge of causes.

Moreover, who is to decide whether in the case of so well investigated a

disease as lead palsy, it will be best treated along with colic and other effects of plumbism, or with other forms of paralysis, or with wasting affections of the muscles, or as a peripheral neuritis? For different purposes each arrangement would be the best.

Every kind of classification which rests on a firm basis of facts is useful so far as it helps us to remember certain common characters of the objects classified, and mischievous if it is allowed to obscure other common characters upon which other classifications may be constructed.

In a systematic treatise, however, it is necessary to choose some one arrangement, and our choice should be decided by reasons of practical convenience.

Our divisions need not be upon a uniform plan, but they should be as few and plain as possible. They should depend as little as possible on hypothesis as to the nature and causes of morbid processes, and as much as possible on clinical and pathological facts. And they should agree in the main with the traditional names and arrangements which do not contradict ascertained facts, and which are generally accepted by the best authors.

When a clinical group of symptoms is found associated with a definite anatomical change in a given organ, it is best treated along with other "diseases" of that organ. When "it has no anatomy," it is most conveniently grouped with its nearest clinical allies. When it affects several organs, it should be referred to that which is its primary or most important seat. On these principles we make a chapter of diseases of the brain, the cord, or the nervous system generally. We arrange epilepsy near cerebral hæmorrhage, asthma not far from cancer of the lung, and rheumatism with osteo-arthritis. Diabetes might be classed with diseases of nutrition, with those of the nervous system, of the liver, or of the blood; but it may be reasonably placed in the neighbourhood of renal calculus, because these affections, differing in every other respect, agree in being both recognised by symptomatic changes in the urine. Rheumatism may be considered among the fevers, among general diseases, or among diseases of the joints; but the latter is the place where it is most conveniently compared with gout, and also where a reader would be most likely to look for it.

The general arrangement in the following chapters is local and anatomical, following the organs of the body; while functional diseases are, as a rule, placed with those presenting similar symptoms.

One important exception, however, has been made, in accordance with general practice.

There is a group of "general diseases" which, though variously defined and more or less extended, has been recognised from the earliest times. The type of this group is the disease with which we shall begin, typhus fever, as it was called, when that name only meant a variety of the genus fever. With it were associated other continued fevers, so called to distinguish them from the intermittent fevers common in Greece and Italy.

In recent times another group of diseases has been associated with continued fevers, the eruptive fevers or exanthemata, smallpox, measles, and their allies. These were classed with diseases of the skin, not only by Willan and Bateman but also by Hebra, although they appear separately in the title of his work.*

* Measles and Scarlatina appear in Willan's 'Order Exanthemata' with Nettlerash and Roserash, Smallpox with Scabies, and Chicken-pox with Eczema. Hebra's treatise is entitled, "Acute Exantheme und Hautkrankheiten," but Measles and Scarlatina appear after the

The presence of pyrexia is, however, no sufficient criterion in itself. The state of fever may be produced by local inflammation or it may be idiopathic, it may be continued or intermittent, accompanied by a rash or not. Since the discovery of the important part played by microphytes in disease, as described above in the chapter on Contagion, it appears probable that idiopathic fevers and irritative pyrexia—perhaps we may add, inflammation itself, or at least purulent inflammation*—are alike due to the reaction of the organism when a brood of microphytes invade the blood and tissues.

Leaving, therefore, the old term fever to denote, not a disease or group of diseases, but a physiological state of which pyrexia is a synonym, we may re-form the combined fevers and exanthems on the basis of their being contagious, and due exclusively to the entrance of living contagia (cf. *supra*, p. 22). In a few cases, as noted in the chapter on Contagion, one can identify the actual contagium; and, without venturing to push the argument from analogy, we may affirm that whether the contagium vivum be a schizomycete or not, typhus and enteric fever, measles and scarlet fever, smallpox, chicken-pox, and ague, are each caused by the invasion of a specific particulate contagium.

But not only do they agree in ætiology, they are all "general diseases." Some, like typhus, are without any local lesion but what is demonstrably the result and not the cause of the fever; while in others, like enteric and scarlet fevers, though local lesions are constant, they are clearly parts of a general process not its precedent cause.

Again, these specific, contagious, and general diseases agree in running a more or less defined course, limited by time and developing periods of incubation, invasion, ingravescence, culmination, and defervescence.

Lastly, each of these maladies confers remarkable protection from a fresh attack while they do not protect from one another.

We have here therefore the most, perhaps the only, natural and scientific group of diseases, each separate and definable, and yet agreeing with one another and differing from the rest.

Have any other affections than the traditional Fevers and Exanthems a right of admission to this class?

Accepting as the best criterion that of conclusive origin by contagion and of "breeding true," we have no difficulty in admitting the typically specific disease Mumps to the list, and with it will come with almost equal claims Whooping-cough and epidemic Influenza. Diphtheria may probably be added, but there are difficulties in its case which will be considered at length hereafter.

Syphilis never arises but by direct contagion; the contagium is particulate; it breeds true; it affects not only the blood, but, as Sir William Gull well put it, body, blood, and bones; it protects against itself; its invasion is accompanied by slight, occasionally by high fever, and by a characteristic rash. It differs from other specific fevers chiefly in the length of its course and in the difficulty of separating this course from what may be called its sequelæ. It also differs in the important act of being hereditary.

Tuberculosis is like syphilis in being hereditary, and in its variable and long-drawn course. It is extremely doubtful whether it can be called in any intelligible sense contagious, it does not affect every organ, and it certainly

introductory chapters, between Seborrhœa and Erythema. Why dermatologists did not include Typhus is not apparent. It is as much an exanthem as Measles, and Measles as continued a fever as it.

* The late Prof. Hüter, of Greifswald, proposed to define inflammation as a septic process due to the entrance of microzymes which are so widely diffused that the disease is pandemic.

does not protect against itself. On the other hand, a definite bacillus probably occurs in all tubercular lesions, and in well-marked cases of acute tuberculosis the rapid course and general symptoms much resemble those of a contagious fever.

Pneumonia, *i. e.* acute lobar pneumonia, has strong claims on clinical grounds to rank as a specific febrile disorder. It has its micrococcus. It invades, runs its course and subsides like a fever. Its symptoms are scarcely more dependent on the hepatised lung, as to time and severity, than are those of enterica on the typhoid ulcers. But in spite of occasional cases of asserted contagion, pneumonia is on the whole less evidently due to previous cases than tubercle. It is also believed generally to arise from common causes, as exposure to cold. Its course though less variable than that of syphilis or tubercle, is far from constant; and it is not self-protective.

Cholera is another doubtful case. Few would now regard it as a mere local enteritis; such a conclusion would have less foundation than Broussais's doctrine that typhoid fever was only symptomatic pyrexia from ulcerations in the bowels. Few doubt that it is contagious, as it certainly is epidemic; and this conclusion is independent of the validity of the organism regarded by Koch as the specific microphyte of cholera. No diseases are more certainly contagious than typhus and smallpox, yet in neither has the microphyte, if it exists, been discovered. Usually there is little or no pyrexia in cholera, but there is reason to believe that this feature is rather masked and partially suppressed than absent.

Two epidemic febrile disorders, contagious and once terribly destructive, have happily been banished from this country—the Sweating Sickness since the sixteenth, and the Oriental Plague since the seventeenth century. The latter without question, and probably the former * is, like cholera, endemic and comparatively mild in certain localities; but, when epidemic, they acquire fearfully active properties.

Three specific febrile diseases of the brute creation are occasionally transferred to man by inoculation,—they are, Anthrax or splenic fever, Glanders and Hydrophobia. In the first the microphyte is known and its whole history investigated; it answers every test of being the true contagion of the disease (*v. supra*, p. 22, note), so that other maladies may be compared with this as a criterion. Glanders is almost as certainly and completely known. Hydrophobia is in many ways obscure and is still usually classed with diseases of the nervous system. It must be remembered that the whole doctrine of specific diseases and microphytes as contagia is strongly supported by observation and experiment of contagious diseases in the lower animals.

Another most remarkable malady, as fatal as hydrophobia, is that known by the cumbrous name of "acute yellow atrophy of the liver." This appears to me to have many of the characters of a specific disease, although, like cholera, it is not a fever.

While we must admit, as undoubted members of this class, some affections in which no microphyte has been found, we must, I think, exclude others, though more or less certainly bacterial in origin, because they are not "general," or run no definite course, or do not protect. Gonorrhœa and Leprosy are instances, also perhaps Actinomycosis.

Several of the above diseases are not included in the following section,

* See Dr Payne's article in the new edition of the 'Encyclopædia Britannica,' "Sweating Sickness."

either because their pathology is doubtful or because they are more conveniently treated with local diseases.

Rheumatism and Gout, though general diseases, and febrile, are not contagious, do not protect, and may be ascribed to meteorological or dietetic causes.

In the following list I have stated in a brief and, of necessity, dogmatic form, the characters of the several diseases which entitle them more or less decisively to a place in this section.

Specific Infectious Diseases

Disease.	Contagious.	Microphyte.	Local lesions	Exanthem.	Extent.	Course.	Protection.
1. Typhus	Exclusively	None known	None	Present	Epidemic	Definite	Complete*
2. Recurrens	,,	Spirillum	,,	Absent	,,	,,	Imperfect.
3. Enterica	Probably exclusively	Uncertain	Ileum, spleen, &c.	Present	Endemic	Less definite	Complete. Relapses.
4. Scarlatina	Exclusively	,,	Throat,&c.	,,	Epidemic	Definite	Marked.
5. Rubeola	,,	None known	,,	,,	,,	,,	,,
6. Morbilli	,,	,,	Bronchi, &c.	,,	,,	,,	,,
7. Variola	,,	Micrococcus ?	None	,,	,,	,,	Complete.
8. Vaccinia	,,	,,	,,	,,	Endemic	,,	Marked.
9. Varicella	,,	None known	,,	,,	Epidemic	,,	,,
10. Mumps†	,,	,,	Parotid	Absent	,,	,,	,,
11. Pertussis‡	,,	,,	None	,,	,,	Indefinite	Complete.
12. Influenza‡	Probably exclusively	,,	Bronchi, &c.	,,	,,	Definite	Limited.
13. Erysipelas	,,	Micrococcus	None	Present	,,	,,	None.
14. Diphtheria	,,	,, ?	Throat,&c.	Absent	,,	Less definite	Marked.
15. Cholera	Exclusively	Vibrio ?	Intestine	Occasional	End. and epid.	Definite	,,
16. Plague	,,	None known	Lymph-glands	Petechial	Epidemic	,,	Incomplete ?
17. Glanders	Exclusively	Bacillus	,,	Absent	Epizootic	,,	?
18. Anthrax	,,	,,	Spleen	,,	,,	,,	Complete.
19. Ague	Miasmatic	,, ?	,,	,,	Endemic	Indefinite	None.
20. Syphilis	Exclusively	Uncertain	Throat,&c.	Present	End. and epid.	Prolonged	Complete.
21. Tuberculosis§	?	Bacillus	Various	Absent	Pandemic ?	Indefinite	None.
22. Pneumonia‡	?	Micrococcus ?	Lung	,,	Sporadic or epidemic	Definite	?

* *I. e.* cases of the same person having two attacks, though they certainly exist, are so extremely rare that they do not affect the practical rules as to infection.

† Described under "Affections of the Mouth and Throat," vol. ii.

‡ Described under "Affections of the Lungs." vol. i.

§ Described above in an Introductory Chapter.

TYPHUS*

Historical sketch—Incubation—Course: first week: second week: crisis; recovery: death and post-mortem appearances—Complications and sequelæ—Ætiology; contagion of typhus: its supposed spontaneous origin—Prognosis—Treatment.

SCARCELY more than thirty years have elapsed since this disease was finally separated from certain other diseases with which it had been confounded. And yet we can trace back its history. For its prevalence as an epidemic, and the fearful mortality which always accompanies it, enable us to feel confident that many descriptions handed down by writers of former times were based upon a study of Typhus, unmixed with any of those forms of fever from which it is now known to be distinct.

The name is of no great antiquity, for it was first applied to a malady, or to a group of maladies, by Sauvages in 1759. Until then it had, from the time of Hippocrates downwards, been employed, in accordance with its etymology, to designate a confused state of intellect, with a tendency to stupor. Murchison and others think that the plague of Athens, recorded by Thucydides, was really what we now call typhus. However this may be, there is no doubt about the nature of certain epidemic fevers which prevailed in Italy, France, and Hungary in the sixteenth century, and of which accounts were given by Fracastorius of Verona, and by many other writers. During the course of the same century occurred the first three of the famous "Black Assizes" in this country, when judges, sheriffs, and jurymen were suddenly attacked with fatal illness, as the result of infection from the prisoners brought before them for trial. One of the older synonyms for the disease, indeed, is *jail-fever*. Another is *Morbus castrensis* or *military fever*, from the ravages which it has committed among soldiers and camp-followers, from the time of the Thirty years' War and the English Civil War down to the siege of Sebastopol. Other names, again, are *spotted fever*, *brain fever*, *putrid fever*, from some of its more conspicuous symptoms. Of late years it has been universally called Typhus in this country and in France; but the Germans are obliged to term it *Typhus exanthematicus* or *Flecktyphus*, because they commonly speak of Enteric Fever as *typhus*, dropping the epithet *abdominalis*, which is properly required to complete the designation of Enterica.

On the Continent the disease with which we are now concerned has generally been much less common than in the British Isles. Both in Great Britain and in Ireland, it has prevailed with great severity on repeated occasions during the last two hundred years. Since the commencement of the present century there have been epidemics of Typhus in 1803, in 1817–19, in 1826–28, in 1836, in 1843, in 1846–48, in 1856, and from 1861 to 1870. It must be noted, however, that in some of the earlier of these epidemics

* *Synonyms.*—Contagious or epidemic fever, spotted fever, febris petechialis, typhus exanthematicus, brain fever, jail or camp fever, fourteen-day fever.

there was a large admixture of cases of another form of disease, Relapsing Fever, which was not known to be specifically distinct from typhus until 1843, but the previous occurrence of which can even now be easily recognised by the small mortality which has always attended it.

Incubation.—This is of variable length. Few cases afford an opportunity of determining it, for the disease is rarely the result of a single definite exposure to contagion. But Murchison collected for the 'St Thomas's Hospital Reports' in 1871 no fewer than thirty-one instances more or less directly in point. In two of them the effect was immediate, and the same thing had been noticed by other observers: the patient having been conscious of an offensive odour proceeding from a case with which he had come into contact, was at once attacked with headache, prostration, nausea, rigors; and all the other symptoms developed themselves in due course. In one instance the period was not more than two days; in two, not more than four days; in one, from two to five and a half days; in one (that of Murchison himself) exactly five days; in one not more than six days; in two not more than ten days. Short incubation periods have been given by other writers; thus Lebert, in 'Ziemssen's Handbuch,' states five to seven days; and some cases have been recorded in which the disease followed exactly eight days after exposure. Murchison, on the other hand, found that in the larger number of his thirty-one cases the period was longer: in four it was exactly twelve days; in thirteen others it was within a few days of this, on one side or the other; and in four instances it was ascertained to be longer than twelve days; being in one patient not less than thirteen days, in another not less than fourteen days, in the third exactly fifteen days, in the fourth not less than twenty-one days.

During the incubation the health generally appears to be perfect; sometimes towards the end of it, there is a little malaise, with headache, pains in the limbs, and loss of appetite. Lebert says that these symptoms may be protracted over several days.

Course.—The course of typhus is best studied by a division into weekly periods. In the following account of it I shall in the main follow Murchison's description, but with regard to the facts all writers are agreed.

First week.—The onset of the disease, from which the first week is reckoned, is generally rather definite, or even sudden: more so than in enteric though less than in relapsing fever. The patient is attacked with headache and with pains in the back and limbs, as though these parts had been bruised. He feels chilly, or he may even shiver; after a little while he may perspire; but the chilliness presently returns and he is glad to sit huddled before the fire. He feels weary and disinclined for exertion. He is thirsty, but he has no appetite, and complains that his taste is perverted. His tongue is large, pale, and coated with fur, which is at first white, afterwards yellowish. There is nausea, but not often vomiting. The bowels are constipated. The urine is scanty, dense, and high coloured. He is restless and his sleep is disturbed by painful dreams and sudden starts. Every day he becomes more prostrate; he totters in walking, his hands tremble when he attempts to use them; by the third or the fourth day at latest he gives in and takes to his bed.

From the first the expression is dull, heavy, and oppressed. The eyes are injected and suffused. The face is of a dusky colour, the flush being general, and not limited to the cheeks as in enteric fever. As the disease

advances, the countenance becomes more and more vacant and stupid-looking. It very seldom happens that the patient himself feels much concern about the issue of his illness. Towards the end of the week there is generally some *delirium*, especially at night, and when he is half asleep. The advent of this symptom is generally earlier in persons belonging to the upper classes, in those who have been intemperate, and in those who are the subjects of mental anxiety and fatigue, than under opposite conditions. In some exceptional cases it sets in at the very commencement of the disease during the first night, and Murchison speaks of having seen cases which were at first mistaken for mania. But, as a rule, it is only after the lapse of several days that the patient's mind begins to wander, and that his intelligence and memory become confused and impaired. About the same time he generally ceases to complain of headache.

The *pulse* does not usually rise above 100 during the first two or three days; afterwards it generally ranges between 100 and 120. When it is much higher, the case is severe, if the patient is an adult; but in children a very rapid pulse even at the commencement of the disease is not an evil sign. Occasionally, however, the pulse remains below 100, or even below 90. Murchison cites instances observed by himself or by others, in which it was not above 40, or actually down to 28, for days together. Sometimes the heart's beats have been twice as frequent as the pulsations felt at the wrist. A very slow pulse is not generally a favourable sign.

The *temperature* rises rapidly in typhus. Wunderlich stated that it is generally 104°—104·9° on the first evening, and that by the fourth evening it is seldom under 104·9°, generally about 105·8°, and often higher still. But although Lebert has since observed 106·5° on the second evening, the tendency of all English clinical work with the thermometer has been to show that the average figures are very much lower than those given by Wunderlich. According to Murchison the highest temperature attained at any period of the disease is generally about 104° or 105°; it is scarcely ever as high as 106°, except in children, and it may be below 103°. The maximum is usually observed between the fourth and the seventh days, sometimes on the third day, sometimes not until the second week. During the latter part of the first week, when the maximum has once been reached, the thermometer varies little. Murchison remarks that a high range of temperature in the first week generally forebodes severe cerebral symptoms in the second. Lebert makes it a point that a rise in the evening is of comparatively little significance, when the thermometer falls each morning; what is really serious is a high temperature continuing without any intermission.

Typhus is attended with a characteristic *eruption*, the mulberry rash, as it was named by Sir William Jenner. But in rare cases this is preceded by a *roseola*, which is very different from it in appearance and may almost be mistaken for the eruption of scarlet fever. In the museum of Guy's Hospital we have models, illustrating this remarkable rose-rash, which were taken from two women under the care of Dr Wilks in 1864. The parts represented are the abdomen, and the forearm with the hand; but in each instance the rash is said to have covered the patient. It was of a bright crimson colour, punctated, macular, or diffused. One of the women was a nurse in the hospital, so that she was under observation from the first. The roseola in each case faded before the mulberry rash came out.

Dr John Harley once saw a copious eruption of *rose papules* upon the chest and abdomen of a man who had severe typhus; they preceded the

mulberry rash, and disappeared when it became petechial ('Reynolds' System of Medicine,' vol. i, 584).

The *mulberry rash* is generally first seen on the fourth or the fifth day of the fever; sometimes, as in a case admitted into Guy's Hospital in 1874, it may be discovered as early as the third or even the second day; Murchison says that it scarcely ever begins to appear after the sixth day. Sometimes it is altogether wanting; but observations made with great care at the London Fever Hospital in 1864 showed that this occurred in scarcely more than 2½ per cent.—among nearly 2500 cases of all ages. In children, however, absence of the eruption is much more frequent. But, on the other hand, when it does come out in them, it is often peculiarly abundant and well marked, so as to resemble the rash of measles; and in such cases it may be seen on the face, whereas in adults it is generally confined to the trunk and limbs. One usually looks for the typhus rash on the chest and the abdomen; but Dr Buchanan, in 'Reynolds' System of Medicine,' says that the earliest traces of it are to be found on the back of the wrists, the borders of the axillæ, and the epigastrium. It consists of more or less numerous spots, of indeterminate form, the largest three or four lines in diameter, isolated or irregularly confluent. They may at first be slightly raised, so that one can feel them with the finger; and as they may then be of a somewhat florid colour, it is sometimes difficult, when they are few and separate, to distinguish them from the rose spots of Enterica. At this time, too, they disappear on pressure. But in the course of a day or two they alter in appearance; their hue becomes darker and more dingy; they are no longer raised; and when the finger is pressed upon them they either remain unaltered or display a yellowish tint. This change is due to the escape of blood from the vessels into the substance of the cutis; in other words, the maculæ have become petechial, according to the modern definition of the word *petechia*, as a "minute ecchymosis," which we owe to Willan and his successors. They remain visible in the dead body if the case should end fatally while they are still present. In many instances they are from the very first of a livid or purple colour, and do not fade on pressure.

Within forty-eight hours from its first appearance the mulberry rash is complete. During this time fresh spots may come out, but they are added to the old ones. There is not, as in enteric fever, a succession of crops of maculæ, one set appearing while another is fading away. With regard to this point, Murchison says that he satisfied himself by observations upon a large number of cases, surrounding every spot with a circle of ink so as to identify it. In addition to the distinct maculæ, there is also a faint, irregular, dusky-red mottling, which one would imagine to be more deeply-seated, and which, therefore, has been called a "subcuticular rash"—an unfortunate expression. After the first day or two, no increase of the eruption occurs.

It is important to notice that the severity of a case of typhus is generally directly proportionate to the amount of eruption, its depth of colour, and the rapidity with which it becomes livid or petechial. Murchison adds that exceptional cases in which no rash appears have a generally mild course; but according to Lebert they are often severe and even fatal.

Second week.—This period is marked by a gradual aggravation of all the more obvious symptoms. The patient is now absolutely sleepless, and he usually passes into a state of continuous *delirium*. Sometimes he is noisy and violent, shouting, talking incessantly, singing, struggling to get out of bed, or fighting with his attendants. When spoken to, he generally

becomes still more excited. The patient may throw himself out of the window, or cut his throat. Some years ago a man was brought into hospital, and was actually admitted into a surgical ward, for a suicidal wound of the neck inflicted in the course of typhus. After two or three days the delirium becomes quiet and muttering; and in the majority of cases it is so from the first. Sometimes excitement comes on as night approaches, while the rest of the day is passed in a state of stupor and prostration. But by the middle of the week there is generally complete unconsciousness. When loudly spoken to, the patient perhaps opens his eyes and stares vacantly; if told to put out his tongue, he may separate his jaws, and leave them gaping until they are closed for him. No other signs of intelligence can be elicited; yet Dr Murchison, who himself went through two attacks of typhus, tells us that the imagination is far from inactive. He took a great dislike to a nurse and to a valued friend; and because they once tied him down in bed, he kept fancying that they intended to murder him, that they were shutting him up in a dungeon, that they followed him to India, Burmah, and other countries which he had really visited in former years, and to which he now supposed himself to have escaped. Dr Gueneau de Mussey, who caught typhus in Dublin in 1843, afterwards remembered imagining that he was tied down in bed, and gradually consumed by spontaneous combustion, while women were taking water from a pond and pouring it over him. Another fancy was that he saw the front of a particular house in Paris in a state of phosphorescence, and a child suspended by the neck from a window. He also believed that he saw one of his friends killed in the street; and so strong was the impression that during convalescence he still maintained that this friend was dead, and felt great concern for his loss.

During the second week of typhus *deafness* is very commonly present; indeed, it begins about the fifth day. It sometimes affects one ear, sometimes both. Its cause is not yet ascertained. Stokes thought that it was due to softening of the muscles of the ossicula; but to this view Murchison objects that the hearing is too soon recovered during the convalescence. For, contrary to what might perhaps have been anticipated, many cases do well in which deafness has been complete; and from the time of Fracastorius there has been a tradition that it is a favourable sign. For this belief, however, there do not appear to be any good grounds; it is certainly present in many cases which end fatally.

The *pupils*, in the advanced stages of typhus, are generally contracted and insensible to light. They may be as minute as pinholes, according to a comparison made by Graves. Jenner first laid stress on the state of the iris as affording a distinction between typhus and enteric fever; and Murchison says that, neither during active delirium nor in profound stupor, has he seen dilated insensible pupils in typhus. Occasionally, however, the supervention of coma is associated with dilatation of the pupils.

The muscular *prostration*, during the second week of typhus, becomes extreme. The patient sinks down in bed, and lies on his back, unable to raise himself, or even to turn on either side. Students at Guy's Hospital twenty years ago all knew one of the favourite remarks of the late experienced apothecary, Mr Stocker, that to find a fever-patient lying on his side was a good sign. The fæces are passed involuntarily. The urine dribbles away incessantly, so that unless it can be caught in a proper receptacle it soaks the sheets and produces great irritation of the skin.

But before it begins to run off it may fully distend the bladder, the muscular coat of this organ being paralysed, as well as the sphincter. One must, therefore, never omit to examine the hypogastric region at every visit, even if the nurse should say that the urine is passed in abundance. Other results of muscular weakness are loss of power to speak, to protrude the tongue, and sometimes even to close the eyelids, as the result of which the cornea may slough, and allow the contents of the aqueous chamber to escape.

At this stage of the disease *tremor of the muscles* is almost constantly present. The whole body may be in a state of agitation, especially in old people and in those who have been exhausted by brain-work or by intemperance: indeed, in drunkards a state of delirium tremens often seems to be superadded to the symptoms of the fever. Sometimes the tremor is limited to the hands and tongue. Murchison speaks of having sometimes seen nystagmus or choreiform spasms of the muscles of the limbs. Much more frequent are those jerking movements of the muscles of the forearms which are commonly called *subsultus tendinum*. Or there may be twitchings of the face, one corner of the mouth being perhaps drawn up from time to time. Jenner saw two cases in which the face acquired a peculiar expression from a spasmodic action of the inferior recti muscles of the eyeballs in association with the levatores palpebrarum. In each instance the movements were excited when either of the arms was suddenly raised. Another variety of spasm is picking or fumbling at the bedclothes, for which we have the pedantic names of "floccitatio" and "carphology." In more rare cases the fingers are forcibly clenched, or the legs and thighs are bent so that the knees almost touch the chin, or there may even be trismus or opisthotonos. It has not yet been ascertained whether these last symptoms are dependent upon the presence of meningitis, which sometimes occurs as a complication of typhus.

The mulberry *rash* generally remains visible throughout the whole of this period. The maculæ may even become darker and more distinct, and may not disappear until the disease has already subsided. But the faint general mottling vanishes after a few days, and in mild cases, where this is the only eruption, nothing of it may be left at a time when the fever has still several days to run.

The *temperature* during the second week is, as a rule, rather lower than in the latter part of the first week. Sometimes, indeed, the maximum is not reached until between the seventh and the tenth day. But during this period there is generally a slight fall, to which Murchison attached importance, believing that the cases in which it was absent were severe. Henceforth, and up to the time of the crisis, there are usually slight recurring remissions of one or two degrees. A decided rise at this period indicates the supervention of some complication, generally pneumonia.

The rate of the *pulse* varies little from day to day, but its general tendency is to become more rapid. Its volume and force decline until in severe cases it is exceedingly feeble or even imperceptible. Sometimes it is dicrotic or undulatory, but Lebert remarks that this is remarkably seldom the case, whereas it is almost the rule in enteric fever. Irregularity of the pulse is not uncommon, and sometimes there are intermissions. Graves laid stress on the fact that a change from the recumbent posture to the erect or semi-erect is attended with an acceleration of the pulse, the amount of which is greater in proportion to the weakness of the patient. His successor, Professor Stokes, pointed out a still more important indication of the progressive

weakness which constitutes one of the chief dangers of the disease, namely, diminution or loss of the heart's impulse with disappearance of the first sound. For several days, even when recovery is to take place, it is often impossible to feel the heart beating, and with the stethoscope only the second sound can be heard. In other cases a systolic bruit is developed, which no doubt belongs to the class of functional murmurs.

The *breathing* at this stage of the disease is almost always hurried, being at the rate of thirty or forty in the minute. Moreover, according to Murchison, hypostatic congestion of the lungs should be regarded as a constant symptom rather than as a mere complication. It is, at any rate, present in all severe cases, and after death is scarcely ever wanting, whatever may have been the cause of the fatal issue. It commonly begins about the middle, but sometimes at the commencement of the second week. It is indicated by diminution of percussion resonance over the bases behind, where the respiratory murmur is feeble, and by the presence of coarse râles, which may gradually spread over the greater part of the chest on both sides. The affected parts of the lungs are found *post mortem* to be bulky, of a dark red or purple colour, and soft; serous fluid oozes abundantly from their cut surface, and from the tubes. Indeed, bronchial catarrh is always associated with this condition, and the patient may spit up considerable quantities of frothy tenacious secretion, mixed perhaps with streaks of blood. But sometimes cough and expectoration are entirely absent.

The *tongue*, in very mild cases, may remain moist and furred throughout the whole course of the fever. But usually during the second week it is dry, rough, and brown. When the disease is very severe it contracts into a ball, and becomes covered with a dark brown or black crust; this crust may be irregularly cracked; but the fissured condition of the tongue itself, which is so commonly seen in enteric fever, is rare in typhus. The lips and teeth are covered with a dirty blackish material, commonly spoken of as *sordes*, which consists of "an accumulation of epithelial débris, which becomes black from desiccation, or perhaps from an admixture of blood." An entire loss of appetite lasts to the end of the disease; and sometimes the patient refuses to take the nourishment which is given to him. He generally does not at this stage appear to suffer from thirst. Murchison met with some instances in which vomiting was a persistent and troublesome symptom, but such an occurrence is exceptional, and such patients have been for the most part previously dyspeptic. Neither he nor Sir William Jenner before him found the abdomen unnaturally large in the majority of cases: sometimes it was flat or even concave; distension amounting to meteorism was very rare. As a rule there is *constipation*, but the opposite state of the bowels is not infrequent. The fæces may be of normal consistence, and even if fluid, they are generally dark in colour. Murchison speaks of diarrhœa as occurring in about 5 or 10 per cent. of all cases of typhus. But in 1866 Dr Buchanan said that it had been seen in at least one third of the cases which had come under observation at the London Fever Hospital during some years previously; he was inclined to refer its greater frequency in the experience of some physicians to the larger amounts of liquid food forced upon the stomachs of their patients, although he did not consider this more than a slight drawback to the practice in question. Murchison remarks that when an aperient has been required the bowels often afterwards remain open or even relaxed.

The *urine* generally remains dark in colour during the second week of typhus, but sometimes it is pale and even alkaline. With regard to the

amount of urea which is voided, I have nothing to add to what has already been stated in the chapter on Fever (p. 47). It is an interesting fact that at this period of the disease the *chlorides* fail to be excreted by the kidneys, even when the patient is made to take large quantities of common salt. The urine sometimes contains no chlorides at all, but Dr Buchanan says that usually two or three grains are passed in the twenty-four hours, just enough to produce an opalescence, on the addition of nitrate of silver. The not infrequent presence of *albumen* in the urine will be referred to presently.

Crisis.—Such is the condition of a patient suffering under typhus until about the fourteenth day, when, if he lives so long, a marvellous change takes place—the crisis. Formerly, what was most often noticed was that he fell into a sound and quiet sle p, from which he awoke a few hours later, rational, refreshed, and cool. But at the present time the use of the thermometer enables us to watch the process much more closely. In some exceptional cases there is a rise of perhaps two degrees the day before the crisis occurs. Much more often the temperature becomes slightly lower during the same period. The crisis itself is marked by a rapid fall to normal or even lower; a difference generally of from 4° to 6° Fahr. The fall may be complete within twelve hours, but according to Lebert it much more often takes thirty-six hours and sometimes two or three days. Murchison says that in nearly one half of the cases of typhus the date of the crisis is the thirteenth or the fourteenth day; in more than three fourths of them it is from the thirteenth to the sixteenth. Lebert gives a much larger proportion of cases as subsiding between the tenth and the twelfth days, some even between the sixth and the ninth days.

Complications may occasionally mask the crisis, or prevent its occurrence. They will be considered presently (p. 140).

Crisis is sometimes accompanied by perspiration, or by diarrhœa, or by the excretion of urine depositing lithates in abundance; but there is no reason to suppose that such phenomena are essential to the defervescence, as was formerly supposed. Afterwards the temperature generally remains normal, except that for a night or two there may be a slight evening rise.

Convalescence advances rapidly. The tongue at once becomes clean and moist, and the appetite is ravenous. Within three or four weeks the patient often completely recovers his bodily strength and is able to return to work.

A true *relapse* of typhus is exceedingly rare. An instance of it was recorded in 1869 by Ebstein, the interval being twenty-five days. At the London Fever Hospital no such case occurred during twenty-three years after 1855. In that year Dr Buchanan had a nurse under his care who after recovering from an attack of the disease, was taken ill a week later, and went through it a second time: on each occasion there was a mulberry rash. The first fever appears to have lasted a fortnight, the second somewhat longer.

Exitus lethalis—Many cases of typhus do not run on long enough to exhibit a crisis. The mortality from the disease at the London Fever Hospital has been at the lowest 15 per cent. Murchison estimates that in the metropolis generally, if slight cases and these occurring in children be taken into account, it is about 10 per cent. It differs, however, slightly in different years, and it is said to be generally higher during an epidemic than at other times. Among soldiers in camp, or in a besieged city, the death-rate has sometimes been enormous. In the Crimea one half of the French troops who were attacked are said to have perished; and still more terrible instances are on record. In certain epidemics death has sometimes occurred on the

second or the third day, or even after a few hours' illness. Such cases have been described as examples of a special variety of the disease—*typhus siderans.** Murchison speaks of having seen several cases end fatally on the sixth or eighth day, usually from pulmonary congestion. But, as a rule, typhus does not destroy the patient until towards the end of the second week. The exact mode of death varies. Sometimes it is by "typhoid" symptoms and coma; sometimes it is through the lungs by asphyxia; sometimes it is by failure of the heart, the pulse being imperceptible, and the skin cold, livid, and bathed in a profuse sweat. In some of the last-mentioned cases the patient lies for a day or longer in a peculiar state, to which Jenner has appropriated the name, *coma vigil*. He has his eyes wide open, so that he might be supposed to be awake, but he is absolutely insensible to all that goes on about him, and his face is devoid of expression. Sometimes death is preceded by a rapid elevation of temperature, as in a case at Guy's Hospital in 1873, in which the thermometer registered 108·7°.

Morbid anatomy. — Most of the appearances observed in *post-mortem* examination of the bodies of those who have died from typhus, instead of being characteristic of the disease, are rather effects of the febrile state through which the patient has passed, such as are found equally in cases of erysipelas, pyæmia, and other acute diseases. The *rigor mortis* is brief and incomplete, putrefaction takes place early, there is much cadaveric discolouration of the skin, the *blood* is fluid and dark coloured, and it stains the lining membrane of the heart and of the great vessels. There is often but little emaciation, yet the *muscles* are soft and friable, and when death has occurred at a late period of the fever their fibres are granular or waxy-looking under the microscope. A similar change is found in the substance of the *heart*, and probably often plays an important part in bringing about the fatal issue. The *spleen* is but little enlarged, weighing on an average seven ounces, though sometimes as much as fourteen ounces; but its tissue is very soft, and may be reduced to a mere pulp, which escapes when the capsule is divided. Jacquot is said to have observed a case in which instant death resulted from rupture of that organ. The *liver* is soft and hyperæmic: in an advanced stage of the disease it is often fatty. The state of the *lungs* has already been noticed. There is, lastly, an entire absence of those intestinal lesions which belong to enteric fever.

Other changes which have been noted are probably altogether independent of the disease. They are common either at a certain period of life, or in dissipated and intemperate persons, such as are apt to succumb to typhus. Thus the brain has often been found atrophied, with an excess of fluid in its ventricles and upon its surface.

In other instances the *kidneys* have been wasted or in an advanced stage of Bright's disease. In such cases death is apt to be ushered in by convulsions, the occurrence of which in typhus should always lead at once to an examination of the urine; it may be then found to be albuminous, and may contain blood, more or less altered, and casts. Sometimes the renal affection appears to be quite early and recent, and probably is itself secondary to the fever. Murchison speaks of having found the kidneys weigh nineteen, twenty, or even twenty-three and a half ounces. It is to be remembered, however, that albumen may be detected in the urine of a large proportion of cases of typhus, in which no cerebral symptoms occur, and which do well.

* Or more properly the patient is described as *sideratus*, planetstruck.

It is usually in small quantities, and it is often present during only a day or two days towards the end of the disease. Even when convulsions set in, an absolutely unfavourable prognosis is by no means to be given. Recovery took place in twelve among one hundred and thirty-two cases in which this complication occurred at the London Fever Hospital between 1862 and 1869. In very rare instances, where a fatal issue has occurred, blood has been found effused upon the surface of the brain; but this may have been the result of the convulsions rather than their cause. It is towards the end of the second week that convulsions are most often observed.

Complications and *sequelæ* of typhus are not numerous. Jaundice is extremely infrequent. Murchison met with only fifteen cases in which it was present. In one of them he considered that the liver was in a state of acute yellow atrophy; and in another leucine and tyrosine were found in the viscera as well as in the urine. One well-marked instance of jaundice occurred at Guy's Hospital in 1869. The patient, a man aged forty-nine, died on the fourteenth day of the fever.

Pneumonia is not very uncommon. Sometimes, when the patient appears to die of the severity of the disease, instead of there being merely hypostatic congestion of the dependent parts of the lungs, a considerable part of one of them is found to be hepatised. Either the lower or the upper lobe may be affected in this way. In other cases inflammation of the lung sets in definitely a few days after the crisis. Of about forty consecutive fatal cases examined at Guy's Hospital, no fewer than six had well-marked pneumonia. In one instance an attack of pleurisy occurred a week after the subsidence of the fever; and after death the left side of the chest was found to contain several pints of fluid with flakes of lymph. Murchison has several times seen gangrene of the lung after typhus.

In some rare cases acute Meningitis occurs as a complication. In addition to the cerebral symptoms which commonly accompany the fever, there is then retraction of the head, strabismus, and dilatation of the pupils. Mania sometimes sets in during convalescence, and may compel the removal of the patient to an asylum; but in the long run it seems always to end favourably. Hemiplegia, with or without aphasia, is an occasional sequela, but in all likelihood it depends upon embolism of one of the cerebral arteries by a fragment of thrombus derived from the left auricle or ventricle of the heart; for clotting of blood in the recesses of these cavities is doubtless of frequent occurrence during the height of the disease. Murchison, however, once saw endocarditis in a case of typhus, there being large vegetations, which gave rise to infarction of the spleen. Embolism of the arteries of the limbs is doubtless the cause of gangrene of the feet, which has now and then been known to occur after typhus. Bedsores ought hardly to arise in this disease, since the stage of coma is of comparatively short duration. Their presence sometimes accounts for the supervention of pyæmia, but in other cases blood-poisoning sets in during convalescence without any such obvious explanation. Thrombosis of the femoral veins is not often seen after typhus. Inflammatory swellings which have been called *buboes*, and compared with those that occur in the plague, occasionally form in the parotid and submaxillary regions, and go on rapidly to suppuration.

When a pregnant woman is attacked with typhus she not infrequently passes through the disease without interruption to the process of gestation, but sometimes a miscarriage occurs between the tenth and the fourteenth

days. As a rule she afterwards does well, and the child, if not too immature, is generally saved.

Ætiology.—The most conspicuous fact in the ætiology of typhus is its contagiousness. That it passes directly to other persons from the sick is established by the clearest possible evidence. Instances of such an occurrence are commonly observed in every hospital into which cases of typhus are admitted; if into general wards, those who are already patients with all kinds of other complaints are attacked; if into special wards, the medical attendants and nurses fall frequent victims. When cases are left in private houses or lodgings, the disease passes not only to relatives and to other inhabitants of the same dwelling, but also to doctors and clergymen whose visits are only occasional. On the other hand, the removal of a single case of typhus from the building in which it arose is often effectual in preventing other occupants from taking it.

Lastly, the diffusion of the disease can often be traced from point to point in a town or in a district. Thus Alison relates how the son of a shoemaker in Edinburgh lay ill with typhus in a room in which his father and two apprentices were at work. Afterwards both of the apprentices were attacked in their own homes, widely apart from one another, and at considerable distances from the workshop; and there speedily followed a succession of cases in the other inhabitants of these two houses, seven persons being affected in one, and twelve in the other.

An epidemic which occurred at Carlisle in 1781 was found by Dr Heysham to have started from a particular house in Richard Gate; one of the persons affected there was a weaver, who on his recovery communicated the disease to his fellow-weavers in a large workshop, and by them it was spread all over the town. In 1847 immigrants introduced typhus into America, and in 1867 into Australia.

With regard to the nature of the contagious principle of typhus, we are as yet altogether ignorant. There can be no doubt that it multiplies in the blood, but hitherto no observer, even with the highest microscopic powers, has succeeded in detecting any special organisms there. It is probably exhaled both by the skin and the lungs, and it may perhaps be identical with the cause of the offensive odour which is so perceptible in the close proximity of almost all severe cases. This odour has been compared with the smell of rotten straw, or with that of mice. Murchison regarded it as *sui generis;* he says that he has known nurses in the London Fever Hospital distinguish typhus from other fevers by it alone. It is given off chiefly during the second week, and there is some reason to believe that infection is scarcely to be feared during the first week. The late Dr Perry, of Glasgow, maintained that the disease was not contagious before the ninth day, or perhaps not until a later period still. He found that at the Glasgow Fever Hospital patients who happened to have been sent in for erysipelas, pneumonia, bronchitis, or other inflammatory affections, escaped typhus so long as they remained in the wards for acute cases, but caught it when they were transferred into a convalescent ward; and he adopted the plan of keeping such patients in the fever wards until they were sufficiently well to go to their homes, with the result that during several months none of them were attacked. Murchison, however, although he adopts the view that typhus is most contagious after the end of the first week, thinks that the spreading of the disease in convalescent wards is due to the patients coming more closely

into contact there, but especially to their wearing their own clothes, saturated with the fever-poison before admission, a view which suggested the importance of having such clothes thoroughly disinfected before they are again put on. He believes that the human body soon ceases to give off the poison after the subsidence of the fever.

It is, at any rate, certain that clothes and bedding may become vehicles for the transmission of typhus. Murchison says that laundry-women are especially liable to contract the disease without direct communication with the sick. Barker and Cheyne, in their account of one of the first epidemics, relate that a child discharged from a fever hospital took to another institution a bundle of clothes which had not been disinfected; a woman who opened it perceived an exceedingly disagreeable odour, and in a few minutes became ill with what proved to be the beginning of the fever, of which there had been no previous case there. Haller, of Vienna, thought that dark-coloured materials were more apt to absorb the poison than light-coloured ones;* and I well remember, I used to notice, when demonstrator of anatomy, that the dissecting-room smell adhered to me more strongly when I wore dark clothes. Very few positive instances seem to have been recorded of the communication of typhus by contact with the bodies of those who have died of it; but Murchison, when he was attacked in Edinburgh, had been dissecting in a close room, in which there were many such bodies, and he had never entered the wards of the infirmary, nor seen a case of the disease. On the other hand, at St Bartholomew's Hospital, in 1838–9, the dissecting room received seventeen bodies dead of typhus; but among six students of the hospital who alone took it, four had not dissected at all, and the other two, who had dissected, had been also exposed to contagion in the wards.

In has seldom been recorded that the typhus was conveyed by third persons, not themselves affected with it. But Murchison relates that in January, 1867, a patient in a surgical ward at the Middlesex Hospital was attacked after she had been there for four and a half months; she had been receiving daily visits from a nurse who was in close attendance on a patient with typhus downstairs. There have been some instances in which the disease has been supposed to have been brought into towns by bodies of men, themselves free from it. Thus in 1861, great interest was excited by the case of an Egyptian vessel, the "Sheah-Gehaad," from which typhus was introduced into Liverpool, when thirty-one persons caught it. Although Dr Duncan, on the testimony of the surgeons who attended the men on board this ship, thought that they suffered from no other disease than dysentery ('Trans. Epid. Soc.,' 1861), Dr Parkes came to the conclusion that typhus had really been prevalent among them before they arrived in port ('Army Med. Rep.,' vol. ii). It does not seem to me that much importance is due to the oft-quoted statement of Fodéré that the French army, during the retreat from Italy in 1799, communicated fever to the inhabitants of fifteen towns and villages on its route, but was not itself attacked until it arrived at its destination.

On the whole, then, it appears that typhus is not nearly so apt as the contagious exanthemata to be propagated by means of inanimate objects, or of human beings themselves unaffected by it. And another very important point is that its poison is very easily rendered inert by free dilution

* But the 60th aphorism of Dr Keil, of Northampton, is that "Black cloaths *cæteris paribus* draw the least moisture of any" ('Medicina Statica,' 1720).

with air. Some writers have stated that there is greater risk of the disease spreading from the lower to the upper stories of a house or of a hospital than in the reverse direction; and this, if true, would show that the upward currents of air that exist in all inhabited buildings are capable of carrying the infection with them. But experience has abundantly proved that it never passes from one house to another through the atmosphere. When the London Fever Hospital was one of a row of houses in Gray's Inn Lane, no case of typhus arose in the others; and afterwards, when it occupied its second site at King's Cross, on the same plot of ground as the Smallpox Hospital, and but a few yards off, Dr Tweedie was able to state that during eight years not one of the officials of the latter institution contracted typhus. So, again, Murchison says that if a patient with this disease is placed in a large, well-ventilated apartment, the attendants incur little risk, and the other residents in the same house none whatever.

Origin de novo.—The facts stated in the last two paragraphs form essential links in an argument by which Murchison endeavoured to establish his conviction that typhus, instead of being always due to contagion from a previous case, is often generated *de novo* in persons placed under defective sanitary conditions, of which overcrowding is the chief. For it is true of all other acute specific diseases, as well as of this one, that a great many instances occur of which the origin cannot be traced; but the more perishable the poison the less likely is it to be carried over long distances, and transferred from one vehicle to another, as we know happen with the contagia of scarlet fever and diphtheria. A further link of great importance is that where typhus is seen, independently of obvious contagion, it is almost always among the poor and destitute. One scarcely ever finds it, like the exanthemata, springing up in an unexplained way in the houses of the rich, or even of the well-to-do. The only instances to the contrary that I remember to have heard of is that mentioned by Dr Buchanan, of two boys, living at an institution with every accommodation, who were attacked within a few hours of one another, but who could not, after the most careful inquiries, be shown to have been in the way of catching the disease. No doubt there had been a more or less direct communication of the poison to both of them together, for the simultaneous development of the fever is unintelligible on any other view. The significance of the rarity of such cases is augmented by the fact that those who belong to the upper and middle classes are by far the most exposed to chance infection, because they move about so much more than the poor, come into contact with so many more people, and receive so many more letters and parcels through hands of which they know nothing.

To demonstrate positively the *de novo* origin of a contagious disease, is, from the nature of the case, almost an impossibility. Perhaps the most favourable opportunity that has ever occurred for investigating the point was afforded by the Egyptian ship, the "Sheah-Gehaad," already referred to. She left Alexandria in November, 1860, and, after calling at Malta, arrived at Liverpool on February 16th, 1861. But, according to Dr Parkes, it was not proved that some of the men did not bring the poison with them when they embarked at Alexandria; they numbered 476, and included not only Arabs, but also Nubians and Abyssinians. Of the supposed instances of the spontaneous generation of typhus collected by Dr Murchison, the most important, in consequence of the care and pains which he devoted to its investigation, is perhaps the group of seven cases which arose in Meridian Place,

Bermondsey, in March, 1859, at a time when the disease was at least very uncommon in the metropolis; for during ten and a half months previously only two examples of it had been seen in the London Fever Hospital. But the real force possessed by this and several other similar observations lies in the fact that one single condition was traceable in every one of them, namely, that those who were attacked by the disease had been crowded and huddled together in narrow and ill-ventilated dwellings or sleeping places. They were often also destitute and famished; but that this is not so essential to the development of the disease as overcrowding, seems to be shown by the circumstance that at Dundee, in 1865, an epidemic of typhus was brought about by the inhabitants of the surrounding country flocking into the town in consequence of work being uncommonly abundant and wages good. The occurrence of typhus in an epidemic form has, indeed, been predicted when, as in 1826 and in 1862, destitution and famine have been very prevalent; and such predictions have been verified by the result. It would be impossible to find a better illustration of the conditions under which, if at all, typhus is generated than was afforded by the "Sheah-Gehaad;" the men on board (who were being carried to Liverpool in order to navigate back to Egypt another vessel, a man-of-war, then in that port) seem to have been crowded together in the most shocking manner; some of them were in a state of starvation; the filth and stench between decks were abominable; and, to crown all, the hatches had been battened down on account of bad weather, and vomited matters and fæces had been discharged in every part of the ship.

No less an authority than Virchow has endorsed the opinion that the poison of typhus can be brought into existence by the concurrence of such conditions as these; and the same view has been maintained by the late Dr Hudson, of Dublin. But, after all, it is possible to bring the facts adduced by Murchison into harmony with the opposite doctrine, namely, that the disease as it occurs at the present day is invariably due to contagion from a previous case. The foundation of this reconciliation is the well-ascertained circumstance that debility and impairment of health augment the susceptibility of an individual to the typhus poison. It would seem, indeed, that everyone is originally capable of taking this disease, although the fact of having once passed through it affords an almost absolute subsequent immunity. At the London and Glasgow Fever Hospitals all the nurses who are not thus protected have been found to contract typhus within three or four months after entering upon their duties. In 1833 Dr Tweedie stated that with one exception every physician who had been connected with the London Fever Hospital had been attacked with fever. But in the case of the higher officers of such an institution there is often a much longer interval between the commencement of their exposure to contagion and the date at which they succumb to it. Thus in 1862–3 one of the resident medical officers at the hospital in question attended a large number of cases for more than twelve months before he took the disease, and more recently a person whose duties were daily in the wards and included cleansing of the dirty linen, died of typhus contracted for the first time after fifteen years' service. The opposite extreme occurred in the case of a medical man who caught it from a single visit to the London Fever Hospital.

In these cases it does not appear whether there was any special reason why the contagion should have proved effective at one time rather than another. But it is often possible to trace accidental conditions which favour

its operation; one such condition is alcoholic *intoxication*. Murchison says he has known several instances of persons exposed for months to the poison of typhus in its most concentrated form, who were not attacked until immediately after a debauch. Habitual intemperance probably acts in a similar way: it was once noted that more than one half of the patients admitted for this disease into the Edinburgh and Glasgow Infirmaries had led intemperate lives.

Other favouring circumstances are excessive *bodily fatigue*, *mental anxiety*, and *want of sleep*. Murchison, in support of the popular doctrine that a dread of typhus increases the risk of taking it, cites the case of an Edinburgh medical student who so feared it that he could hardly be induced to enter a ward in which there were any cases; he was one of the first students to be attacked during the epidemic of 1847. Again, the debility which accompanies convalescence from other complaints predisposes to typhus, and in armies it has been frequently observed to follow scurvy. Murchison altogether rejects the improbable notion, which has, however, found supporters, that typhus is less likely to occur in phthisical persons than in others.

Tweedie long ago made the remark that butchers appeared to be comparatively exempt from the liability to typhus, and Murchison suggests that the reason for it is their eating so much nourishing food. Most of the butchers admitted into the London Fever Hospital for the disease have, he says, been out of employment and destitute. Poverty and starvation are, indeed, among the most important predisposing causes. No less than 95·76 per cent. of all the typhus cases observed at the Fever Hospital during twenty-three years, more than 18,000 in number, had been inmates of hospitals or dependent on parochial relief, and many of them had been on the verge of starvation for several weeks or months. Epidemics in Ireland, in England, and on the Continent have repeatedly been associated with the failure of crops and with the widespread destitution consequent; so that although relapsing fever has of late been declared to be the true "famine-fever," that name was formerly often given to typhus, and not without warrant.

Another very important predisposing cause of this disease is *overcrowding*. Beside its obvious influence in increasing the spread of typhus by contagion, there seems to be no doubt that it also augments the susceptibility of individuals to the poison. Thus it is quite possible that it may favour the occurrence of a first case among the occupants of a lodging-house or cellar, even though it should not be capable of generating typhus directly, as Murchison supposed.

Other so-called predisposing causes probably act only by facilitating contagion. Thus epidemics occur rather in the winter than in the summer because the poor for the sake of warmth block up every hole by which air can enter their wretched dwellings during the cold season. But in some years it has happened that more cases have occurred in July, or in September, than in January. So far as is known, the variations of temperature which occur in temperate climates have no direct influence upon the prevalence of the disease. But it is as yet doubtful whether this form of fever has ever been observed in Africa, or in the tropical parts of America, although it is certainly met with in the gaols of India. If very hot countries should prove to be exempt, it will not be possible to attribute the fact to any idiosyncrasy of the inhabitants, like that of negroes in respect of the ague-poison. For Murchison says that several Africans and East Indians have been admitted into the Fever Hospital, and that the rash was characteristic.

It is worthy of notice that patients sometimes themselves attribute the disease to their having "caught cold" or got wet. Murchison says that this has been the case with a considerable proportion of the persons admitted into the London Fever Hospital. In 1856 a young man was taken into Guy's Hospital who gave as the history of his illness that he went for a long walk on damp ground, and felt that he took cold; two days later he had headache and fever, and the typhus eruption followed in due course. In that instance the poison had no doubt already been received, and the relation between the chill and the typhus was one of mere coincidence. But it is quite possible that long-continued exposure to cold and wet may render a person more susceptible than he had been before.

Age and sex.—Typhus may occur in persons at all ages, from one month to eighty-four years old. The quinquennial period at which the disease is most common is from fifteen to twenty; one-half of the cases admitted into the London Fever Hospital have been in patients from ten to thirty. The proportion of males to females among typhus patients scarcely differs from that in the population generally.

Protection.—I have already remarked that typhus protects against the subsequent action of its own contagion. A second attack of this disease is indeed as rare as one of smallpox; far more so than a second attack of measles or scarlet fever. It does, however, sometimes occur, and there was a notable instance of it in the case of Murchison himself, the interval between the attacks being ten years. That writer knew of two other physicians who contracted this form of fever twice, and in one of them it proved fatal on the second occasion. A very remarkable fact, for which he vouches and of which he saw at least six examples, is that during an epidemic, a person exposed to the poison may have what appears to be an abortive typhus fever, malaise, dry tongue, and even slight delirium, but no distinct rash; may recover, sometimes after exactly fourteen days, and then a few weeks later may go through a regular attack attended with the characteristic eruption. Such a "*typhisation à petite dose*" in fact, seems to afford no protection whatever.

There appears to be no evidence whatever that typhus is related to any of the contagious fevers to which the lower animals are subject, although it was at one time imagined that the "cattle plague" might be an equivalent disease. Mosler injected fresh blood from patients with typhus into the veins of dogs, without any result. Zülzer, however, has since been more successful; his injections were made upon rabbits, and he found that when the blood was taken while the disease was at its height those animals died in two or three days, but not if the crisis had been passed.

Diagnosis.—This is seldom, if ever, doubtful when one has an opportunity of watching a case throughout its whole course; but there may be great difficulty in forming an opinion upon a single visit, or if the patient is brought to a hospital a day or two before his death. At the commencement of the disease, what suggests the correct view of its nature is generally the fact that there has been exposure to the specific contagion, or at least that cases have already occurred in the neighbourhood. Otherwise it could scarcely be distinguished from smallpox and the other exanthemata, and not at all from enteric or relapsing fever, some cases of which begin exactly in the same way. As a rule all uncertainty is removed by the development of the eruption, but sometimes, and especially in children, it is by no means easy to say whether this may not be measles rather than typhus. Lebert

makes a point of the harassing cough which belongs to the former complaint, whereas the bronchitis of fever seldom causes the patient much inconvenience. Certain cases which are commonly classed under purpura might also be sometimes mistaken for typhus; they are attended with eruptions which rapidly become petechial, and with much febrile disturbance; and they no doubt depend upon blood-poisoning, the exact nature of which, however, is not yet understood. Lastly, the copaiba rash has been mistaken for typhus, as in an instance alluded to by Dr Hudson.

But it sometimes happens that the mulberry eruption is not so distinct as to be conclusive, or it may even be altogether absent. An error which it is very important to avoid is that of taking for cases of this kind those in which the disease is really an acute pneumonia or an acute meningitis.

As regards *pneumonia*, although the mistake was formerly very common, and although Murchison says he has seen many instances sent to the London Fever Hospital for typhus, I believe that the existence of this disease when its symptoms are such as could be confounded with those of a specific fever, can always be discovered by auscultation, which no careful physician would omit before giving a diagnosis of typhus in a patient without eruption. But it may still be a question whether pulmonary inflammation is or is not a primary disease. Murchison says that those cases of pneumonia in which the apex of the lung is first affected are particularly apt to be unattended with local symptoms, and to assume the mask of a "typhoid" condition. He remarks that if the temperature should reach or exceed 104° after the fourth day of illness the pneumonia is probably secondary.

The distinction between *meningitis* and typhus is one of far greater difficulty. Among the symptoms which point to the former disease are an anxious and pained, or wild and defiant expression of face, intensity of headache, the concurrence of headache with delirium (for in fever pain almost always ceases before delirium sets in), the presence of convulsions early in the case, and the repeated occurrence of vomiting.

Several further questions arise in the diagnosis of cases which come under observation at a time when the patient is comatose and with "typhoid" symptoms already fully developed. Pyæmia may be mistaken for typhus under such circumstances, or if there be erysipelas, jaundice, or delirium tremens, one may be unable to say whether these diseases are complications of fever or altogether independent of it. In tropical climates there may be great difficulty in distinguishing severe forms of malarial fever from typhus. And in any part of the world it may be altogether impossible to tell typhus from enteric fever if the duration of the patient's illness should be unknown. In persons advanced in years, mere bronchitis is sometimes attended with a dry brown tongue, stupor, and other "typhoid" symptoms, the real cause of which may be overlooked.

But the condition which is most apt to be mistaken for typhus at this stage is *uræmia*, dependent upon chronic disease of the kidneys. Murchison says that patients have often been sent to the London Fever Hospital whose symptoms were all of renal origin; and at Guy's Hospital in former years the doubts with regard to such cases have sometimes never been entirely cleared up. Conversely, in 1865, a man who was in a surgical ward for stricture became feverish and drowsy, and his illness was attributed to an ascending nephritis, until a mulberry rash was discovered upon him; he died, and the kidneys were found to be healthy. It must be borne in mind that the existence of a chronic renal affection, when established by *post-mortem*

examination, affords no proof that typhus was not also present. According to Murchison, however, the thermometer generally affords a sufficient means of diagnosis; the temperature is at or below the normal in cases of Bright's disease, unless complicated with an acute inflammation.

Prognosis.—It is very important to remember that the prognosis in typhus must be based upon a variety of considerations, apart from or in addition to the mere severity of the symptoms. Chief among these is the *age* of the patient. The older he is, the greater in this disease is the danger. In children it seems never to be fatal except by some complication. Murchison found that at the London Fever Hospital, the average death-rate for all ages being from 15 to 19 per cent., the rate in persons above thirty was 35·39 per cent., in those above forty it was 43·48 per cent., in those above fifty it was 53·87 per cent., in those above sixty it was 67·04 per cent.

Again, typhus is especially apt to terminate fatally in persons who have been intemperate, who have suffered severely from gout, who have been exhausted by fatigue of body or mind, or who have been suffering from privation of food or from severe mental depression. The greater frequency with which these various conditions occur among adult males, as compared with females, is probably the reason why the mortality is higher in them; among children between five and fifteen it is lower in boys than in girls. Patients who have gone on struggling against the disease during the first few days often do badly; having exhausted their muscular power, they become rapidly prostrate.

With regard to the prognostic significance of particular symptoms, I need add but little to what has already been incidentally stated. Murchison remarks that a presentiment of death is a very unfavourable, but not necessarily a fatal, indication. The danger in a given case is generally best measured by the intensity of the cerebral disturbance and by the degree of prostration; the earlier the date at which severe symptoms are present the worse is the prospect. Sleeplessness continued throughout several days is of evil omen. So is the occurrence of convulsions. A very abundant rash is usually a bad sign, especially if the spots are dark and rapidly become petechial. A pulse over 120 is always a serious matter in an adult; and when it exceeds 150 death is almost certain to occur. Relaxation of the sphincters before the tenth day is unfavourable; but towards the end of the second week it is not uncommon in severe cases, which may nevertheless do well. Great lividity of the face and limbs and coldness of the distal parts of the body with profuse sweating are among the most threatening symptoms. A well-marked "typhoid" state, however, with dry, brown tongue, sordes, and subsultus is observed in many patients who recover. Indeed, even in what appear to be the worst cases, one should hardly ever despair.

Treatment.—We as yet know of no means of arresting the course of typhus, or of bringing it to a uniformly favourable termination; but a great deal may be done to increase the chance of a patient's recovery, and many of the most painful and distressing symptoms may be alleviated or checked. Murchison cites observations made both at Philadelphia and at Belfast, from which it appears that the death-rate from this disease was greater in proportion to the duration of the fever before admission into hospital.*

* It must not be supposed that neglect of medical assistance was the sole, even if it was the principal cause of the varying mortality in question; in many instances a fatal issue was doubtless the direct result of removing the patient at an advanced stage of the illness,

When typhus attacks a person in comfortable circumstances, he may be safely left at home, provided that his apartment is spacious and well-ventilated, and that he can be properly nursed. But those who are poor should at once be carried to a hospital. The establishment of special institutions for the reception of fever cases dates from the commencement of the present century; but so recently as 1842 many London physicians, among whom was Richard Bright, were in favour of mixing such cases with others in general wards, rather than of setting apart special wards for them, or of collecting them in buildings devoted to that particular purpose. However, so far as typhus is concerned (and the same may be said of relapsing fever) this was certainly a mistake. For (1) it has now been proved by experience that if a sufficient cubic space of 2000 feet be allowed, and if ventilation be well attended to, the death-rate is not greater among typhus cases accumulated in the same ward than when they are scattered. (2) In every hospital in London where such cases have been placed in general wards there have been terrible examples of the spread of fever to other patients. To cite but a single instance: in 1862 one or two cases of typhus were admitted into one of the large medical wards in Guy's Hospital; seven other patients took the disease, and five of them died. What makes the argument stronger is that in a large proportion of medical cases, particularly in those of Bright's disease and of diabetes, the risk of a fatal termination, if typhus should be caught, is very much greater than among healthy persons. Moreover, the free ventilation, which is the chief condition of safety under such circumstances, is directly prejudicial to persons suffering from pulmonary complaints. It would be far safer to admit smallpox cases into a general ward than those of typhus, because protection against the former disease could easily be given by a general vaccination. Murchison has shown that the proportion of cases of typhus which have arisen at the London Fever Hospital, in comparison with the number of cases admitted, has been far less than in the general hospitals of the metropolis. (3) So far as medical men and nurses are concerned, the danger of the extension of typhus may be greatly reduced by retaining for as long a period as possible the services of those who have once passed through the disease, and by invariably selecting persons under thirty, for all vacancies that may arise. Every large town ought to have a fever hospital, capable of enlargement during an epidemic by the erection of temporary buildings of iron or wood. All general hospitals should have one or more separate fever wards.

A patient suffering from typhus should be placed upon a spring bed, or upon a hair mattress, with not too many bedclothes. The temperature of the ward or room should not exceed 60°. After the first few days he should not be allowed to get up even to pass his excreta. It is important that his nurse should be strong enough to raise him when necessary, and instructed enough not to lift him into a sitting posture. From time to time he should be turned over to one side or the other, and kept from rolling back by pillows.

Food.—A nourishing diet is of the highest importance. Dr Graves, of Dublin, desired, as his own future epitaph, the testimony, "He fed fevers;"

whereas at an earlier period it might have done him no injury. Murchison says he has repeatedly known patients die from exhaustion after being carried for several miles in a shaking vehicle. But that, with proper precautions, a moderate journey may be effected without risk appears from the fact that, among a number of patients admitted into the London Fever Hospital from the immediate neighbourhood, the death-rate was during five years almost exactly the same as among those brought from the distant parish of St George's-in-the-East.

and it is to typhus rather than to any other fever that this is applicable. So long as the patient can be tempted with food, it is important to vary it as much as possible. Besides milk, eggs, and beef-tea, he may have broth made of mutton, veal, or chicken, meat-juices and extracts, jellies, custards, blancmange of isinglass or ground rice, white wine-whey, &c.; and vermicelli may be given in the beef-tea, small pieces of bread or toast in the broth, and arrowroot in the milk.

For beverages he may be allowed to choose among barley-water, toast-and-water, lemonade, tamarind-water, currant-water, effervescing drinks, and cold weak tea. Murchison recommends that he should drink often, in order to keep up free excretion by the kidneys, but he must only take a small quantity at a time. Before many days have passed, however, he acquires a distaste for everything but cold water. It is then essential that food should be pressed upon him and administered at regular intervals of from one to three hours; to give it more often than this is, according to Murchison, injurious rather than useful. If he be drowsy, he should be roused for the purpose, and this can generally be done without seriously disturbing him; but if he should be sleeping quietly after having been very restless, the interval may sometimes be a little lengthened. It is, however, of great consequence that his strength should not be allowed to run down at night, or during the early morning, when the risk of exhaustion is always greatest. When, as is often the case, he clenches his teeth, and obstinately refuses food, it must either be introduced into the stomach by means of a tube passed through the nostril, or else injected by the rectum. When there is obstinate vomiting the latter is the only plan that can be had recourse to.

Stimulants.—In many cases the administration of alcohol is a very important part of the treatment. Whether it should be regarded as a food or as a drug, is still a question. The former view, in which I for my own part concur, is held by Buchanan; the latter was held by Murchison. But as to the practical rules for its use there is at present very little difference of opinion. All observers are agreed that alcohol seldom need be given to children, that it is almost always required for patients over forty, and that persons whose habits have been intemperate must have it earlier and in larger quantities than others. The state of the heart affords the best indication; the necessity for stimulants is greater in proportion as the cardiac impulse is feeble, the first sound low, and the radial pulse rapid, compressible, or, above all, irregular. The propriety of continuing to give alcohol may often be determined by its effect on the heart's action. If the pulse becomes quicker than when the patient was taking none, it is likely to do harm; if slower, it may be expected to do good. It is most needed during the second week; very seldom before the appearance of the mulberry rash. Low muttering delirium is often controlled by stimulants; a dry, brown tongue becomes moist, and other "typhoid" symptoms become less marked. On the other hand severe throbbing headache and violent maniacal excitement are often aggravated. A burning dry skin is so far a reason for withholding alcohol; profuse perspiration, especially if the limbs are cold, calls for an increased supply. As a rule it should not be given when the urine, although scanty, is of low specific gravity, or when there is suppression of the renal function.

Probably there is no direct advantage in employing one alcoholic liquor rather than another. Some observers prefer brandy or whisky, others port wine; Buchanan speaks highly of beer. The amount must vary with the

urgency of the symptoms. Murchison thought that it was very rarely necessary to give more than eight ounces of brandy in the twenty-four hours, but some physicians order as much as twenty ounces. Of port wine as much as a bottle may be taken with advantage by patients who are desperately ill. But only small doses should be allowed at a time, repeated very frequently, at intervals of an hour or two; and when the daily quantity is very considerable, the fact that milk mixes with brandy much better than with wine is an important reason for preferring it. When there is great prostration with cold sweats, hot brandy or whisky-punch or hot wine-whey is recommended by Murchison. At Guy's Hospital it has of late been the fashion with house-physicians to inject stimulants under the skin in such cases. The practice was I believe first suggested by Zülzer. Thirty or forty drops of brandy, or of the spirit of sulphuric ether, may be administered in this manner. Ether, carbonate of ammonia, camphor, or musk may also be given by the mouth.

Treatment of symptoms.—The *pyrexia* itself seldom requires direct treatment in typhus. So far as I know, the routine use of cold baths has not yet been sufficiently tested in this disease to prove its value. Probably, when the temperature rises high cold affusion or the wet pack is better. Murchison satisfied himself that quinine in large doses of ten or twenty grains, although it lowers the temperature by three or four degrees for twelve or eighteen hours, does no real good. Only in exceptional cases, when at the crisis the thermometer was rising instead of falling, did it seem to him to be sometimes instrumental in saving life. The medicine usually given at the London Fever Hospital appears to be dilute hydrochloric acid, in doses of twenty minims, with a little syrup and tincture of orange. Murchison says that he has often "observed the tongue become moist, and a marked improvement follow the commencement of the acid treatment, at whatever stage it was tried." When there is insatiable thirst, he recommends a very weak infusion of cascarilla or quassia.

The early *headache*, which is often a very distressing symptom, may sometimes be checked by an emetic. When the patient is young and robust, there is no objection to applying three or four leeches to the temples, and they frequently give complete and permanent relief. Or the hair may be cut, and the head be covered with a bag of ice, or a tubular cap through which cold water is running. In other cases a blister to the forehead is of service; or hot fomentations, as recommended by Graves, may be employed, especially in old or debilitated subjects. But in many instances it is advisable to prescribe opium for the relief of headache, particularly when it is accompanied by an inability to sleep.

Murchison says that *sleeplessness* at any stage of typhus, if it continues for two or three nights, is of itself sufficient to kill; and that whenever it lasts for thirty-six hours it should be combated by medicine. During the first week one may almost always give fifteen minims of Battley's liquor opii or a quarter of a grain of morphia at night, following this, if necessary, by another half dose two hours later. But at more advanced periods of the disease there is often danger in prescribing these drugs. When the patient is in a state of maniacal delirium, Murchison recommends chloral hydrate in a dose of twenty grains, which may be once repeated should the first dose not succeed. If this treatment fails, recourse must be had to opium, with which antimony may often be combined, as suggested by Graves. The chloral hydrate may also be given in some cases in which the delirium

is low and muttering or like that of alcoholic origin; but when the heart's action is feeble or irregular, it sometimes does harm. Murchison then preferred opium, prescribing it in conjunction with tincture of digitalis and sulphuric ether. Other drugs which are more or less useful are bromide of potassium, the extract of Indian hemp, and the tincture of hyoscyamus or of belladonna. They must each be given in rather large doses to do any good.

When profound *stupor* appears to threaten the patient's life, Murchison advised that a cupful of strong coffee should be given every three or four hours. He also thought it important to use dry cupping to the loins, and to apply mustard poultices, or flannels wrung out of hot water and covered with mackintosh, especially when the urine was scanty and albuminous. He believed that he had saved some cases by blistering the scalp with a piece of lint soaked in strong liquor ammoniæ, and applied for five or six minutes under oiled silk. Should high fever accompany coma, cold affusion to the head may sometimes be employed with great advantage.

For the *pulmonary complications* of typhus, ammonia is the chief remedy. But in some cases turpentine is said to be even more decidedly effectual; it is given in doses of fifteen minims every three hours. Mustard poultices should also be applied to the back and sides of the chest.

For *inflammatory swellings*, usually in the parotid region, both Murchison and Buchanan recommend the application of a blister at an early stage, for it sometimes appears to prevent the occurrence of suppuration. When an incision is required, it should be made early.

Convalescence.—After the subsidence of the pyrexia, the patient should be kept upon a restricted diet for the first two days; but upon the third day, if the tongue be clean, he may have a little boiled fish or chicken or the lean part of a chop. If wine or brandy has been given, beer should be substituted while the case is still under treatment, so that there may be no excuse for carrying on the habit of tippling when health is restored.

When pyrexia has disappeared, the patient, as a rule, recovers his strength as well as his appetite with rapidity. Relapses are excessively rare; neither A. P. Stewart, Sir William Jenner, nor Murchison met with a single case. Sequelæ occur seldom, in striking contrast to their frequency after enterica. Perhaps the least rare are deafness and phlegmasia dolens. Moreover, while a tedious convalescence often leaves the typhoid patient weaker for years, typhus, like acute pneumonia, leaves no seeds of future disease behind it. Hence the joys of returning strength are unalloyed by misgivings for the future. The contest with this disease is often perilous and usually severe, but it is neither protracted nor indecisive. *Horæ Momento cita mors venit aut victoria læta.*

RELAPSING FEVER

History of its distinction from Typhus—Its occurrence in Epidemics—Course of Relapsing Fever—Occasional mortality, direct or indirect—The spirillum—Mode of contagion—Famine as a predisposing cause—Diagnosis—Treatment.

We shall hereafter find that the differentiation of enteric fever from typhus was brought about by a long series of researches which occupied all the first half of the present century. During the same period a third fever also was discovered which is generally known as Relapsing Fever,* from its regularly consisting of two or more attacks separated by intervals of apyrexia.

According to Murchison, the distinction between this affection and typhus was first drawn in Ireland in 1826, when they both prevailed at the same time; but there is evidence that it also formed part of the epidemic of 1817–19, and of several earlier Irish epidemics, as far back as one recorded by Rutty as having occurred in 1739. Being far less fatal, and of shorter duration than typhus, it was in 1826 regarded as a mild variety of that disease. But in the year 1842, when it next appeared, Dr Henderson, of Edinburgh, brought forward good grounds for believing that it was a distinct fever and due to a different poison. The same view was strongly upheld by Jenner in 1849–51. The arguments used by these writers were chiefly three. 1. That the symptoms and course of the two diseases were different. 2. That even when they prevailed together in the same town, one could never be traced to infection from the other. 3. That neither of them afforded protection against a subsequent attack of the other. To these arguments may now be added (4) the fact that in relapsing fever the blood always contains a microscopic organism, a spirillum or spirochæta, which is absent in typhus. But although the doctrine in question is at the present time universally accepted as true, the evidence adduced under the second and the third heads appears to have been somewhat weakened by the investigations which have been made since the publication of Henderson's and Jenner's papers. For whereas they maintained that cases of relapsing fever were not proved to come from the same houses or families as those of typhus, Murchison has shown from the later records of the London Fever Hospital that exceptions to this rule sometimes occur, and moreover that in certain narrow streets or courts, it is no uncommon thing for typhus fever gradually to replace relapsing fever in the course of some months. Again, since it has been ascertained that the same individual often has relapsing fever more than once, the fact that this disease does not protect against typhus has lost its significance. But in the opposite direction it has still been thought that there is an immunity. Dr J. C. Steele (then of Glasgow, now for many years our valued Superintendent at Guy's Hospital) remarked that in the epidemic of 1848 persons who had previously suffered from typhus were not attacked by

* *Synonyms.*—Febris vel typhus recurrens, fièvre à rechutes, bilious remittent, seven-day fever, famine fever.

relapsing fever; and the same thing is said to have been noticed recently in epidemics at St Petersburg, and at Breslau. Dr Henderson, however, related six cases in which typhus occurred first and relapsing fever afterwards; and Murchison says that of thirty-one persons who contracted relapsing fever in the London Fever Hospital in 1868–69 no fewer than thirteen were known to have had typhus. Lastly, Lebert draws attention to the fact that in his cases of typhus, among those who had already passed through relapsing fever, the mortality was only half as great as in the population generally. Do these facts indicate that after all there is between the two diseases some connexion, the nature of which is at present not understood?

At the beginning of the great epidemic of 1847–48, and also in 1851, it was particularly noticed, both in London and in other large towns of Great Britain, that the chief patients were destitute Irish, many of whom had recently left their own country; afterwards the English or the Scotch were attacked. Whether in 1842 the disease was derived from Ireland is not clear. The latest epidemic, that of 1868–71, seems to have had its origin in the east of Europe. There is no doubt that in 1847 relapsing fever had prevailed in Upper Silesia. With this exception it is not known to have ever existed on the Continent of Europe, until in 1863 it was observed in Odessa, and in 1864 at St Petersburg. Next, in 1867, it broke out for the second time in Silesia, and in the following year it prevailed in Berlin and in other German towns, as well as in Breslau. In 1868 it reappeared in England. The first case, that of a woman of Irish birth who had been for several years in London, was brought into the Fever Hospital on the 4th of July. Four days later, another case occurred in the person of a Polish Jewess, who could speak no English; she came from a house about a quarter of a mile distant from that in which the former patient resided. From the same house, on the same day, a Polish family was admitted consisting of father, mother, and child; they, however, were merely in a state of extreme prostration, having no fever then upon them. Three weeks later there arrived from the house next adjacent, a girl who had lived all her life in the metropolis. The only other persons who are known to have been attacked in London in that year were eight German Jews; they all were admitted into the Dalston German Hospital. But in October, a severe outbreak occurred at Tredegar in South Wales. In the autumn of 1869 relapsing fever became epidemic in several of the large cities of England; in London the disease reached its height in December of that year, and then gradually declined until June, 1871, when it finally disappeared. In Breslau it was again epidemic in 1872–73. I think there can hardly be any doubt that the Poles admitted into the Fever Hospital in 1868 had either directly introduced the contagion from their own country, or perhaps had received it in a letter or in a parcel, or had derived it from other Poles or Germans who had themselves so received it or brought it with them from the Continent. In a city like London one can but seldom expect to trace such events to their sources, and it is instructive to observe that the disease did not spread then until a year later, although it had in the meantime broken out in a remote part of Wales. In September, 1869, it appeared at Philadelphia, and in November at New York. The patients were chiefly poor Irish or Germans, and it seems to be almost certain that the contagion was imported from Europe, although its origin could not be discovered. On two or three former occasions it had been introduced into the very same cities by Irish emigrants, but it never showed any tendency to spread among the American population.

With regard to the occurrence of relapsing fever in Africa and in Asia, our information is still very imperfect. Griesinger observed it in Egypt in 1851.* There is reason to believe that it has repeatedly prevailed in India. The latest epidemic there, that of 1877 at Bombay, was carefully studied by Dr Vandyke Carter ('Med.-Chir. Trans.,' vol. lxi).

Course.—The period of incubation for this disease appears to be very variable; Murchison says that it sometimes breaks out immediately after exposure to its exciting cause, sometimes not until from two to fourteen days have elapsed. During the interval, no symptoms whatever are present; only in some very exceptional cases is there malaise, with a little loss of appetite for a few hours, or a day or two, before the beginning of the attack. This generally takes place with remarkable suddenness. The patient, while engaged as usual during the day, or perhaps on first waking in the morning, is seized with chilliness or with rigors; his head aches, he feels giddy, he has pains in the back and limbs. In a short time his skin becomes burning hot. All his complaints grow rapidly worse; he at once takes to bed, and he often seeks admission into a hospital on the first or the second day. One of the distinctions from typhus is that it is giddiness rather than prostration which prevents his keeping about. He is sometimes able to walk to the hospital two or three days after the seizure.

The thermometer indicates a very rapid rise of temperature. This begins even before the rigor, and within twelve or twenty-four hours it reaches 104° or 106°. The pulse, too, becomes quickened much earlier than in typhus; it is scarcely ever found below 110, and not infrequently it reaches 140 on the second day. The respiration is not quickened in proportion to the pulse and temperature. The tongue is moist and covered with a white or yellowish fur. There is great thirst. Appetite is generally wanting, but sometimes it remains good, or even voracious, the patient being able to take solid food without any harm resulting. In some cases, however, there is much nausea, and even vomiting of a green, bile-stained fluid. Pain, with tenderness on pressure, is often present in the epigastric and the hypochondriac regions of the abdomen. By percussion one may make out some increased dulness of the liver; the spleen usually becomes very decidedly enlarged, so that its edge reaches far below the costal cartilages. Jaundice often occurs on or after the third or the fourth day; in some epidemics it has been observed in one of every four or five cases, but generally not oftener than in one of eleven or twelve. It is now and then very bright, and the urine may be loaded with bile pigment; but the fæces appear always to remain of a dark colour.

The face is flushed and the eyes are injected in relapsing fever, but the countenance is not usually dusky, nor is the expresssion stupid and confused as in typhus. As a rule, there is no eruption upon the skin; but several observers have noticed a roseola in certain exceptional cases, and in the epidemic in Silesia of 1847 a rose-rash on the second or third day was the rule. Murchison, who met with this in eight out of about six hundred cases, says that the rash consisted of small spots, or of a reddish mottling, sometimes resembling measles, but more often undistinguishable from typhus at an early stage; yet always disappearing under pressure, and fading after a few hours or within three or four days at the latest. It came out sometimes during

* He saw a fever in Egypt which he called Bilious Remittent, and afterwards visiting London saw cases of undoubted Relapsing Fever and identified the two diseases, but it is not certain that he was correct.—Ed.

the first attack, sometimes in the relapse; and either as early as the third day, or immediately before the crisis. Petechiæ are not uncommonly present; no doubt fleabites have frequently been taken for them, but Murchison says that this is certainly not always the case, for large numbers of them may come out in a single night after the patient's admission into hospital. The surface of the body often remains very dry, but sometimes on the second or third day there is profuse sweating which may last for hours without in any way relieving the other symptoms of the disease. Sudamina may be developed in large numbers; I suppose that they are the cause of the marked desquamation of cuticle which frequently occurs at a later period.

When relapsing fever occurs in a pregnant woman, gestation is almost invariably brought to an end, whatever may be its period; but it is curious that the abortion or miscarriage is not seldom delayed until the relapse. The child, if not stillborn, survives only a few hours. There is, in these points, a marked contrast with what occurs in typhus.

In the regular course of the disease, the patient's condition undergoes but little alteration after the first day or two, for about a week. The temperature and the pulse remain high, oscillating upwards and downwards a little at one part of the day or another, the pulse-rate sometimes reaching 150, 160, or even 180. The patient continues to suffer from severe headache, often of a throbbing character, and from intense pains in the muscles and joints, much increased by movement. Murchison remarks that relapsing fever is altogether attended with far more suffering than typhus; persons who have passed through both diseases invariably look back upon the former as the worse to bear. Reasons for this are that sleeplessness is almost always a marked symptom, and that the mind generally remains clear, although there may be a little delirium towards the end of the attack.

It is generally on the fifth or the seventh day that a sudden subsidence of the fever occurs, constituting the most remarkable instance of a *crisis* with which we are acquainted. Sometimes, however, this takes place as early as the third day, sometimes not until the tenth. Just before it, the fever often reaches its *acme*, touching a point higher than at any previous time; there may even be a rapid rise through as many as 4°. In some exceptional instances a maniacal delirium suddenly comes on at this time, the patient screaming and struggling violently, and passing his evacuations under him, but within a quarter of an hour becoming again calm and having no recollection of what has occurred. In other cases the crisis is said to be ushered in by epistaxis, diarrhœa, or the catamenia. But, as a rule, what characterises it is the occurrence of profuse sweating. The change is wonderfully complete. In the course of a few hours the temperature becomes normal, or even lower than normal, having fallen probably 8° or 10° without a break; Murchison cites one case in which there was a range of 13° in six hours, and another in which the difference amounted to 14·4° in twelve hours. At the same time, the pulse drops from 120 or more to about 70, the tongue becomes clean, and the patient loses all his pains. Lebert remarks that the period from evening to morning is that at which the crisis is most apt to occur. Litten has pointed out that it is sometimes preceded by a *pseudo-crisis*, the temperature falling below normal, but within twenty-four hours rising to as high a point as before, again to fall on the following day.

During the *interval* which follows, the patient feels perfectly well. At first he is rather languid and exhausted; but his appetite is good, he quickly regains his strength, he gets up and walks about, he may insist upon leaving

the hospital, and may even return to work. His temperature for two or three days is almost always lower than in health; but afterwards it becomes normal. On the other hand, the pulse is at first rather above the natural standard; but at a later period it is often remarkably infrequent, perhaps not more than 44 or 50. Murchison remarks that in such cases it may rise to over 100 when the patient assumes the erect position.

Relapse.—In exceptional cases this apparent convalescence is in reality the end of the disease. But, as a rule, it is abruptly terminated, when it has lasted seven days, by a sudden reappearance of all the symptoms which had belonged to the first attack. Sir Robert Christison related a striking anecdote in reference to his colleague, Dr Hughes Bennett, who took relapsing fever at the commencement of the epidemic of 1843, when its characters were known only by the older members of the profession. "When he had detailed to me his case," being then supposed to be convalescent, "I told him he had sustained an attack of my old acquaintance . . . whose face I had not seen for a good many years; that he was not yet done with it, and that he would have another attack, commencing with rigor, on the fourteenth day" (of the disease). "Dr Bennett, surprised—I will not say incredulous—replied that the relapse had no time to lose, as there were only three or four hours of the fourteenth day to run. It did, indeed, lose no time, for I must have scarcely reached home from his house before the rigor set in with violence." Sometimes, however, instead of occurring on the seventh day of the interval, the relapse begins on the second, or the third, or the fifth day; sometimes it is postponed for several days, and even as late as the twenty-fifth day. Lebert says that it usually sets in at night.

This second attack is generally shorter than the first one, lasting three days, or not more than one or two, but sometimes it is prolonged to five days, or even to as many as seven or eight. Both in the details of their symptoms and in their severity the two attacks may resemble one another exactly, but in some patients the relapse is the worse, especially when the original attack was very mild; in others it is by far the less severe of the two, and it may be so slight as almost to escape notice, being indicated only by a trifling malaise with a little elevation of pulse and of temperature. When it is well marked, it ends in a sudden crisis, just like the former one.

Recovery.—The patient now generally passes on to a permanent recovery, but sometimes there is a third attack, which begins between the twenty-first and the twenty-fourth days of the disease; it is almost always very mild, and seldom lasts more than forty-eight hours. There may even be a fourth or a fifth attack. In any case convalescence after relapsing fever is slow, as compared, for example, with that after typhus. The patient is a long time in regaining his strength. Notwithstanding that the duration of the disease up to the second crisis is only about eighteen days, he is seldom able to return to work within six weeks.

I have already remarked that relapsing fever seems to confer little or no immunity from subsequent attacks. All writers record instances in which persons have taken the disease two, or even three, times in the same epidemic.

Fatal event.—It is only in exceptional cases that relapsing fever proves fatal. Murchison states that in this country the mortality has been 4·03 per cent. In Bombay, however, Dr Vandyke Carter estimated it at 10 per cent. In persons advanced in years the disease is decidedly more dangerous than in younger persons. One way in which death may occur is by sudden collapse. At and after the crisis the pulse is often very feeble, small, or irre-

gular; there may even be a temporary impairment of the first sound of the heart and of its impulse. These symptoms usually soon disappear, but the liability to their occurrence probably affords some explanation of the fact that a patient who may have had a mild attack, and who may have appeared to be doing perfectly well, is now and then found to become pulseless, cold, livid, and unconscious, and dies in a few hours. This sometimes takes place at about the period of the first crisis, but sometimes during the interval. In three such cases, observed by Murchison, the heart was found fatty and dilated. Another mode of death is by suppression of urine leading to convulsions and other cerebral symptoms. In some instances of this kind, recorded by Dr Henderson as far back as 1843, Dr Maclagan discovered urea not only in the blood, but also in fluid from the cerebral ventricles. It is probable that on *post-mortem* examination the kidneys would be found to be in a state of chronic disease, and that albuminuria would have been present at an earlier stage of the attack, but the urine not seldom contains albumen, and even blood, in relapsing fever, without any serious consequences resulting. Again, there is a variety of the disease attended with typhoid symptoms, and invariably accompanied by jaundice, which often terminates fatally; this Griesinger described under the name of "bilious typhoid" as a distinct form of fever. In it the interval is imperfectly marked, the pyrexia running on more or less continuously from the first attack to the second. As, however, Heydenreich, in St Petersburg, and Carter, at Bombay, have each shown that the spirillum was present in cases of this kind, we may now safely follow Murchison in rejecting Griesinger's view with regard to them. Among his symptoms are hæmorrhages into the skin and from various mucous surfaces, including that of the stomach, so that the vomited matters may be black, like those of yellow fever.

There is little to be said about the state of the organs in the cases of relapsing fever which prove *directly* fatal. The spleen, if death occurs during the attack, is greatly enlarged, more so than in either typhus or enteric fever. Küttner is said to have found this organ four and a half pounds in weight. It is often, but not always, soft. Infarctions are sometimes seen in it. In the one case which came to an autopsy at Guy's Hospital during the epidemic of 1869–71, the spleen, which weighed fifteen ounces, had a remarkable appearance. It contained a number of small yellow softened patches or abscesses, the largest being of the size of a horse-bean, which seemed to follow the branchings of minute veins. Dr Moxon could discover no thrombi in any of the vessels, even with the aid of the microscope. Very similar appearances have been described by Litten as having been observed at Breslau in the epidemic of 1872–73. As a rule, the kidneys are gorged and swollen, and the epithelium in the renal tubules is in a state of cloudy swelling.

In other cases, however, relapsing fever proves fatal *indirectly*, through some secondary lesion arising as a complication. Thus pneumonia has in certain epidemics been of rather frequent occurrence, as, for example, at Breslau in 1872–73. Lebert says that it was generally double, that it occurred chiefly in those who had been intemperate, and that it sometimes set in during the attack, sometimes in the relapse. It has been known to lead to pulmonary gangrene. Bronchitis is commonly present, but it is seldom severe. In St Petersburg hæmorrhagic pachymeningitis is said to have been observed. The spleen has sometimes ruptured, discharging a quantity of blood into the peritoneal cavity, and causing death by rapid col-

lapse. In other instances an infarction in this organ has broken down and set up a fatal peritonitis or pleurisy. Another cause of peritonitis may be dysentery, which has been a frequent and a very dangerous complication in some epidemics. The occurrence of abortion is sometimes fatal by profuse hæmorrhage, or by rapid sinking. Inflammatory swellings or "buboes" in the parotid regions or in the groins are said to have been often the cause of death at St Petersburg, but in England they appear to have been more frequent in cases which recovered.

Sequelæ.—The disease sometimes leaves trouble behind. Severe articular pains often continue during convalescence, and effusion into the knee- or ankle-joint has now and then been seen.

Another affection, which sometimes does not set in for several days, weeks, or even months after the subsidence of the fever is a form of ophthalmia. This is described as beginning with an amaurotic stage, in which the retina, or perhaps the choroid, is alone attacked; afterwards, in what is termed the second or inflammatory stage, when the more superficial structures are involved, there is intense pain in and around the eye. It fortunately seldom happens that both eyes are affected, for recovery is always tedious, and there is sometimes permanent loss of vision.

Origin and spread.—In reference to the ætiology of relapsing fever, the first question is as to the significance of the minute organisms which are present in the blood, and which were originally described by the late Dr Obermeier, of Berlin, in 1873, although he had observed them as far back as 1868. They are generally known as *Spirillum Obermeieri.** It seems to be doubtful whether the form found in relapsing fever is identical with that which had previously been observed in water containing decaying vegetable matters (*S. plicatilis*, Ehr., Cohn) and in the saliva of healthy persons (*S. denticola*). Dr Vandyke Carter, at any rate, says that the dimensions of an organism of this kind which he met in the water of a tank at Bombay were much larger than those taken from his fever patients ('Med.-Chir. Trans.,' vol. lxi). Provisionally we may leave to the former Ehrenberg's name of *S. plicatilis*, distinguishing the latter as *S. Obermeieri.*

Its ordinary appearance is that of a delicate, homogeneous, spirally-twisted filament; its length is from $\frac{1}{2000}$ to $\frac{1}{500}$ of an inch, or equal to from one and a half to six times the diameter of a red blood-disc (*circa* 15—40 μ). It is never still, and its motion is compounded of a rotation on the long axis, a progression forwards or backwards, and a lashing movement, which lengthens it out and causes the twist for a time almost to disappear.

All observers admit that the presence of the spirochætæ is coincident with the attacks of relapsing fever, and that during the interval or intervals it cannot be detected. Birch-Hirschfeld, indeed, once found it for two days after the second crisis, and several writers, including Litten, have stated that it is seldom to be seen during the first two or three days of the disease. But Heydenreich, of St Petersburg, who has since made what seems to have been a careful series of investigations, declares that while it appears before the thermometer begins to rise, it ceases to be discoverable before the commencement of the crisis. He has specially inquired into the influence of heat upon this organism outside the body, and he finds that it remains alive,

* They are also called *Spirochæta*, for Ehrenberg in 1833 distinguished two genera of *schizomycetes* under these names, the difference between them being that *Spirochæta* possesses greater flexibility than *Spirillum*. This distinction is preserved in the nomenclature of Cohn and Flügge, but the two "genera" are closely allied and the names are practically interchangeable.—Ed.

as evidenced by the persistence of active movements, much longer when it is kept at a temperature of 60° or 70° F. than at blood-heat, whereas at fever temperatures it dies more quickly still. His hypothesis accordingly is that the pyrexia, which is itself caused by the existence of this organism in the blood, proves directly fatal to it. In all probability the filament breaks up into a number of minute granules, and some of them may perhaps constitute the germs from which fresh crops of spirochæta are afterwards developed. Heydenreich has occasionally seen some of the filaments beset with granules so as to resemble a necklace, and the late Dr T. R. Lewis, of Calcutta, speaks of having once noticed a beaded appearance. If the germs remain alive in the blood throughout the whole of the interval, they must possess the attributes of *Dauersporen*, for the spirochæta itself dies at the temperature of the blood in about twenty hours.

Even during a single attack, Heydenreich finds that there are extraordinary fluctuations in the numbers of the spirochætæ from day to day. He therefore supposes that successive generations are more or less constantly being produced throughout the fever. Sometimes, after the filaments have been present for two or three days, they suddenly cease to be discoverable; but a few hours later they reappear in greater or less abundance. Their recognition in the blood, at the end of an interval, has repeatedly enabled him to foretell the approach of a relapse, which, however, he admits may be so slight as to be scarcely noticeable. On the other hand, Dr Carter says that at Bombay he sometimes detected the spirochætæ at periodic dates, when there was absolutely no rise of temperature; while in other cases a single one-day febrile attack would occur at the proper time for a relapse, without the blood containing the organism. This observer remarks that the numbers of them must often amount to hundreds of millions, dozens being seen in the field of the microscope at the same time. It even seems likely that they may become aggregated together into dense masses with blood-corpuscles, and seriously interfere with the circulation, so as to produce lividity of the face, &c. In fatal cases they cease to be discoverable after death. None of the solid organs have hitherto been found to contain them. Heydenreich could neither detect them in the urine, nor in the conjunctival secretion, in the fluid from the pleura, the intestine, or the bronchial tubes.

As to the origin of the *Spirochæta Obermeieri* we at present know nothing whatever. It has been successfully cultivated out of the body, but all attempts to reproduce the disease experimentally for a long time failed. Injections of blood infected with it into the circulation of dogs, rabbits, or guinea-pigs were made by Obermeier himself without result.* Nor did he find that the disease was propagated by the penetration of minute quantities of blood from patients with relapsing fever into scratches upon the hands of healthy persons, although Dr Carter has since recorded the fact that a few days before he himself was attacked he had scratched his own finger in making an autopsy upon a fatal case.

It is, however, certain that contagion from the sick plays a very important part in the spread of the disease. Thus in hospitals it is exceedingly apt to pass from patients to nurses and clinical clerks. In 1843 the post of house-physician in a fever hospital at Edinburgh had to be filled six times in

* The same negative result followed inoculation of sheep. But Dr Vandyke Carter succeeded in inoculating monkeys with the spirillum taken from the blood of patients suffering from relapsing fever. The organisms were found after death in the viscera of the animals experimented upon.—'Lancet,' 1879 and 1880.—Ed.

five months, five gentlemen having been attacked in turn. In the London Fever Hospital, during the years 1869 and 1870, twenty-seven of the nurses and officers, and five patients admitted for other diseases, contracted relapsing fever. In 1870 a nurse from the Fever Hospital was transferred to St Mark's Hospital for fistula; and about the same time a wardmaid from St Mark's paid a visit to the Fever Hospital. These two women both fell ill at St Mark's, and gave the fever to five other persons there. Two striking instances were recorded by Mr Reid, of Glasgow, in 1843, to show that the spread of the disease was not due merely to a widely diffused atmospheric influence. (1) At Dalmarnock Colliery there was a large building consisting of three stories, which was entered by separate stairs and contained forty families; some Irish people brought relapsing fever with them into a single apartment on the uppermost flat, whereupon it spread from room to room, and ultimately descending a stair, attacked twenty-two individuals in the space of two months. (2) Into a house of two apartments, in which eleven human beings were lodged, a person from a neighbouring village introduced the fever; every person there fell ill, but all the occupants of the next house, separated only by a brick partition, escaped, although they were almost as closely crowded together. That the disease may be transported to a distance by infected clothes seems to have been proved by two cases recorded by Dr Parry, of Philadelphia, in 1870; and in 1843 it was noticed in Edinburgh that a large number of laundry-women contracted relapsing fever, although they had no communication with the sick, beyond having to do with their clothes and bedding. Litten has related an instance from the epidemic at Breslau in 1872–3: a mason, who himself remained well, but who slept in a street in which the disease was prevailing, carried the contagion to his mother, who lived where no case had before appeared.

Special interest, indeed, attaches to this particular epidemic, because it is the only one which has as yet been traced out in detail with the object of referring each case if possible to infection from some previous one. It is impossible for me here to cite the facts in reference to it which are to be found in the 'Deutsches Archiv' for 1874, but I may say that they strongly support the view that this is the mode in which the disease really spreads, even where, as must often be the case, this cannot be directly demonstrated. On the assumption that the contagious principle consists in the spirochæta, or in its germs, we must suppose that they are given off either with the breath or from the skin. In all probability they are received into the bodies of other individuals with the inhaled air. Litten has clearly shown that at Breslau they were not conveyed in drinking-water. And in this connection it is perhaps worthy of mention that Dr Carter has ascertained that the spirochæta does not exist in the contents of the thoracic duct.

The cases observed by Litten in Silesia seem to have belonged to the very poor classes, with the exception of the medical men who took the fever in the hospital; and English writers have generally been disposed to state as the cardinal fact in the ætiology of this disease its occurrence in those who have been in a state of starvation. It is the *famine fever* of Ireland. Murchison cites instance after instance to prove that those who had suffered from it in England and in Scotland have, with certain exceptions, been in a state of extreme destitution, often reduced to live upon raw turnips, rotten apples, &c. When it occurred in Silesia in 1847, the inhabitants, in consequence of a succession of bad harvests, had been reduced to subsist on clover, grass, mushrooms, and the roots of trees. Carter states that it was brought to

Bombay in 1877 by the peasantry flocking into the city from famine-stricken districts of Western India. Murchison himself was of opinion that the specific poison of the disease might be generated *de novo* in the human body as the result of inanition. He cited the observations of Holland and of Donovan to prove that persons in a state of starvation suffer from delirium, stupor, and coma; that they may have a quick pulse, a dry tongue, flushing of the face, intolerance of light, and neuralgic pains over the body, and that they exhale putrid odours from the skin. He believed it to be a point in favour of such a view that tramps have often been found to bring the disease with them into London after sleeping by the roadside under hedges. Dr Carter also says that one of the earliest cases he saw in Bombay was that of a destitute lad who, while tramping up from the famine district of Poonah, was seized with fever in the upland country, and was barely able to crawl into the hospital on his arrival at Bombay. But it is obvious that in none of these instances could the reception of the disease by infection be positively disproved. A further argument adduced by Murchison is the fact that the relief of extreme destitution in districts where relapsing fever was prevailing has repeatedly been followed by the subsidence of the epidemic. But this may only show that starvation renders individuals more susceptible to the contagion, not that it is capable of generating it; and the same remark applies to the statement that the disease is never seen in private practice among persons belonging to the higher classes, who do not come into direct contact with the poor.

Age and sex.—Relapsing fever prevails at various seasons of the year. It may attack persons of all ages, from five months old to seventy-five years, but the majority of cases are between the ages of fifteen and twenty-five. More males than females suffer from it, the reason probably being that a larger number of beggars, hawkers, and vagrants belong to one sex than to the other.

Diagnosis.—It is only at the commencement of relapsing fever that its diagnosis in ordinary cases presents any difficulty. Murchison remarks that during the first two days one may be scarcely able to distinguish it from smallpox; but in consequence of the frequency with which it prevails epidemically at the same time with typhus, the early differentiation of these two diseases, so unlike one another in the danger which they entail, is of special importance in practice. I have already insisted on the greater rapidity with which the temperature and the pulse rise in the less severe form of fever. How useful for diagnostic purposes the detection of the spirochætæ may be, has been shown by Dr Carter at Bombay, where there was often difficulty in distinguishing the effects of malaria from the more continued varieties of relapsing fever, corresponding with the "bilious typhoid" of German writers. This observer states that so multiform were the phases and degrees of the spirillum fever that about 25 per cent. of his cases could properly be termed irregular.

Prognosis.—Most patients attacked by relapsing fever recover. It is far less fatal than typhus or enterica. Out of 441 cases collected by Murchison from the records of the London Fever Hospital only 11 proved fatal, and in the great Scottish epidemic of 1843 of 6300 cases only 260 died. On a basis of over 14,000 cases he estimates the mortality at less than 5 per

cent. As usual in epidemics the worst cases come first, and the disease becomes gradually milder. Age is the most serious adverse circumstance as in other fevers. Hæmorrhage, petechial rash, delirium and scanty urine are the most grave symptoms.

Treatment.—This is not so successful as might be hoped, for we have as yet no means of preventing the relapse. With this object quinine and arsenic have been freely employed both in England and in Germany, but in vain. At Breslau carbolic acid, and in America the sulphites, the hypo-sulphites, and the preparations of chlorine have been used with no better result. It remains to be seen whether the salicylate of soda, or the sulphocarbolates, or any of the drugs which have recently been introduced into the practice of "antiseptic surgery," may be more effectual. According to Murchison patients often find great comfort from frequent sponging of the surface with cold or tepid water, and from cold affusion to the head. An emetic at the first onset of the disease affords much relief to the severe pains in the hypochondriac regions. Throughout the febrile attacks castor-oil, or some other mild aperient, should be given when necessary. Murchison also recommended the systematic administration of diuretics, by which he believed that it was often possible to prevent one of the chief dangers of the disease, the supervention of uræmic poisoning. He directed that from one to two drachms of nitrate of potass, one drachm of dilute nitric acid, and half a drachm of tincture of digitalis should be taken in divided doses in the twenty-four hours. He allowed rather a liberal supply of food, and to persons beyond middle age he administered stimulants about the period of the crisis, especially when he suspected that the heart was weak. He often found it necessary to give opium for the relief of headache, or of the muscular and arthritic pains which cause so much distress, but states that the hydrate of chloral proved a useful substitute for it. Lebert recommends frictions with a liniment of chloroform and oil for temporary alleviation of the pains in the limbs.

On account of the contagiousness of relapsing fever, it is important that patients should be taken to a hospital as early as possible. They should always be placed in wards set apart for the purpose, since Litten seems to have shown that the accumulation of a large number of cases together neither augments the mortality of the disease nor increases to an appreciable extent the danger of its spreading to medical attendants and nurses.

ENTERIC FEVER*

History of the recognition of Enteric Fever and of its distinction from Typhus—Nomenclature—Characteristic lesions—Incubation—Clinical course and symptoms—Recovery—Abortive cases—Fatal cases—Complications—Sequelæ—Relapses—Diagnosis—Ætiology—A contagious disease—Always produced from a previous case—Methods of contagion: by air, water, &c. Illustrative cases—Predisposing causes: soil-water, climate, age—Prognosis—Treatment—Food, stimulants, drugs—Cold baths and antipyretics—Treatment of other symptoms—Convalescence—FEBRICULA.

ALMOST at the beginning of the present century, the study of morbid anatomy in Paris was rewarded by the discovery that in the bodies of those who died of fever in that city the intestines were inflamed and ulcerated. The true bearings of such observations were not, however, at once fully appreciated. Broussais and others maintained that a "gastro-entérite" was the essential cause of "fever." Presently the suggestion was made by Serres and Petit that the intestinal lesions were specific, and resulted from the introduction of a poison into the system, and that they were of an eruptive nature, like the pustules of smallpox. Bretonneau, of Tours, pointed out, in 1826–29, that they were specially localised in the solitary and the agminated follicles of the ileum. He endeavoured to introduce the name "dothiénentérie" (δοθιήν, a pustule, and ἔντερον, intestine) for the fever in which they occurred. This clumsy term was used by his pupil Trousseau, but it never met with general acceptance. Unfortunately, much greater success attended a proposal made by Louis, in 1829, to designate it "fièvre typhoïde." This proposal was, at least in England, universally adopted; and it greatly impeded a right understanding of the matter. Up to that time, all the French physicians who had written upon the intestinal affection had assumed that the same lesion would be found in every form of the disease known by the various names of typhus, putrid fever, jail fever, &c. But when the morbid anatomy of patients who had died of these varieties of fever came to be investigated in Scotland and in England, the bowels were often found to be healthy. For a time the difference was supposed to be accidental, or of little consequence. But gradually it became apparent that two distinct diseases were being confounded with one another. The credit of indicating the clinical distinctions between them is divided among a number of observers; Gerhard, of Philadelphia (1836), Lombard, of Geneva (1836), Shattuck, of Boston (1839), and particularly A. P. Stewart, of London (1840), deserve mention.† Louis, in the second edition of his work, published in 1841, expressly announced that his "fièvre typhoïde" and the typhus of English writers were very different. There were, however, many

* *Synonyms.*—Typhoid Fever, Gastric, Pythogenic, Infantile remittent Fever, Slow nervous Fever of Huxham (1739), Typhus abdominalis or Ileo-typhus.

† Dr Stewart, from observations made in Glasgow and in Paris (1836—1839), concluded that the differences between the two diseases were "so marked as to defy misconception, and to enable the observer to form with the utmost precision the diagnosis of the nature of the disease and the lesions to be revealed by dissection."

who maintained the opposite view, that the two forms were identical. The ultimate issue of the controversy still appeared uncertain when, in 1849–51, Jenner published a series of researches, based upon observations made at the London Fever Hospital. The peculiar value of his papers lay in the proofs which he brought forward of the fact that the different forms of fever owe their origin to distinct specific causes. During two or three years he investigated with great care every instance in which more than one patient was brought from the same house; and he found that the later cases invariably corresponded in character with the first one, even where both kinds of fever were prevalent in London at the time. All subsequent writers, whose opinions carry weight, have adopted the doctrine for which Jenner contended, and there no longer remains any doubt about the matter.*

It has naturally been a point of great interest to inquire whether the presence of enteric fever can be recognised in the description given by medical writers in former centuries; and the answer is decidedly in the affirmative.† In John Hunter's Museum there still are two preparations showing the characteristic intestinal lesions; and a well-marked case was dissected by Morgagni. It appears, too, that the disease is identical with the "slow, nervous fever" which Gilchrist, of Dumfries, described in 1734, and which Huxham, in 1738, distinguished from "putrid malignant" fever. In works of a still earlier date, it must probably be looked for rather among the "remittent fevers," attributed to the same cause as that of ague. The mistake of confounding enteric fever with malarial fevers was commonly committed in the marshy districts of England up to a very recent period; and in India they are even now separated with difficulty. These facts would in themselves suggest that in former times, when the marsh-miasm was much more widely spread than at present, the distinction would fail to be recognised; and an instance in point is probably to be found in the writings of Baglivi (1696) and Lancisi (1718), who recorded cases of fever at Rome in which the intestines were ulcerated, but which they identified with the "hemitritæus," *i.e.* semitertian, a form of ague mentioned by Galen.

Name.—Within the last few years, Louis' name of "typhoid fever" has in this country been generally discarded. It is an unfortunate term, since the object in giving a title to a disease really distinct from typhus, but liable to be mistaken for it, is to mark the difference between them, rather than the resemblance. But, further, the very same word has long been fitly used to designate a group of symptoms, consisting of stupor with muttering delirium, a dry brown tongue, sordes, &c., which are really *typhoid*, since they are like those which are seen in typhus. The two meanings necessarily led to much confusion. The term *enteric fever*, or *Enterica*, as Dr Wilks proposes, has therefore been substituted, and it appears to me to be very suitable, as indicating the fact that it is characteristic of the disease to be attended with intestinal lesions, though not, indeed, always with intestinal symptoms. Murchison proposed as an alternative name "pythogenic fever;" but this has met with very little support, and it involves, as I believe, an erroneous ætiological theory.

* Dr Wilks ('Guy's Hosp. Rep.,' 1855 and 1856), Dr Peacock ('Med. Times and Gaz.,' xiii, 1856), Sir Thomas Watson ('Lectures,' 4th ed., 1857), Dr Murchison ('Continued Fevers of Great Britain,' 1862).

† An interesting example of the possibility of interpreting historical records by the light of modern science is afforded by an admirable essay in which Dr Norman Moore has satisfactorily proved, from the memoranda of Dr Mayerne, physician to James I, that the disease of which Henry, Prince of Wales, died in 1612, was enteric fever.—Ed.

Characteristic lesions.—No other acute specific disease is accompanied by a definite series of internal lesions, which, being peculiar to it, constitute a certain test of its presence in all cases that end fatally. It is on account of the importance of these lesions, and also because I wish to mark the distinction which is created by them between enteric fever and the various diseases with which it is most closely allied, that I describe them, before entering upon the clinical symptoms and course of the fever.

The most important lesions are situated in *Peyer's patches and in the solitary follicles* of the small and large intestines. These first become injected and swollen, so that they project further above the level of the surrounding mucous membrane than is naturally the case, even in children. As a rule, they rise to the height of one or two lines only; but in some exceptional cases, according to Hoffmann, the patches become as much as three eighths of an inch thick. In these structures, Louis and other French writers have described two separate forms of affection under the names of *plaques molles* and *plaques dures;* the difference between them is, however, merely one of degree, the hard ones being those in which the change is most intense and most rapidly developed. After a few days, the redness passes off, and the diseased follicles become grey and even white. So marked is sometimes their whiteness, that they have been compared to a thin layer of a soft medullary or encephaloid new growth spread out beneath the mucous membrane. The microscope shows that there is a very abundant formation of new cells, which are considerably larger than leucocytes, have large nuclei, and often exhibit signs of multiplying by fission. These cells, besides distending the lymph-follicles, infiltrate the intervening fibrous septa, and may even spread into the mucous membrane above, and into the interstices of the muscular coat and subserous tissue beneath. I have never seen them forming grey granules on the peritoneal surface, as is described by Hoffmann. But it is not uncommon for adjacent Peyer's patches to become fused together by an extension of the morbid growth, which thus may affect the whole circumference of the lower part of the ileum for some inches above the valve.

The next occurrence is generally ulceration. This seems sometimes to begin as an abrasion of the surface of the diseased solitary follicles or patches, and gradually to extend through their substance. But much more often the whole of the infiltrated tissue, or at least a large part of it, dies *en masse.* It then forms a soft, shreddy, flocculent slough which is of a bright ochrey-yellow colour, apparently from imbibition of bile pigment from fæcal matter. Presently the slough is detached, either entire or in fragments. There is then exposed the floor of an ulcer, in which the transverse muscular fibres of the intestine are often plainly visible, and which is extremely thin and translucent when held up to the light. The muscular fibres themselves may undergo destruction to a greater or less extent, so as to lay bare the subserous tissue, to the imminent risk of perforation into the peritoneal cavity. The edges of the ulcer are thin, soft, and rounded; and when examined under water they are seen to be slightly undermined. Its form at first corresponds with that of the lymphatic organ in which it took its origin; if in a solitary follicle, it is small and rounded; if in a Peyer's patch, it is usually elongated in the direction of the axis of the intestine. In the latter case, too, its position is always remote from the line of attachment of the mesentery. In these respects, as well as in the characters of their floors and edges, the ulcers of enteric fever differ from tubercular ulcers. They never

seem to show any signs of spreading by gradual destruction of their edges. And yet, at advanced stages of the disease, it not seldom happens that they are found to be elongated transversely to the axis of the intestine, their form being in fact the reverse of that which generally characterises them. At length, their floors become covered with a thin grey layer of granulation-tissue, and they heal by the gradual growth of mucous membrane from their edges towards their centres. I find, in our records of autopsies at Guy's Hospital, five cases in which death has occurred from some other disease within a few months after recovery from enteric fever; in two of them the patches were of a blackish colour, with slaty margins; but in the others the cicatrices seem to have been white; and in one of them it was only on very close examination that a deviation from the normal appearance, consisting in a slight unevenness of surface, could be recognised. There is never any thickening or puckering of the affected tissues after this disease, so that it cannot lead to narrowing of the bowel.

Enteric fever by no means affects all the lymph-glands of the intestines equally. In a large number of cases the solitary follicles entirely escape; in many others those of the ileum are alone attacked; and when those of the large bowel suffer, it is often only in the cæcum, or in the ascending colon; in a very few instances the morbid change extends even to the rectum. It seems, in fact, to spread from the ileo-cæcal valve as from a starting-point. Precisely the same thing is observed with Peyer's patches; the highest of them are very seldom, if ever, affected. As a rule, the lesion is limited to patches within two or three feet of the valve, and sometimes it does not reach beyond a few inches. In very exceptional instances the agminated follicles remain altogether free, and the solitary follicles bear the whole brunt of the disease. This apparent caprice of distribution in different cases cannot but suggest the inquiry whether there may not be some in which no glands suffer at all, so that, if the nature of the disease were otherwise doubtful, the criterion afforded by morbid anatomy would fail. The nearest approach to this, with which I am acquainted, is afforded by a specimen which I showed at one of the meetings of the Pathological Society in 1875. The only lesions in the intestine were the following:—"One ill-defined purplish-red patch, of about the size of a shilling, situated a foot above the valve; and a little higher up another patch, presenting similar characters, except that in its centre there was a darker spot the size of a pea, with a breach of surface, visible only when it was examined under water." I think it is by no means unlikely that in mild cases of enteric fever, such as could never prove fatal except by some accident, the intestinal lesions are often very slight, and may possibly in rare cases be altogether absent.

Again, it seldom or never happens that at an autopsy all the diseased glands in a case of enteric fever are found in the same state. Those close to the valve are almost always the farthest advanced, and very often every stage is present, from clean ulcers below, through ulcers containing scattered shreds of slough, to those in which sloughs have just been formed; and from these, again, to patches which are but slightly excoriated on the surface or only swollen. Sometimes, indeed, the progression is not perfectly regular, a patch less affected being seen at a lower point than one which is more affected. Most pathologists think that these differences depend upon the fact that the follicles are attacked, not simultaneously, but in succession. Dr Moxon, however, is of opinion that the morbid change is of less severity, rather than of later date, in the higher patches. He thinks that

the greater tendency to destruction of those which lie at the extreme lower end of the ileum is due to the irritating contact of the intestinal contents, held back by the action of the valve upon them. Such a view, however, seems to me to be contradicted by the details of the differences which are observed, as I have stated them above, and I shall presently point out that in some of the more protracted cases of enteric fever there can be little doubt that the glands are affected successively and at considerable intervals of time.

But the swollen glands, instead of sloughing or ulcerating, sometimes entirely subside, as was long ago pointed out by Chomel and Louis. The original idea was that the morbid material in the follicles underwent softening, and that they ruptured so as to allow it to escape into the channel of the intestine. Hoffmann still says that this often occurs, and that the follicles become stained in consequence of there being at the same time a little hæmorrhage, so that the Peyer's patches acquire a dotted appearance, or exhibit, as the French term it, the *état pointillé*. I thought that this condition, which has also been compared to a shaven beard, had been proved to be at any rate not peculiar to enteric fever, seeing that it is not uncommon in those who have died from other causes and at all ages. But Hoffmann is still disposed to maintain that its origin is always in a former attack of the disease, which may have occurred so long ago as to have been forgotten. Its frequency in the bodies of those who have lived in Basle, where enteric fever is very prevalent, has led him to speak of it in joke as the *légitimation* for residence in that town. However this may be, there seems to be no doubt that subsidence of the swollen glands often takes place in a very different manner, namely, by disintegration of the cell growth in the follicles, and by its undergoing absorption like an inflammatory exudation. Among cases which end in recovery it is perhaps not uncommon for all the patches and all the solitary glands which are affected to take this course, but when death occurs at an advanced stage it very rarely happens that some are not found ulcerated. I have only met with one instance of the kind; the patient died on the twenty-first day, and the patches were found swollen, raised, and reddened, but marked by irregular depressed lines and spaces so that they had a reticulated appearance. They were not "medullary" in character as would probably have been the case if the affection had been early; there was no trace of sloughing or ulceration. I preserved the specimen in spirit for the museum, but after a few weeks it showed nothing. Chomel described *plaques à surface reticulée* as a special modification of the enteric lesion, but since, as Hoffmann points out, reticulation indicates that the septa between the follicles are more swollen than the follicles themselves, and since the morbid change begins in the follicles and affects the septa secondarily, such a condition can hardly occur except as the result of their subsidence. As a rule, absorption is found to have taken place in certain patches only, or even in parts of them, while elsewhere there are sloughs or ulcers. Thus in 1862 Dr Wilks examined a case in which, while the solitary follicles had each a depression in its centre, some patches appeared to be in a state of retrogression, parts of them only being slightly raised and nodular.

It is a matter of some practical importance to determine as accurately as possible the dates at which, reckoning from the commencement of the disease, one may expect to meet with the several stages of the intestinal lesions. According to Liebermeister, the first week is occupied by swelling and infiltration of the lymph-glands of the bowel; during the second week they either slough or begin to subside; in the third week any sloughs that may have

formed become detached, so that by the end of it all the ulcers have clean floors; during the fourth week they begin to heal, but the process is often not completed until a considerably later period. This statement is easy to remember, and for many cases it is probably accurate, but it certainly is not universally applicable in such a way that a pathologist should be able, from the appearances observed after death in a given case, to state positively the duration of the patient's illness. According to Trousseau the swelling of the intestinal glands does not begin to appear until the fourth or fifth day; according to Chomel and Louis not until the seventh or eighth day; but Bristowe and Hoffmann found them enlarged in cases fatal on the fifth and fourth days respectively; and Murchison relates one instance, that of a girl who died forty-seven hours after being suddenly attacked with vomiting and fever, in which the solitary follicles were of the size of hemp-seeds or split peas, Peyer's patches being also similarly affected. But it seems to me very doubtful whether the disease did not, in that case, begin earlier than its symptoms. The same remark applies to certain cases quoted by Murchison, in which sloughs with the usual characters have been found in patients who have been but a short time ill; and in 1871 a man died in Guy's Hospital, who had been definitely attacked twelve days before his death with shivering and aching pains in his limbs, so that he was obliged to go home, but his intestine contained extensive ulcers, all of which had clean floors except one, this having a few fragments of slough still adherent to it. Surely the lesion must have been in progress before his illness began. On the other hand, it is not uncommon, in cases fatal at the end of six or seven weeks of continuous fever, to find sloughs still adhering to several of the patches, while others are merely swollen and medullary looking. But probably the lesions are there of different dates, and have been developed in successive outbreaks at intervals of a fortnight or longer. In the case of a boy who had been ill for five weeks, and who died in Guy's Hospital on October 26th, 1879, Dr Goodhart describes the glands as fleshy-looking, and as just beginning to ulcerate, so that he would have supposed the disease to be at about the eighth or the ninth day. Doubtless some other glands had been affected from the first but had afterwards subsided.

Another lesion in enteric fever has its seat in the *mesenteric lymph-glands*. This does not appear to be absolutely constant; at least, I find, in the records of autopsies at Guy's, two cases, one of them fatal on the seventeenth day, in which the glands are said not to have been enlarged. It may be present at a very early period of the disease; thus, in Hoffmann's case, fatal on the fourth day, the glands were already of twice the normal size. That it results from the absorption by the lacteals of matters from the affected parts of the intestines is probable from the fact that in many instances the lower glands, corresponding with these parts, are alone attacked; but sometimes the glands in the upper portion of the mesentery become also involved, and even (as in a case which I inspected in 1878) glands in the portal fissure, close to the liver. As a rule, the enlargement is moderate, the diseased glands not being larger than hazel-nuts, but they may be as big as horse-chestnuts, projecting conspicuously from the mesentery. Their substance is white, or grey, or pinkish, with spots of hæmorrhage. Under the microscope they are found to contain large cells, some of which have two or more nuclei, like those in the intestinal follicles. Suppuration sometimes occurs, or partial sloughing, and under such circumstances a cheesy mass may result if the disease subsides. But I confess to feeling very doubtful whether Hoffmann

is right in saying that caseation, or the deposition of calcareous salts, may form part of the ordinary retrograde process.

Wagner and other German pathologists describe the frequent occurrence of *minute grey nodules*—sometimes visible to the naked eye, sometimes microscopic—in the substance of the liver and of the kidneys. Hoffmann noted their presence in thirty-eight among two hundred and fifty cases. The only instance that I know of in which a similar observation has been made at Guy's Hospital was by Dr Goodhart, in 1879, in the case of a girl, aged 17. If it stood alone, one might of course suppose that an acute tuberculosis was accidentally developing itself at the same time, just as occurred in a child who died a few weeks later of scarlatinal nephritis; but against such a view is the fact that the lungs in the fever case were free.

Incubation.—This period appears to vary within somewhat wide limits. The date at which a patient receives the poison can seldom be directly fixed. At Guildford in 1867 an epidemic was traced to the fact that contaminated water was supplied on a single day, the 17th of August; a large number of cases came under medical observation on the 3rd and the 4th of September, so that, allowance being made for the gradual development of the symptoms, the incubation probably lasted about eleven days. On the other hand, that it may be as short as two or four or eight days seems to have been shown by instances in which the disease has broken out within such periods after a definite exposure of the patient to sewer-gas, or after his having drunk contaminated milk, or after his having first arrived in an infected locality. With regard to cases in which the incubation has been supposed to be protracted there is great theoretical difficulty. Persons sometimes have not fallen ill until three weeks or longer after leaving a place in which there is reason to believe that they must have taken the fever; but the question is at what date the intestinal lesions began to develop themselves in such cases; and this I shall discuss almost directly. Some observers think that the incubation varies according to the mode in which the poison enters the body, being shorter when it is inhaled with the breath, longer when it is swallowed in drinking-water.

Course.—The commencement of enteric fever is generally slow and insidious. The patient feels depressed and weary, more and more so every day. He has headache, and giddiness, and pains in his back and limbs. He loses appetite; he may vomit or have diarrhœa; or, if he takes an aperient under the idea that it will set him right again, his bowels may remain relaxed. After about five or six days he becomes so ill that he has to give up work and take to his bed. In such a case the duration of his illness is reckoned from the day at which he first felt ailing. There are, however, some cases in which marked symptoms set in with chilliness, or even a rigor, so that medical assistance is at once sent for. If, then, on inquiry, it appears that the patient has been unwell for a few days previously, the disease is said to have had a *prodromal stage.* It is clear that we ought to count the whole duration of such cases. For I have already remarked that the lesions are often found after death to be further advanced than had been expected from the clinical history. It may even happen that malaise and other slight symptoms run on for two or three weeks, or longer still, and that afterwards a definite illness begins, the subsequent course of which is not shorter than usual. These instances create a difficulty from which Dr Irvine fairly

enough proposed to escape by assuming that the apparent primary attack is in reality a relapse, which according to circumstances may either be intercurrent or independent. But it must not be forgotten that such a theory involves the impossibility of proving that the incubation of the disease is ever prolonged, since one could not deny that in certain cases the real primary attack may be altogether latent. A case referred to at p. 167 shows that even the evidence afforded by an autopsy would not be conclusive. After all, however, the only practical point is that in every instance of this kind, whether the patient may or may not have been ailing before he fell ill, one must be prepared in case of death to find some of Peyer's patches presenting appearances corresponding with an advanced stage of the disease. In some very exceptional cases, enteric fever sets in with sudden maniacal delirium, so that the patient's removal to a lunatic asylum may appear to be necessary until the real nature of his case is manifested.

According to Wunderlich the *temperature* during the first three or four days rises in a zigzag fashion; from morning to evening there is an ascent of about 3° F., from evening to morning a fall of about 1°. The result therefore is that each evening the thermometer stands at about 2° higher than on the evening before, and he declared this course to be in itself tolerably decisive for diagnostic purposes, so that enteric fever might generally be excluded if the temperature rose during the first two days to 104°; if (at least in a patient under middle age) it did not rise between the fourth and the sixth day to 103·1°; if it stood at the same level on two successive mornings or on two successive evenings; or if it was ever lower than at the same hour on the previous day. He did, indeed, point out that this stage of the disease comparatively seldom comes under medical observation, but he seems to have overlooked the fact that the exceptional cases in which its onset is marked by definite symptoms are almost the only ones in which one is likely to have to use the thermometer during the first few days, so that they afford no proof that similar results would be obtained in cases beginning in the usual insidious manner. Jenner remarked to the Clinical Society in 1875 that in private practice there were found to be numerous exceptions to one of Wunderlich's rules, and this certainly accords with my more limited experience in the matter. In 1879 a patient in Guy's Hospital, convalescent from pleuritic effusion, was attacked with enteric fever. He first felt unwell on April 2nd, and his temperature immediately rose from 98·6° to 102·6°; next day he had rigors, on April 4th there were characteristic stools, on April 8th rose-spots appeared. In 1878 a girl was admitted who had been carefully watched from the third day of her illness by Dr Ingoldby; the morning and evening temperatures were on that day 103·4° and 105·2° respectively, on the fourth day they were 103·4° and 104·2°, on the fifth day 103·4° and 102·3°, on the sixth 103·5° and 104·2°.

As the disease advances the patient's state slowly becomes more and more serious. He generally grows weaker from day to day, his mental faculties are more obscured, he manifests less concern about his own condition. Yet the maximum temperature to be attained in the whole course of the fever is often registered by the fourth, the sixth, or the eighth evening. From that time the thermometer indicates only trifling variations during the next ten or fourteen days; in the morning it generally stands one or two degrees lower than in the evening. The skin may either be dry or moist. Profuse perspirations are by no means infrequent, especially at night; they may be accompanied by an abundant eruption of sudamina, especially towards the

end of the second and in the third week, and then again lead at a still later period to desquamation of the cuticle of the chest and abdomen, the importance of which lies only in the fact that its cause may be misunderstood.

The *aspect* of a patient with enteric fever is in general that of languor and weariness; the face is not dusky nor the aspect stupid as in typhus. There is usually pallor; with a circumscribed pink flush on one or both cheeks, especially towards the latter part of the day or when food or stimulant has been recently given.

The *pulse* is not always very rapid; its beats are generally from 100 to 110 per minute, but sometimes they remain at the normal rate, or are even less frequent. Murchison lays stress on the liability to variations in the pulse-rate at different periods of the day, in correspondence with the fluctuations of the temperature or independently of them. Changes of posture also affect it much more than in health, but the most important peculiarity of the pulse is its soft, compressible character, with a dichrotism which may be so marked that an unskilled observer may actually count it at twice the frequency of the heart's beats. These features are well shown by the sphygmograph.

The *breathing* is more or less quickened. In many cases bronchitis is present from an early period, but otherwise the respirations are not quickened in the same ratio as the pulse until hypostatic congestion begins to set in.

The *spleen* becomes enlarged in the course of the first week, and by the end of the second week it is often two or three times its normal size. Sometimes its edge can be felt below the ribs, but in most cases its position can be mapped out only by percussion. Indeed, I for my part think that even this method of detecting enlargement of the spleen often fails, and that as a symptom it is of far less clinical value than most writers state. With resonant lungs and a full tumid abdomen, I believe that the organ may be many ounces heavier than natural, without there being any appreciable percussion-dulness over it. Nor is it a fact that the spleen is always found after death to be swollen, even in young subjects who have succumbed when the disease is at its highest. I have notes of instances in which it weighed only four, five, or six ounces. Its substance, as a rule, is much softened, but it is not unfrequently firm.

Epistaxis is of rather frequent occurrence, especially soon after the commencement of the disease. It may be so profuse as to destroy life.

At about the end of the first week there appears the *rose-rash*, which is the one symptom of enteric fever that is almost if not quite pathognomonic. This is not always the earliest cutaneous affection observed in the disease, for it is sometimes preceded by a diffused scarlatinoid eruption which comes out two or three days sooner, and which may be attended with a slight sore-throat so that it has led to a mistaken diagnosis in several instances. The rose-rash itself is, of all rashes, generally the least conspicuous; to an untrained observer the idea seems absurd of attaching great significance to the presence of small red spots hardly larger than pins'-heads, the whole number of which may not exceed ten or twenty. Yet, although a case which I shall briefly relate further on seems to show that similar spots may very rarely be seen in at least one other disease, miliary tuberculosis of the lungs, one is, I think, justified in regarding them, when well-marked, as practically conclusive of the presence of enteric fever. But I quite agree with Murchison that it is often impossible to say of individual spots whether they are really rose-spots or what, for want of a better name, he terms "ordinary pimples."

The regions in which they are most often looked for are the lower part of the chest and the front and sides of the abdomen; but the same author speaks of having often found them on the back when there were none anywhere else. In exceptional cases they are scattered thickly over the whole of the trunk, and even upon the face and the limbs. The date at which they first appear is usually between the seventh and the twelfth day, sometimes as early as the fifth or sixth day from the commencement of symptoms, sometimes not before the fourteenth or even the twentieth day. Liebermeister speaks with hesitation as to their ever being absent throughout the whole course of the disease; but it is certain that, in children especially, one fails to observe them in many cases in which careful search is made every day. They can not only be seen but felt, as rounded, smooth, solid papules; their colour, which is pink or rose-red, disappears beneath the pressure of the finger; they never become petechial; after death they are invisible. In some exceptional cases it is said that their summits are vesicular. One of their most important characters is that they appear in successive crops, so that although the duration of the rash as a whole may be from one to three weeks, each individual spot never remains for more than three, four, or five days, sometimes not longer even than twenty-four hours. It has sometimes been noticed that they have come out in large numbers after a warm bath or that they have first shown themselves on a part that had been reddened by mustard.

Next in importance, as helping to distinguish enteric fever from other acute specific diseases with which it may be confounded, are certain symptoms referable to the digestive organs. The *tongue*, even at first, while it is covered with a white fur, has commonly a bright-red tip and bright-red edges. In the course of the second week it often becomes clean, and its whole surface may then be of the same vivid colour, and smooth as though it had been glazed. It sometimes remains moist, sometimes becomes dry; and deep painful transverse fissures often form in it.

I need hardly mention anorexia and thirst, which are common to this and to almost every other febrile complaint; nor is much stress to be laid upon nausea and vomiting, although they are very often complained of at the commencement. But the occurrence of *diarrhœa* is of considerable diagnostic significance. Sometimes, indeed, it is not present at any period of the disease. The proportion of such cases probably varies in different places and in different years, but Murchison, as the result of his experience from 1862 to 1873, places it at one fifth. And in many other instances the bowels cease to be relaxed when the patient has been ill for a few days, or they first begin to do so during the third or the fourth week. Louis stated, and Murchison has confirmed it, that, as a rule, the severity of enteric fever is proportionate to the urgency and persistence of the diarrhœa. But it is to be observed that there is no necessary relation between the symptom in question and the extent of the intestinal lesions in this disease. It was thought by Addison that diarrhœa was more constantly present when the colon was ulcerated than when the morbid change was confined to the glands of the ileum;* but if such a rule exists, it is certainly not without exceptions. The number of the evacuations is generally about four to six in the twenty-four hours; but sometimes it is much more. The stools have an appearance which has been compared with that of pea-soup, and which, although not uncommon in other intestinal affections, is probably seen in no general disease except enteric fever. Their colour is yellowish, almost like that of yellow ochre. Addison

* The same fact was observed by the late Mr Busk at the Dreadnought Hospital.—Ed.

used to lay stress upon their being almost identical with the normal contents of the small intestine, and he supposed that they were hurried on through the large bowel and discharged without having undergone the usual changes there. Albumen is present in considerable quantity in typhoid stools. The reaction is alkaline, and there are numerous crystals of the "triple phosphate. Blood may often be recognised microscopically or in the form of small clots even when the patient has been ill less than a week, so that ulceration is not likely to have begun. Of abundant intestinal hæmorrhage, as a serious complication, I shall speak further on. I am under the impression that Dr John Harley first pointed out the importance in all doubtful cases of searching during the third week for fragments of the sloughs from Peyer's patches. He advises that the stools should be strained through a net, and that the matters caught upon it should be washed and then examined by a lens; flocculent shreds may thus be obtained, in which the remains of intestinal follicles can be plainly recognised. In 1873 a man died in Guy's Hospital, who two days before his death passed a slough an inch and a half long, in which muscular fibres were discerned; at the autopsy the corresponding ulcer was found to be two inches in length and the peritoneum was exposed in its floor.

Another sign of intestinal disturbance is, in some cases, the production on pressure (which must be very cautiously exerted) of a gurgling sound in the right iliac fossa. There may also be more or less tenderness in this region, and the patient may complain of pain there or in other parts of the abdomen. During the second week of the disease the bowels generally become distended with gas, so that the abdomen assumes a rounded form. Sometimes the distension is extreme, constituting what is termed "meteorism." This is a grave symptom. Murchison says that the colon is the chief seat of the accumulation, and that the bulging is therefore at the sides rather than in front.

The *urine* is at first scanty and high coloured, and may remain so throughout the whole course of the disease, but after a time it may become copious, pale, and of a low specific gravity. Towards the end of the third week it often contains albumen in small, and sometimes in large, quantity; there may even be hæmaturia.

In some mild cases of enteric fever, *cerebral symptoms* are almost absent. In the course of the second week the headache subsides, and the patient may seem to have but little the matter with him. There is, indeed, no doubt that before the thermometer came into use persons were often wrongly allowed, and even persuaded, to leave their beds with the disease still upon them. There is not always very much muscular prostration. Murchison says that forty-four out of one hundred patients under his care were all always able to sit up, and to get out to the night-chair. But in the third week, or even earlier, many do become altogether helpless and lie upon the back, unable even to turn over to either side. In such cases the hands and the tongue are tremulous; there is sometimes, but rarely, inability to protrude the tongue. Jenner believes that a disproportionate intensity of tremor, as compared with other nervous symptoms, is of diagnostic and prognostic significance, pointing to the presence of deep ulceration of the intestine, such as is likely to lead to perforation or to dangerous hæmorrhage, and a similar statement was made by Murchison. In some rare cases the limbs or the trunk become rigid, or the back of the neck is retracted, or there is strabismus, or spasm of the pharynx, or trismus. Murchison's large experience yielded him only six instances of the occurrence of epileptiform convulsions. There may be all

degrees of delirium in this disease, from the slightest rambling or moaning during sleep up to the most violent maniacal excitement, with the patient screaming or shouting for hours together. Liebermeister remarks that one may sometimes fail to appreciate the extent to which the mental powers are enfeebled; a man may answer questions as to his actual condition clearly, but when asked how long he has been ill, what is the day of the week, what the month, what the year, he may be altogether unable to reply. Active noisy delirium (which is more common in enteric fever than in typhus) is always a dangerous symptom. A condition resembling delirium tremens is comparatively infrequent, even in persons who have been intemperate. Sometimes the patient unexpectedly jumps out of bed, and may throw himself out of the window. Or, as Gairdner observes, he may lie for several days perfectly still and quiet, apparently understanding everything that is said or done, but unable to articulate intelligible replies. The *pupils* in enteric fever are almost always dilated, as Jenner first remarked; but Murchison observes that where there is complete unconsciousness they may be as narrowly contracted as in any case of typhus.

Resolution.—When recovery is to take place the fever sometimes, though very rarely, subsides by a crisis. A medical friend of mine, in whom the temperature from morning to evening had been ranging from 101° to 103° with almost absolute regularity, was found, on what was calculated to be about the twentieth evening, to have a temperature of only 101·1°; next morning it was 100°, in the evening 99·3°; on the twenty-second morning it was 98·3°, and for several days afterwards it remained slightly below normal. But the rule is that at some period of the disease, generally towards the end of the third week, the morning temperatures begin to fall, while the evening temperatures rise to the same level as before. In other words, on a daily chart, a series of exceedingly acute zigzags is formed. After a few days, these become still more exaggerated, there being perhaps a difference of 4° or 6° between the temperatures of a single day; but the absolute height of the evening rise now begins to decline in its turn, so that each day it is about 1·5° or 2° lower than on the previous day; and thus, towards the end of the fourth week, a temperature is reached which is normal or slightly below normal. This *intermittent* type of pyrexia at the termination of the disease is so characteristic that I have known a correct diagnosis based upon it alone, in the case of a child about whose earlier symptoms no information was attainable; but, on the other hand, there are not a few instances in which the fever runs on for four, five, or even six weeks without any definite change. In a woman who was under treatment at Guy's Hospital in 1874 the temperature at the expiration of six weeks was still 103·8°, then it fell suddenly, and within two days became normal. It is in such cases that, if death occurs after several weeks' illness, one finds all stages of the lesions in Peyer's patches and solitary glands down to early swelling. I therefore do not concur in the statement which has been made that fever protracted for more than four weeks is generally due either to some complication or to non-cicatrisation of intestinal ulcers formed in the early period of the disease. Murchison himself says that he met with several instances in which rose-spots appeared as late as the thirty-fifth day, and one in which fresh spots were noted almost every day up to the sixtieth. Some observers think that the right view to take of such cases is to regard them as examples of relapse occurring "intercurrently;" that is, that

before the primary attack comes to an end, a second one begins and carries on the fever without apparent break.

During the stage of subsidence of the fever the pulse sometimes remains rather disproportionately high. This is often a period of much suffering to the patient who, as his mind becomes clear, complains more and more of weakness and of prostration and of the impossibility of finding a comfortable posture for his wasted body and limbs.

The loss of weight during an attack of enteric fever is often very considerable, amounting to twenty pounds or even more. Moreover, the microscope reveals changes in many of the tissues. Thus Zenker, in 1864, pointed out two kinds of degeneration in the fibres of voluntary *muscles;* some become granular, others undergo conversion into a glassy-looking substance, in which no striæ can be recognised, and which splits up transversely into discs. As he himself remarked, however, the same lesion occurs likewise in other febrile diseases, if sufficiently severe and protracted. It is especially marked in the adductors of the thighs and in the recti of the abdomen. Muscles so affected are said to be sometimes obviously altered in appearance, being of a yellowish-grey colour, but I have never been able to convince myself that such is the fact. They may during life be so much softened that they become extensively lacerated; thus, in 1870, a patient died of enteric fever in Guy's Hospital both of whose recti were torn across, and much blood was extravasated into them and into their sheath; and in another case, in 1866, the inner part of one rectus was found to be not only ruptured, but in a state of suppuration. Hoffmann has shown that similar forms of degeneration occur in the tongue, accounting perhaps for the tremor of its movements. The *liver*, again, has been described by many pathologists as undergoing a diffused change; it is soft, and on section it has a pale or "clayey" look. Hoffmann maintains that its cells become granular and break down. I doubt whether this can account for jaundice in those very exceptional cases in which it is present. It is worthy of remark that the occurrence in the gall-bladder of a colourless mucus, or of a muco-purulent fluid, after an acute illness, is not to be taken as showing that there has been a deficient secretion of bile in the liver. Probably it only indicates that for several days before death there was no storage of bile, in consequence of the very short intervals at which food was taken. The *kidneys* are often of a greyish-yellow colour and have their epithelium granular. Perhaps this causes the albuminuria which we have seen to be sometimes present. Another organ in which Hoffmann believed that he detected changes was the *brain*. He describes the nerve-cells in the great basal ganglia as loaded with brown and black pigment granules. Lastly, he found an enormous overgrowth of cells in the acini of the salivary *glands* and of the pancreas. He says that those structures feel unusually hard and dense.

In cases which recover, all these various lesions are doubtless repaired. The process of regeneration of muscular fibres has already been traced, and according to Hoffmann the liver-cells are reproduced by proliferation, of which there is evidence in the great excess of nuclei which these cells contain when death happens to occur at advanced stages of the disease.

The *convalescence* from enteric fever is slow in comparison with that from typhus, even when its course is uninterrupted by any accident such as will be presently described. Many weeks elapse after the subsidence of the pyrexia before the patient is fit to resume the active duties of life.

It will, I fear, have been by no means easy for the reader to take full possession of all the facts stated in the previous section with regard to cases of enteric fever which, although severe, yet terminate favourably. But the subject is by no means exhausted. I have still to describe (1) those forms of the disease which run an abortive course or are of exceptionally slight intensity, (2) the mode in which death is directly brought about, (3) a number of complications, some of which may themselves be fatal, (4) sequelæ, and (5) relapses.

1. *Slight and abortive cases.*—The symptoms are sometimes from first to last so slight that there is the greatest difficulty in persuading the patient that he is really ill, and in preventing him from going on with his usual work. Such cases are often seen in hospital out-patient practice in the second and third week, and the clumsy but expressive name of *ambulatory typhoid* has of late been adopted for them. Their duration may be as long as that of typical cases, or it may be slightly shorter. In many of them the affected intestinal glands probably subside without breach of surface, but that this is not always the case is shown by perforation sometimes occurring. When I come to speak of acute peritonitis I shall have to mention latent enteric fever among the very few causes that may give rise to it without warning.

Again, not a few cases of enteric fever attended with well-marked symptoms subside before the end of the third week. About some of them, which run on for sixteen or eighteen days, very little need be said. In all probability the intestinal lesions are comparatively slight, and the swelling of Peyer's patches or solitary glands very generally disappears by absorption and without ulceration. But since it is only by the rarest accident that their condition can be verified by an autopsy, I think it would be extremely unsafe to hazard an absolute statement about it, especially as in other forms of the disease there are so many exceptions to the rule of there being a correspondence between clinical symptoms and morbid changes.

Cases which terminate before the sixteenth day, however, require special study, because, their real nature being overlooked, they are very apt to be set down as examples of a simple febricula, or of a non-specific gastric or intestinal catarrh. Yet it is unquestionable that such cases may depend upon infection with the poison of enteric fever, for they often form part of "house-epidemics." Jürgensen, for example, mentions an outbreak near Kiel, in which fourteen out of twenty persons had an abortive form of the disease. Moreover, in addition to malaise, headache, and anorexia, many cases present rose-spots, diarrhœa with characteristic stools, and enlargement of the spleen. The spots, indeed, are said to appear at an exceptionally early date, sometimes on the second, generally by the fourth or the fifth day. It is also to be noted that the onset of this variety of enteric fever appears to be particularly definite. In seventy-four of eighty-seven cases collected by Jürgensen it was sudden, and in forty-two it was attended with chilliness or even with a well-marked rigor.

The temperature generally rises quickly, and it may be 104° by the second or the third day. The maximum may be high; Liebermeister says that he has seen cases in which the thermometer rose to 106° in the axilla, and which yet subsided between the fifth and twelfth days, but in many instances the temperature at no time exceeds 101° or even 100°. Its subsidence is generally rapid, presenting scarcely any indications of the short zigzags seen in the commoner and more severe form of the disease, and is completed in from one to three days.

Lastly, enteric fever is sometimes not only of short duration, but also of very slight severity. Liebermeister even maintains that it may be altogether unattended with pyrexia, appearing as a slight non-febrile catarrh of the intestinal canal.

2. *Fatal cases.*—The proportion of deaths to recoveries in enteric fever varies in different cities and at different times to some extent, but perhaps less than might have been expected. Murchison speaks of having seen the disease much more fatal in one village than in another a few miles off, and he seems to have been struck by the fact that several members of the same family have sometimes died of it at long intervals and at distant places. But, so far as I know, one never sees malignant outbreaks of it, such as occur in the case of the exanthemata. At the London Fever Hospital the average death-rate from 1848 to 1870 was 17·26 per cent. of all cases admitted, the extreme figures in particular years being 28·42 and 12·82. For purposes of comparison, in estimating the effects of special modes of treatment, it would, however, be necessary to exclude all cases which ended fatally within forty-eight hours of admission; and this would reduce the average death-rate to 15·82 per cent. Statistics from various sources given by Murchison show a range of mortality from 11·37 to 32 per cent. At Basle, the average death-rate from 1843 to 1864 was 27·3 per cent.

The cause of death is sometimes the direct severity of the disease: and this may bring about a fatal issue in two different ways, namely, either through the brain by coma or through the heart by asthenia. Cases which are cut short by *coma* sometimes end within the first, and generally in the course of the second or by the beginning of the third week. It then scarcely ever happens that any special morbid appearances are discoverable in the brain or in its membranes; but one must be prepared for the discovery, in certain very exceptional cases, of meningitis. Cases in which the mode of death is by *asthenia* commonly run on until the third or the fourth week, or even longer still. The heart is then found to be soft and pale, and sometimes its right ventricle is dilated, or even its left ventricle. Its muscular fibres are commonly more or less granular, and may even have lost their transverse striation. In one instance Hoffmann detected in them a marked degree of the glassy change which I have already described as occurring in the fibres of voluntary muscles. Among 159 cases in which he examined the substance of the heart it was more or less profoundly altered in 103, but it is not stated whether in these cases there was a special preponderance of deaths by asthenia over those by coma.

3. *Complications.*—In a very considerable number of cases death is brought about indirectly, by affections which can only be regarded as complications. Such affections are not all of them necessarily fatal, and they require to be somewhat fully described; they are more numerous and varied than in any other acute specific disease.

Foremost among these complications come certain *abdominal* affections arising out of the intestinal lesions. Indeed, it is here that the clinical importance of the lesions in question is chiefly shown; for I have already commented upon the small part which they play in the production of the regular symptoms of the disease.

One complication is severe *hæmorrhage* from the bowels. Its frequency is difficult to estimate, because more or less blood may generally be found in the evacuations, if it is looked for from day to day. But Murchison says that in 3·77 per cent. of his cases it occurred to the extent of more than six

ounces. The colour of the blood may be either bright red or dark, and it may be fluid or partially clotted; if it has been retained for a time in the intestinal canal it is dark brown like chocolate, or almost black. When it is passed during the second week, it must be due to a general oozing from the swollen Peyer's patches, unless indeed the real date of the commencement of the disease should have been earlier than was supposed. But at later periods it often comes from an artery exposed during the separation of the sloughs. I have more than once seen one particular ulcer deeply blood-stained or with a clot attached to its floor; and Jenner, in a case of his, injected water into the superior mesenteric artery, and found that it escaped from one of the ulcers. In other instances, however, after four or six weeks of illness, I have found a number of ulcers all equally reddened, or the whole mucous membrane of the last two feet of the ileum intensely congested, soft, and swollen. Probably intestinal hæmorrhage is sometimes an indication of a scorbutic condition; I have known it accompanied by epistaxis, and by the appearance of purpuric spots upon the legs.

When the amount of blood lost is large, the patient exhibits the usual symptoms of such an occurrence, and becomes blanched and cold. The temperature of even the internal parts is lowered by perhaps two degrees, a point of some importance, as it may afford the earliest indication of what has occurred, if none of the blood should for a time be discharged per anum.

Liebermeister says that in such cases the part of the bowel which contains the blood may be dull on percussion. According to him hæmorrhage also diminishes the rapidity of the pulse, and is often attended with a marked alleviation of the cerebral symptoms. But all these effects are transitory; by the end of twenty-four hours the fever is as high as before or even higher.

There has been a remarkable difference of opinion with regard to the influence of intestinal hæmorrhage upon the course of the disease. Graves, and after him Trousseau, declared that it was not unfavourable, but probably the real basis of their opinion was the fact that a good many patients recover from it, or, in other words, that it is not so often directly fatal as might have been anticipated. For the statistics of Murchison and Liebermeister show conclusively that the death-rate among cases in which this complication occurs is far higher than the average death-rate of the disease.

The rejoinder may, however, be made that the cases in question are altogether of greater severity than average cases, independently of their being attended with hæmorrhage. As Murchison remarks, the deep ulceration which opens an artery is very likely to extend still further and to perforate the peritoneum; and thus it is well established that hæmorrhage, when it subsides, is often followed after a few days by a fatal peritonitis. In connection with this point, however, it is worthy of notice that hæmorrhage appears never to afford the earliest clinical indications of an enteric fever hitherto latent, as is often the case with perforation.

But even if we admit the force of the rejoinder, it by no means tends to support the dictum of Graves and of Trousseau, and one can hardly doubt that a considerable loss of blood in enteric fever must impair the patient's power of resisting the disease and favour the occurrence of cardiac failure. Thus Murchison says that he has repeatedly seen patients who had been doing well die unexpectedly of syncope a few hours after a copious bleeding. A case in point occurred at Guy's Hospital in 1879; the patient, a child aged six, went on favourably for a week after the hæmorrhage and then

suddenly expired. Dr Goodhart found considerable dilatation and some degree of fatty degeneration of the left ventricle of the heart. But the change after all was perhaps only such as we have seen to be of very frequent occurrence in uncomplicated cases; I doubt whether one can say more about it, than that the hæmorrhage probably intensified it, and gave to it a special clinical significance.

The other chief abdominal complication of enteric fever is *peritonitis*, which is generally dependent upon perforation of the bowel. Its frequency is very great, especially in England. Thus, whereas Liebermeister gives it as the cause of death in 8 per cent. of fatal cases of the disease, Murchison found it present in nearly 20 per cent. of fatal cases, and in no fewer than 3 per cent. of all his cases, including those which ended in recovery. All writers are agreed that it more often occurs in males than in females, and in seventy-three instances of it, observed by Murchison, the proportion was as fifty-one to twenty-two. When there is an actual perforation, the aperture is sometimes exceedingly small and rounded, but in other cases, as Dr Bristowe pointed out in vol. xi of the 'Pathological Transactions,' it is linear, showing that it was due to laceration; and stress must be laid upon the fact that its occurrence is sometimes directly traceable to disturbance of the bowel during defæcation, by the administration of an enema, in the act of vomiting, or in sitting up to take food. Or it may perhaps be due to irritation from solid fæcal matters, or to the presence of pent-up gas. Again, in certain cases, the gangrenous process affecting a Peyer's patch extends directly through the whole thickness of the bowel, and then a large slough may drop out, leaving a hole of considerable size. One may even find a number of such holes at different spots. Or the mere spreading of a putrid inflammation to the serous surface may light up a peritonitis without any actual perforation taking place. The distinction is said by Liebermeister to be sometimes manifest during life from the fact that when gas escapes freely it allows the liver to fall backwards from being in contact with the ribs, so that the percussion-note in the right hypochondriac region becomes tympanitic. Sometimes, too, a large quantity of fæcal matter is extravasated, and round worms may make their way outwards into the peritoneal cavity. The seat of perforation is almost always in the lower end of the ileum within a foot or two of the valve. It is said to be sometimes in the cæcal appendix or in the colon, but I believe that no instance of either kind has occurred at Guy's Hospital within the last twenty-six years. The date at which peritonitis sets in is usually in the third, fourth, or fifth week. Cases have been recorded in which it has been as early as the eighth, ninth, eleventh, or twelfth day of the patient's illness, but in all probability the disease has then really been of longer duration. Perforation is often the cause of death in some cases in which there has been profuse diarrhœa, and in which all the symptoms have been well marked; but, as I shall hereafter have again to point out, it is sometimes the first sign that anything is amiss with the patient, the course of the disease having hitherto been altogether latent. So, also, it may occur at a very advanced stage, when two, three, or even four months have elapsed, and this may be the case not only when the fever has been unusually protracted, or when there has been a definite relapse, but even when convalescence has apparently been established, and when perfectly formed and healthy fæces have been passed. In two instances that have occurred at Guy's Hospital, each at the end of the sixth week, perforation has been distinctly traced to the ingestion of improper food. One patient

was seized with pain very soon after eating two raw apples, another while in the act of eating watercresses. The symptoms of perforation are identical with those of a sudden attack of peritonitis from whatever cause, but it is important to note that enteric fever yields even more than a due proportion of cases in which peritonitis sets in insidiously, and may even remain altogether unnoticed. In a case observed in 1864 at Guy's Hospital the chief sign of an unfavourable turn in the patient's illness was his sudden refusal to take food, after which he died in a few hours. Perforation and consequent peritonitis are often found at a *post-mortem* examination when they had not been suspected, the fatal issue having been attributed directly to the intensity of the primary disease. It has been much discussed whether recovery from this complication is possible. The doubt lies in the difficulty of determining whether there is an actual perforation in any given case in which peritonitis has set in, but Liebermeister says he has had four cases of recovery in which the abdominal cavity contained pus, and several instances have been recorded in which death having occurred from some other cause, a perforation has been found to have been sealed up by adhesions, or at least closed off from the general peritoneal cavity. In other cases a circumscribed abscess has formed, which has discharged itself either externally or into the bowel.

But acute peritonitis does not always start from an intestinal ulcer. It may be set up by sloughing of a swollen mesenteric gland, as in a case recorded by Jenner, in which recovery took place, so that the real nature of the complication would not have been known if the patient had not afterwards died of erysipelas. It has also sometimes been due to extension from an inflamed and ulcerated gall-bladder, or from a suppurating infarctus in the spleen.

Infarctions of the spleen are in fact not infrequently found after death from enteric fever. They are doubtless the result of plugging of branches of the splenic artery with portions of clot that had formed in and been washed away from the dilated and softened left cavities of the heart. In the same way *embolism* of one of the cerebral arteries, causing hemiplegia, with or without aphasia, may arise either during the course of the disease, or later on when convalescence has already occurred.

In two cases at Guy's Hospital suppurative *nephritis* has occurred as a complication of enteric fever, and has apparently been the cause of death. The inflammation probably ascended from the bladder as the result of retention of urine.*

Other complications of enteric fever have their seat in the respiratory organs. We have seen that a certain degree of *bronchitis* is almost always present, but sometimes it becomes so severe as to constitute a very important part of the disease. I do not, however, remember to have ever seen a case diagnosed as simple bronchitis which on an autopsy has proved to have been one of enteric fever. Not infrequently there is broncho-pneumonia, and lobar pneumonia is far more common than in typhus, and sometimes passes on to gangrene. Pleurisy, too, is often seen, and may lead to empyema.

Ulceration of the *larynx* is present in a considerable number of cases. Hoffmann observed it in twenty-eight cases out of two hundred and fifty. It has generally been set down among the primary lesions of enteric fever, but the absence of a specific cell-growth constitutes a distinction which seems

* In a case which proved fatal under my care in August, 1886, there was found acute parenchymatous nephritis after death.—Ed.

important. Probably the explanation of its occurrence lies in some subtle association in morbid tendencies between the larynx, the lungs, and the intestine, of which we have other examples in acute pneumonia and in phthisis. As a rule there is a sharply defined ulcer over the base of one or both of the arytænoid cartilages. I believe that such an affection very rarely gives rise to any symptoms, but sometimes there is hoarseness, or even aphonia. In some instances, however, the morbid action involves deeper structures, and it may even be attended with necrosis and exfoliation of the cartilage, as in a case which I inspected in 1879. Hoffmann once saw the cricoid cartilage almost completely denuded. The entrance of air may, under such circumstances, be greatly obstructed. When I come to describe laryngeal perichondritis in general, I shall have to discuss an opinion of Dittrich's, which would assign to this affection, when occurring in any other cases as well as in those of fever, a mode of origin analogous to that which produces bedsores. And as, according to Ziemssen, modern antipyretic treatment has much diminished the frequency of laryngeal complications in enteric fever, it seems fair to conclude that the pyrexia is in some way concerned in producing them. In another of Hoffmann's cases tracheotomy was rendered necessary by the formation of a polypoid blood-clot, which hung down into the interior of the larynx. Or a laryngeal ulcer may endanger life by eating its way deeply into the connective tissue. In a case that occurred many years ago to Dr Wilks subcutaneous emphysema arose from this cause; the patient was a boy aged twelve. A similar instance, in a girl four years old, has since been recorded by Ziemssen. In his report of this case it is specially stated that the seat of the ulcer, which led down to the necrosed cricoid and arytænoid cartilages, was below the cords, and as the child had had much bronchitis there can be no doubt that the cough had forced air through the ulcer into the connective tissue. In two cases I have seen sloughs in the larynx of a bright yellow colour, exactly like that which in the intestine is attributed to the imbibition of bile. In one of them there were two linear ulcers, situated one on each side of the epiglottis, a position which is described by Hoffmann as frequent.

Sometimes a thin pellicle of lymph has been found lining both the interior of the larynx and the epiglottis. This brings one to the consideration of cases in which a membranous layer resembling that which characterises diphtheria has been observed as a complication of enteric fever. Whether the specific poison of diphtheria has then been in operation it is difficult to say, but Murchison relates an instance in which fluids escaped from the nostrils when the patient attempted to swallow them, exactly as in that disease.

Another complication of enteric fever is *parotitis*. This, however, is much less frequent than in typhus, although Hoffmann regards it as only an exaggeration of the morbid change which he finds so often present in the former disease. It sometimes affects one side, sometimes both. It may either subside after a week or two, or it may lead to suppuration, with extensive brawny induration and purulent infiltration of the tissues of the side of the neck as far as the sterno-mastoid muscle.

I have already remarked that in some very rare cases *meningitis* is believed to occur as a complication of enteric fever.

A point of some interest is that, as Liebermeister observes, the occurrence of enteric fever often leads to the reopening of fistulous openings which have healed up, with consequent *necrosis* of extensive portions of bone. In

patients who have been suffering from soft chancres he has seen wide-spreading gangrene under the influence of an attack of fever. In two cases at Guy's Hospital an ordinary gonorrhœa has led to sloughing of the penis or of the scrotum under similar circumstances.

4. *Sequelæ.*—In certain cases, affections which are described as complications of enteric fever do not begin until the patient is already convalescent. It is therefore impossible to draw a fixed line between them and the sequelæ of the disease, which themselves may set in before it has come to an end. What really justifies the distinction and renders it useful in practice, is that the latter often run on for many weeks, and obviously constitute the sole obstacle to the restoration of health.

This is the case with *bedsores*, which are apt to form over the sacrum and hips unless great care is taken to prevent them, and which sometimes destroy life by exhaustion, or indirectly by setting up pyæmia.

Another sequela, is *thrombosis* of the femoral vein, with the attendant liability to pulmonary embolism.

Again, in some cases enteric fever is followed by *marasmus*. Murchison says that there is not always a repugnance to food; the patient may eat well, but what he takes is not assimilated, and slight errors in diet cause flatulence or diarrhœa. Yet the temperature is normal or below normal, and if death occurs, perhaps after many months, nothing can be discovered except an unusually smooth appearance of the mucous membrane of the ileum, and a shrivelled condition of the mesenteric glands. In other instances, however, where diarrhœa has persisted after the subsidence of the fever, it has been supposed that the ulcers have remained unhealed, or (to employ the usual phrase) have passed into an "atonic" condition. But although Murchison supports this statement with the weight of his authority, I am not sure whether it rests on strict pathological evidence; Dr Wilks long ago taught me to doubt its accuracy, and I have never myself met with any example of it. I remember one case in which a patient was admitted into Guy's Hospital, whose sole complaint was pain in the right iliac fossa, which appeared to have been left behind by an attack of enteric fever; after a few weeks this pain subsided.

Writers generally speak of pulmonary *phthisis* as frequently occurring after enteric fever. Murchison seems to have thought that the correctness of this opinion might almost be taken for granted, on account of the long duration of the fever and the emaciation which it causes. But it is a very remarkable circumstance that, after searching the records of *post-mortem* examinations at Guy's Hospital, I have failed to find a single case in point. Can one suppose that the febrile disturbance which often accompanies the early stages of lung disease, where no physical signs may be discoverable, has been mistaken for enteric fever in the cases which have been so interpreted?

5. *Relapses.*—But the return of the temperature to normal is not necessarily followed by the restoration of the patient's health; enteric fever is apt to relapse. The frequency of such an occurrence seems to differ in different places: at Basle, Liebermeister met with it in 8·6 per cent. of 1743 cases, Murchison says that in the London Fever Hospital it was observed in 3 per cent. of 2591 cases; the figures given by other writers vary from 11 per cent. to 1·4 per cent. As a rule, there is an interval of some days between the termination of the first and the beginning of the second attack. Murchison states it as eleven days on an average; Liebermeister found that

among 111 cases it was less than four days in twenty-seven, from five to seven days in seventeeen, from eight to fourteen days in thirty-five, still longer in thirty-two. The late Dr Irvine, however, in a valuable paper in the 'Medical Times and Gazette' for 1879, maintained that the average interval is not longer than five days. He believed that a relapse sometimes begins before the original illness has come to an end. I have already remarked that this may explain those anomalous cases in which enteric fever seems to run on indefinitely for six or seven weeks or longer. It is not very uncommon for the termination of a relapse to be followed after a second interval by a second relapse, and this, again, may be succeeded by a third, and even by a fourth. There is scarcely a more important use of the thermometer in clinical practice than in the detection of such secondary attacks of fever. For it sometimes happens that the patient is unaware that anything is amiss with him, and yet to allow him to get about and to return to ordinary diet is to expose him to fearful risk. Thus at Guy's Hospital in 1876, a man was apparently going on favourably through convalescence, when on the twenty-ninth day his temperature was found to be 104·2°; he looked ill; but he declared that he did not feel so. A few days later he died of perforation of the intestine. In other instances the thermometer itself indicates but a very moderate degree of fever, ranging from 100° to 101° or 102°, but nevertheless taking a perfectly typical course. Dr Irvine suggested that in those cases in which a relapse has been supposed to occur many weeks after convalescence, there have really been one or more intervening attacks which escaped notice. The duration of a relapse is by most writers said to be generally shorter than that of the original attack. Murchison found it to be on an average sixteen days. Dr Irvine, however, maintains that it is typically twenty or twenty-one days. Cases in which it appears to be longer he explains by the hypothesis of an intercurrent second relapse, interrupting the middle of the first relapse, and subsequently running a regular course. According to this observer the temperature in a relapse generally rises pretty steadily, until on the fifth day it attains its maximum; it remains high until the ninth or the tenth day, when it undergoes a marked fall of from 2° to 6°; directly afterwards, however, it rises again, but it henceforth shows daily remissions, which at length bring it to an end.

It is now well ascertained that relapses of enteric fever are attended with a renewal of the intestinal lesions, although Trousseau maintained the contrary. We have had at least nine fatal cases at Guy's Hospital within the last twenty-two years, and in every one of them recent morbid changes have been found. As a rule, some Peyer's patches are in a state of early swelling, or have partly formed sloughs, while others show clean ulcers or cicatrices; but in one instance the floors of the patches are said to have been smooth and bare, while their edges showed the pink tumefaction of commencing disease. The clinical symptoms of a relapse may be identical with those of a primary attack. Rose-spots are said by Murchison to appear somewhat earlier; in twenty-two out of thirty-eight cases they were visible on the third, the fourth, or the fifth day. In all of Dr Irvine's cases the stools were characteristic in appearance. The patient is often delirious and insensible, so that, considering the debility resulting from his first illness, one is surprised that he does not more often succumb. As a fact, however, relapses are seldom fatal, except by some complication, such as perforation, from which recovery is practically impossible.

The cause of the liability of enteric fever to relapse is still imperfectly

understood, and has naturally been the subject of many speculations. It cannot be due to a fresh infection with the specific poison from without, for the patient is often in a hospital, far removed from the original source of his disease, and little exposed to other chances of acquiring it. Very often, the time at which he falls ill for the second time is when he has just begun to take solid food, or when he has once or twice been out of bed. But other instances cannot possibly be thus accounted for; and, at the best, such an apparent exciting cause can only be regarded as setting up the symptoms of a morbid process which would otherwise have remained latent; since, if the patient happens to die a few days later, the intestinal lesions are found to be so far advanced that they clearly must have begun before the obvious relapse set in. For instance, in 1876, a case ended fatally on the sixth day of relapse, and several of the Peyer's patches had already ochrey yellow centres. Such cases, indeed, seem to me to afford the key to the whole question. In August, 1861, a man was admitted into Guy's Hospital with bronchitis and emphysema. A month later he was attacked with enteric fever, which ran a regular course and from which he recovered, so as to be able to sit up. But early in October the weather became very cold, whereupon his chest symptoms underwent rapid aggravation, and in a few days he died of them. At the autopsy, the lower Peyer's patches were found to be roughened and flocculent, as if sloughs had separated from them; but high up in the ileum one or two patches were still somewhat raised, and several solitary glands showed an early stage of the affection. This case seems to prove that after an attack of enteric fever has to all appearance passed off, intestinal lesions may smoulder on, without giving rise to any symptoms. Dr Maclagan some years ago suggested that sloughs thrown off from the patches first diseased might perhaps infect other patches in their turn. Such a hypothesis is difficult either to prove or to disprove. But with the analogy of spirillum fever to guide us, we can hardly be wrong if we suppose that the actual relapse of enteric fever is due to a reinfection of the blood with a virus (doubtless consisting of specific organisms) derived from patches thus recently diseased.

Protection.—In connection with this question it may be observed that there is a marked immunity from a second attack of the disease among those who have once passed through it. Murchison alludes to observations made by Gendron and Piedvache, in which after an interval of many years a particular house or locality became for the second time the seat of an outbreak, whereupon it spared those who had previously suffered, but attacked almost everyone else. Murchison, however, had himself met with several exceptions to this rule, and he cites others.

Diagnosis.—The diagnosis of enteric fever may either be easy or difficult. In some cases a single examination of the patient may enable one to speak positively of the nature of his illness, especially in the second or third week; in some others, the most careful clinical observation, from day to day, may leave one to the last in a state of doubt.

At its commencement, the disease can never be determined with certainty, although very strong suspicions may be excited if there should have been other cases in the same house, or even in the same neighbourhood. The commonest thing, in the absence of such circumstances, is to mistake it for a "bilious attack;" and immense harm is often done by the aperient dose which inevitably follows. The fact that the onset of enteric fever is gene-

rally insidious is apt, when it begins suddenly, to suggest the idea that the case is rather one of typhus, of some exanthem, or of influenza. Liebermeister lays stress on the rarity of nasal catarrh and of sneezing at the beginning of enterica; and another point, which has long been known, is that it is very seldom attended with herpes about the lips. I have already mentioned cases of enteric fever that were at first set down to mania.

At about the end of the first week, that is, within two or three days of the time at which cases generally come under medical observation, a positive diagnosis can, as a rule, be given. It has already been remarked that the early roseola has sometimes been mistaken for scarlet fever, in spite of its not coming out until the fourth or the fifth day. Murchison notes that he has known a copious eruption of rose-spots attributed to smallpox. Even where no rose-spots appear one can often be tolerably confident of the nature of this disease from the course of the pyrexia, the presence of an enlarged spleen, and the characteristic appearance of the stools. But in most cases, one's opinion is in reality mainly founded upon negative considerations, and, as Leibermeister remarks, he who is sensible of the danger of committing errors is the most likely to avoid them.

The diseases which are liable to be mistaken for enteric fever without eruption, or for which it may be mistaken, may be divided into two groups.

First come certain general diseases. Of these the most important in many countries, although not in England, is *ague*. I have already more than once remarked upon the extreme difficulty of distinguishing the remittent forms of marsh-poisoning from enteric fever, whether in individual cases or even throughout a district, unless opportunities should arise for making autopsies. Another is *general miliary tuberculosis*, without marked symptoms indicating localisation in any particular organ. But although it is quite true that at an early period of a case it is often impossible to say whether a patient is entering upon one or the other of these diseases, I find from our *post-mortem* records at Guy's Hospital that of late years there have been very few, if any, fatal cases in which a correct diagnosis has not been made.* The most remarkable instance of persistent obscurity with which I am acquainted is one recorded by Senator in the 'Berlin klin. Wochenschrift' for 1881. A man, aged forty-two, was admitted into the Augusta Hospital of Berlin on October 25th, 1880. He had not been quite well since the beginning of September, but his illness had been more marked for about ten days before his admission. His pulse was 80 to 84. His temperature was 100·4°, and for some days it ranged from 101° in the morning to 103° in the evening very regularly. On October 29th the spleen was found to be distinctly enlarged, and on October 31st distinct rose-spots appeared, and fresh ones again on the following day. On November 1st and 2nd there was repeated epistaxis. The pulse, still 80 to the minute, was now plainly dicrotic. Signs of bronchial catarrh appeared and increased greatly up to November 11th. After November 2nd the range of the temperature became narrower, scarcely moving downwards below 101° in the morning, or upwards above 102° in the evening. The pulse remained between 80 and 100. On November 10th the patient was found to be slightly deaf. The bowels acted every day or every other day. On November 18th it is noted that there

* It has been my fate to supply this deficiency by a case in a boy of enteric fever, which was without diarrhœa or rash, and accompanied by severe bronchopneumonia. It ran a protracted course, with cyanosis and high irregular temperature, and I regarded it as tuberculosis and not enterica up to the time of death.—Ed.

was slight lividity. On November 21st suppuration of the left parotid occurred, with discharge of pus through Steno's duct. The diagnosis, about which up to this time there had been slight doubts, was now finally given in favour of enteric fever. Yet after the patient's death, which occurred on November 22nd, miliary tubercles were found in the lungs and in other organs, while the appearances characteristic of enteric fever were altogether wanting. The ophthalmoscope had been used with negative results. I think that a correct diagnosis of this case was impossible.

Another disease that may be mistaken for enteric fever is *pyæmia*, when its starting-point is some deep-seated affection, and when it affects, not joints, but internal organs. Murchison says that he saw several cases of pyæmia due to caries of the temporal bone, of which the course was very like that of enteric fever. At Guy's I know of two instances in which such a mistake was actually made. In each of them the source of the mischief was latent disease of the lumbar or the dorsal vertebræ, there being secondary abscesses in the lungs and the kidneys, and one in the heart. Murchison says that in his experience the variations of temperature are in pyæmia always much greater. *Trichiniasis* has generally been set down as enteric fever by those who are unacquainted with its peculiar symptoms; but with due care this error might perhaps be always avoided.

Secondly, a great many local diseases may be overlooked, and their effects attributed to enteric fever, if a positive diagnosis of it be rashly hazarded, in the absence of rose-spots.

Foremost among these is a cerebral affection, *tubercular meningitis;* but I reserve what is to be said with regard to this affection until I shall have described it. One very important point, which has been insisted on by Jenner, is that in enteric fever headache ceases before delirium begins; the two symptoms are not present simultaneously, unless indeed there actually is meningitis, as a complication. In the days when the clinical thermometer was not in use I have known enteric fever mistaken for hysteria; in a diabetic patient I have seen it set down as diabetic coma; and in a man who was the subject of lead-poisoning I have known uræmia, dependent upon granular disease of the kidneys, mistaken for enteric fever.

Among thoracic affections, *miliary tuberculosis of the lungs* must especially be borne in mind. I have already pointed out that enteric fever is often accompanied with bronchitis; and even when miliary tuberculosis produces marked thoracic symptoms it may be difficult to say whether they are not rather due to this complication of fever. The points of distinction I shall discuss when I have described the other disease. Formerly, *acute phthisis*, causing a rapidly spreading consolidation of one or of both lungs, was sometimes mistaken for typhoid fever; and a similar error was even made with regard to *simple pneumonia*, where there was no pain in the chest, nor cough, nor expectoration. And even now, although the routine use of the stethoscope keeps us from overlooking the presence of a pulmonary lesion, we may sometimes be in doubt whether it constitutes the disease from which the patient is suffering, or is merely a complication. *Ulcerative endocarditis* has often been set down as enteric fever; the discovery of a cardiac murmur should in most cases suffice to put one on the right scent with regard to it, but sometimes the heart-sounds remain normal.

Various abdominal affections are liable to be confounded at the bedside with enteric fever. Foremost among them is *tubercular peritonitis.* In the absence of the positive signs of this affection its diagnosis from enteric

fever may be impossible. I have seen more than one case of which the nature has still remained doubtful at the end of several weeks of illness. Again, two or three instances have occurred at Guy's Hospital in which *acute diffused inflammation of the colon*, generally of diphtheritic character, has been mistaken for enteric fever, there having been no symptoms of dysentery, so far as could afterwards be ascertained. Other diseases which have failed to betray their presence during life have been *typhlitis*, *perinephric abscess*, and *abscess of the liver* secondary to ulceration of the colon, or (in another case) to suppuration in the broad ligament of the uterus, with an opening into the intestine.

Finally, it is important to remember that some patients omit to complain of *affections of the genito-urinary organs* sufficiently severe to cause profound constitutional disturbance, and that others conceal them from mistaken modesty. Sir William Gull used in his lectures to speak of cases of extravasation of urine to which he had been called under the idea that they were enteric fever, there being stupor, muttering delirium, a dry brown tongue, &c. Some years ago I was asked to see a young lady at a school at Brompton who had been suffering for a day or two from febrile symptoms, with a very quick pulse. It was not until she had been sent home that she mentioned to her mother that she had severe pain in micturition, and that her private parts were swollen. There was then found to be an intense diphtheritic inflammation of the labia and nymphæ, and she very narrowly escaped with her life.

Ætiology.—Within the last few years our knowledge with regard to the ætiology of Enteric Fever has made great advances. We are not, indeed, yet acquainted with its actual exciting cause; but many of the chief details as to the way in which it is propagated have been positively determined, certain theories concerning its nature have been no less decisively negatived, and thus we can now limit somewhat narrowly the scope of future researches.

Origin of the disease.—It has sometimes been said to be an *endemic* malady, but although it may prevail in certain districts rather than others, and may even remain limited to them, there could be no greater mistake than to suppose that its diffusion is, like that of ague, independent of the movements of human beings and of their intercourse. I shall presently adduce numerous instances in which an outbreak has followed the entrance into a place of a patient suffering with this fever, although for months or years there had not been a single case there.

Among the most famous examples of such an occurrence are those recorded by Dr William Budd, of Bristol, in his well-known paper on the epidemic in and near North Tawton, Devonshire, during the autumn of 1839. Three persons left that village after having taken the fever. Two of them went to Morchard and gave it, one to his two children, the other to a friend, and he again to his two children and to his brother. The third went to Chaffcombe, seven miles off, where ten others were attacked in turn, and two of these carried the disease to fresh places, with the result that several more cases occurred at each of them.

Dr Budd tells this story as if direct infection from the sick to the healthy had been at work, at least in some instances. Thus the friend of the second patient at Morchard is described as having been called upon to assist in raising the sick man in bed, as having been overpowered by the smell from his body, and as having felt very unwell from that time. In 1875 Sir

William Jenner, in his Presidential Address to the Clinical Society, stated that he had twice known enteric fever contracted by students who diligently took temperatures before the registering thermometer was in use, so that they many times a day put their heads almost into the beds of patients suffering under the disease. In 1879 Dr Collie, of the Homerton Fever Hospital, expressed his belief that certain cases which were among the attendants at that institution were caused by direct infection, emanating either from the freshly-passed evacuations of patients or from their lungs or skins.

But striking as such occurrences naturally appear to the individual observer who watches their progress, there are the strongest possible grounds for rejecting this interpretation of them. Murchison tells us that during nine years, from 1861 to 1870, cases of enteric fever were treated in the same wards of the London Fever Hospital with various non-specific febrile complaints, to the number of 3555 of the former class, and 5144 of the latter. The same night-chairs were used by both sets of patients, and the employment of disinfectants was exceptional. Yet enteric fever was not contracted by one of those who were under treatment for other diseases. In the 'British Medical Journal' for 1879, Dr Shirley Murphy has brought down to 1878 the experience of this hospital in regard to the occurrence of enteric fever among the nurses and other attendants. During twenty-four years only nineteen persons engaged in the institution were attacked by it. "Of these, ten were in no way connected with the enteric-fever patients or the enteric-fever wards. Of the other nine, one was a laundry-maid, whose duties would bring her into contact with the soiled linen of the patients, but who otherwise was not in contact with them. Of the remaining eight there were special circumstances connected with drainage which would probably account for fever, leaving four for whose attacks there is no explanation given." In this time 5569 patients with enteric fever passed through the wards. Is it conceivable that if the disease were directly infectious there would not have been more numerous instances of its spreading? In other buildings one often cannot trace the origin of enteric fever to sewage emanations or to its other known causes, and the same thing must necessarily sometimes occur in hospitals. Some writers have brought forward instances in which two or more cases of enteric fever have arisen successively in the same house, as indicating that it is directly infectious; but much caution is required in drawing such a conclusion on account of the long duration of this disease, which allows abundant time for the production in other ways of new cases, while the original one is still running on. Murchison cites, but without attaching very much importance to them, a few instances in which nurses contracted the disease immediately after having been attending upon patients suffering from it, but most of them lose much of their force when it is remembered that the patients probably lay ill under the nurses' care for three or four weeks. It is obvious that the fact of a nurse taking enteric fever who is engaged with a patient labouring under this disease is not even presumptive evidence of direct infection, unless the patient is away from the place in which he himself became attacked. So again, the circumstance that several inmates of a house are affected in tolerably rapid succession proves nothing, unless the first case was introduced from elsewhere.

Dr William Budd, although he believed all the emanations from cases of enteric fever to be contagious, made it his chief object to show that the intestinal discharges were incomparably more virulent than anything else. He seems to have thought that an important argument in favour of such a

view could be based upon an analogy which he drew between the specific cutaneous eruption of a contagious fever and the lesions in this disease which affect Peyer's patches and the solitary follicles. To express this very relation, the word *enanthem* had been already coined by German writers as a correlative to the term *exanthem;* and the doctrine propounded by Dr Budd appears to have been previously taught at Munich by von Gietl. It is now widely adopted. But in order to account for the fact already stated, that nurses do not take the disease notwithstanding that they come frequently into contact with the stools of patients suffering from it, an additional theory has been promulgated: that fresh typhoid stools do not contain the poison, which is afterwards developed in them. We shall hereafter see that exactly the same thing is known to be true in the case of cholera; in that disease the "rice-water" discharges have been proved to be innocuous when first voided, and to become virulent subsequently. I may remark, in passing, that if the poison of enteric fever is specific, the theory of its being evolved outside the human body after an interval, necessarily implies that it must be a living organism, and not a mere chemical substance. Dr Cayley, in his 'Croonian Lectures' for 1880, expresses his belief that this change may occur within twelve hours; for in the Middlesex Hospital patients have apparently caught enteric fever from using closets in which pans were placed containing stools from other cases set apart for the inspection of the physicians. Probably the development of the poison may occur even in fæcal matters smeared upon linen or sheets, for washerwomen have often been observed to take the disease after washing the clothes and bedding of patients, having perhaps inhaled particles of the dried fæces, which had become detached and suspended in the air. Biermer, in one of the 'Clinical Lectures' published by Volkmann, says that he has met with several instances of this. Dr Cayley mentions two cases which occurred in patients already in the wards of the Middlesex Hospital, and which were traced to emanations from dried discharges upon the bedding of a typhoid patient in a neighbouring bed. Dr Murchison relates the case of a woman who brought to her house in Warbstowe, on the Cornish moors, the bedding of a sister who had died of enteric fever at Cardiff, in Wales. She remained free, but her sister, who was employed in hanging out the clothes, took the disease; and it spread from her as a centre.

Murchison, although he teaches that the stools of patients suffering under the disease are at first incapable of propagating it, and become so only when they have undergone a change, takes a very different view from that of Dr Budd. According to him, the change in question is not the development of a specific poison, but a decomposition, to which typhoid are more prone than healthy fæces, on account of their being alkaline and containing ammonia and triple phosphates in abundance.

It is almost a necessary part of his theory, that it should be possible for the intestinal discharges of persons who are not affected with enteric fever to be decomposed in a precisely similar manner, and so to give rise to the disease *de novo*. He was, indeed, so strongly convinced that this was of frequent occurrence, that he at one time proposed the name of *pythogenic fever* (πύθομαι=*putresco*) in place of the objectionable term "typhoid fever," which was then generally in vogue. Sir William Jenner also, in 1875, expressed his opinion that the weight of evidence was in favour of the development of the disease, in many instances, independently of any specific poison derived from previous cases. The main argument adduced by both these observers

is that it is so generally impossible, at the commencement of an outbreak, to trace any probable, or even possible, source from which the specific poison could have come. Jenner cited the case of a young lady who, being an invalid, had been confined to her own room in a detached villa, where she saw very few people, for some months, before she was attacked with enteric fever. A sewer-gas odour was detected, and when the flooring was taken up a crack was found in the soil-pipe of a water-closet on the floor on which she slept. This was no doubt the cause of the disease, but for about two years, during which she had lived in the house, no one who was at all likely to have been the subject of enteric fever had used the water-closet; and from the town drains it was cut off by new and efficient traps.

Murchison laid especial stress upon certain outbreaks of the disease in which it was traced to emanations from cesspools, or from choked-up sewers, having no communication with the drains. But the only one of his cases in which the attempt was made to show that the cesspool or sewer could not have recently had the specific poison introduced, in the fæces passed by someone suffering under mild enteric fever, was one which occurred at a school at Colchester. And even of this, all that is said is that "there were no other cases of fever, before or after, in the rest of the Union."

Before, however, we can determine the real value of such observations, we want to know how long the contagion of the disease is capable of remaining undestroyed in sewage. If it be a living organism, which may germinate and multiply outside the human body, there is apparently no reason why it should not survive, under favourable conditions, for an indefinite period. A case in point is related by von Gietl. A man, who had acquired enteric fever elsewhere, brought it to a village. His evacuations were buried in a dung heap. Some weeks later, five persons, engaged in removing some of the dung, were attacked by the disease; their discharges were sunk deep in the heap. At the end of nine months it was completely cleared out by two workmen, one of whom fell ill and died. In such a case as that related by Jenner, one can imagine that the typhoid poison might have been lurking in some stagnant corner of the water-closet or soil-pipe from the time when, perhaps many years previously, someone connected with a former proprietor of the house suffered from the disease. That no one should have been attacked in the interval is sufficiently explained by the fact that this young lady was the only person who always remained on the same floor of the house, breathing the infected air both by day and night; possibly, too, her being an invalid and being confined to her own room may have rendered her more susceptible. Or, again, what is more likely than that a living organism, if it constitutes the exciting cause of enteric fever, should sometimes remain for years in a dormant state, multiplying itself just sufficiently to escape extinction; and then that, under the accidental supervention of more favourable conditions, it should suddenly undergo an immense development? Such an interpretation seems to be the only one applicable to a fact which Murchison himself adduces; namely, that he has seen single cases of enteric fever arising in the same house again and again at intervals of a year or longer. Thus between 1849 and 1857 six cases were admitted from a certain house into the London Fever Hospital; one in June, 1849, one in October, 1851, one in February, 1854, one in November, 1855, one in November, 1856, one in July, 1857. It would surely be a remarkable coincidence that the disease should have been six times generated *de novo* in a single building during these eight years, or that its specific poison should have been six times

introduced from without. But if the poison was there all along, perhaps undergoing an excessive development from time to time when the conditions were especially favourable to it, we can never, I think, exclude the possibility of its presence in any cesspool, or sewer, or water-closet.

Again, there is the clearest proof that neither the inhalation of ordinary sewer-gas, nor the drinking of water impregnated with ordinary fæcal matters, sets up enteric fever. In an immense number of villages throughout England the water-supply is exceedingly impure; and both in villages and in towns the drainage is often as bad as it can possibly be. Yet for years the inhabitants of such places escape the disease, until a case is introduced from elsewhere, and then an outbreak at once occurs. It is probably only under such conditions as these, when the channels for the entrance of the poison of enteric fever into the human body are already prepared, that there are seen such distinct indications of its being virulently contagious, as were recorded by Dr Budd in the case of the outbreak at North Tawton. And, commonly, if the conditions are absent, no evil consequences follow the entrance of a case of enteric fever into a place. How frequent this is is shown by a statement of Murchison's that in private practice more than forty instances had come under his notice, in which persons came to a house ill with enteric fever; but that in two only out of the entire number was there any evidence that the disease spread further, and that one of these two was perhaps not really a case in point.

Transference.—The poison of enteric fever may be conveyed into the human body in various ways:

First, *it may be transported by currents of air.* Many instances have now been recorded in which the disease was caused by exhalations from drains, or sewers, or water-closets. The four following are mentioned by Murchison.

1. In 1857 six policemen were admitted into the London Fever Hospital from the Peckham police station. The men declared that they had often complained of dreadful odours in a room where they sat. On investigation, a cesspool was found under a passage adjoining the room, and covered only by flag-stones. The fever ceased when its cause was removed.

2. In 1862, at Chatham, nine persons out of twelve who occupied a newly-built house were attacked with enteric fever. The master had for weeks complained of a bad smell in the cupboard of his dressing-room. It turned out that between the syphon-pipe of the water-closet and the soil-pipe below, there was a gap of several inches, and there had been extensive leakage. After the defect was made good no fresh cases occurred.

3. In 1848 a formidable outbreak, of what appears certainly to have been enteric fever (although Sir Thomas Watson held that this was not the case), occurred in the School and Abbey Cloisters at Westminster. Its distribution followed the line of a foul and neglected sewer, into which the contents of several smaller cesspools had been pumped immediately before the outbreak began. It communicated by direct openings with the drains of every house in which the disease appeared, except one; and the boys from that house were in the habit of playing every day in a yard in which there were gully-holes leading from the sewer.

4. In August, 1879, twenty out of twenty-two boys at a school at Clapham were attacked with a disease which was believed to be typhoid fever; the only point adverse to this view was the rapidly fatal course which it took in two cases. Two days previously the boys had been watching the workmen

engaged in opening and cleaning out a drain at the back of the house, which gave off a most offensive effluvium.

Secondly, *it may be conveyed by drinking-water.* The propagation of the disease in this way is of immense importance, on account of the very large number of persons who may be affected by it, whereas the action of sewer gas, when carried through the air, is necessarily limited. In their details the outbreaks of enteric fever that have been traced to impure water vary greatly; no two, indeed, are exactly alike. I must briefly refer to a few, and it will be well to begin with those in which the conditions are least complicated. Such are the small epidemics that occur so frequently among the inhabitants of a village, who derive their water-supply directly from one or more surface wells, into which sewage finds its way through a porous soil.

1. At Wicken Bonant, in Essex, the disease prevailed in 1869, and Dr Buchanan investigated its origin for the Privy Council Office. He found that there was a broad division among the people in regard to the sources from which they obtained their water. One hundred and eighteen persons used private wells, and among them there was only one positive case of fever; eighty-eight drank the water of one well called the "parish well," and no fewer than forty of them were attacked. Now, this well was situated four or five paces distant from a brook-channel which ran through the place. At the upper end of the village the brook always contained water; but lower down the channel was dry during the greater part of the year, the water being carried beneath the surface in a stratum of gravel, to reappear as a stream at the bottom of the village. That there was a direct communication between it and the parish well was evident from the fact that in times of flood, when the channel was full, the water in the well ran to a corresponding height and became discoloured. On June 24th the first case of fever occurred in the person of a boy, who lived in a cottage about thirty-five yards above the well. He had much diarrhœa, and his stools were thrown, without being disinfected, into a privy which stood almost on the edge of the channel. At this very time the soil-water in the village was falling, after abundant rains which had taken place a month before, and pools of water were to be seen here and there in the channel. A month after the boy's illness the persons who made use of the well began to fall ill with the fever. Can it be doubted that the well-water had become impregnated with the specific poison? Previously, on May 30th, two cases of fever had been imported from London into a house, of which the sewer opened into the brook two hundred and fifty yards above the well. I think it is almost certain that the boy derived the disease from that source, although it is not clear why he was the first to suffer.

2. At Page Green, in the parish of Tottenham, a great many cases of enteric fever occurred in 1864 and 1865. Dr Seaton investigated the matter and found that whereas there was to some houses a supply of water from the works of the Local Board of Health, the occupants of many other houses drank water from shallow surface wells. In three instances, in consequence of the families having removed from the place, he could not learn from which source the drinking-water had been taken, but in all other cases, with the single exception of one child, it was ascertained that those who were attacked had used well-water. Some of them had had the water the Local Board distributed to their houses, but had been in the habit of borrowing water from their neighbours' wells, because it was bright and pleasant, whereas the other was hard, turbid, and red, from rust in the

pipes. When, however, the well-waters were analysed by Prof. W. A. Miller, he declared them to be quite unfit for dietetic purposes.

3. At Terling, in Essex, between the beginning of December, 1867, and the end of February, 1868, there occurred an epidemic of enteric fever, upon which Dr Thorne reported. It was of extraordinary severity: amongst a population of nine hundred persons, at least two hundred and sixty were attacked during the first two months; there were in all forty-one deaths, and so panic-stricken was the village, that it was necessary to discontinue the tolling of the church bell at deaths or funerals. Whether the disease was introduced from elsewhere could not be ascertained, for there had been isolated cases during previous years. But the extension of it was clearly traced to contamination of the drinking water by sewage. The cottages were supplied, singly or in groups, by shallow surface wells, sunk in a loose and porous gravel. Round about them, but at a higher level, there were numbers of manure heaps, cesspools, and privies, the ordure from which was often spread out for yards over adjacent fields. During the autumn the water in the wells had been unusually low; doubtless, therefore, the filth had accumulated in the soil; so that it was washed into the wells in very large quantities, when, towards the end of November, a sudden rise of water took place. Among seventy-one persons, living on the outskirts of Terling, who procured their water from ponds, only six cases of fever occurred.

It is often impossible to trace the source from which the drinking-water derived the poison, but we have seen that there are slight forms of enteric fever, the real nature of which is never suspected. Thus a person who seems to be suffering under a trivial ailment may introduce the disease. Dr Cayley cites the following three cases of which the origin was clear:

4. At Over Darwen the water pipes were leaky and the soil through which they passed was soaked at one spot by the sewage from one particular house. No harm resulted until a young lady with the fever was brought to this house from a distant place; within three weeks of her arrival the disease broke out and fifteen hundred persons were attacked.

5. At Calne a laundress occupied the middle one of three houses supplied by one well, into which the slops of her house leaked. She received the linen soiled by the discharges of a case of enteric fever, and after fourteen days cases occurred in all those houses.

6. At Nunney a number of houses got their water supply from a foul brook contaminated by the leakage of the cesspool of one of the houses, but no fever showed itself until a man with the fever came into that house from a distance. Then in about a fortnight it appeared in all the houses.

Still more interesting are certain outbreaks of enteric fever which have been traced to contamination of water supplied by pipes; and their importance is the greater, because similar occurrences are likely to be more frequent as water companies multiply.

7. In Caterham, during the fortnight which ended February 2nd, 1879, there occurred forty-seven cases of enteric fever; and, at the same time, no fewer than one hundred and thirty-two cases were observed at Redhill, eight miles distant. In each town the persons attacked were using the water furnished by the Caterham Waterworks Company, but considerable numbers of persons who derived their supply from other sources escaped altogether. On the other hand, cases occurred at the Earlswood Asylum and in other places which also used the Company's water. Now, this water is drawn from chalk wells more than 500 feet deep, and it had a deservedly high

reputation for wholesomeness. Every point in regard to its sources, storage, and distribution was carefully inquired into, but for a long time in vain. At last, however, the attention of Dr Thorne was drawn to the fact that in January, 1879, the Company had been constructing an adit, at a depth of 455 feet from one of their old wells, to a new bore which was then being sunk. A number of men had been employed upon this duty, and one of them, it was found, had been ill and had left work in the course of the month. He was sought out, and on inquiring it appeared clear that he had been suffering from a mild attack of enteric fever, which began on January 5th, and which he had probably acquired at Croydon, where he had spent December 25th and 26th. He had much diarrhœa, the bowels acting at least two or three times during each shift of eight or twelve hours, and in accordance with the usual practice under such circumstances he made use of the buckets by which the excavated chalk was being raised to the surface. He denied that he had ever relieved himself in the adit without waiting for a bucket, but it seems to be almost certain that in some way his fæces passed into the water of the well in which he was working and gave rise to the epidemic. The poison must have been diluted to an extraordinary degree.

In this almost infinitesimal subdivision of the contagion of enteric fever, no epidemic seems to approach that which occurred in August, 1872, at Lausen in the Valley of Ergolz in the Jura, at least if its origin was correctly interpreted. But I must confess that it seems incredible that the effects of a poison should persist after it had been thrown into a running stream, and after the water taken from the stream had been poured out over the ground, to make its way by subterranean channels into a distant spring!

8. At Guildford, in September, 1867, a severe epidemic of enteric fever occurred; within ten days 150 cases came under treatment, and the number reached 264 by the end of the month. A singular circumstance with regard to this outbreak was now observed, namely, that it was almost restricted to the higher parts of the town to which water from a new well had been distributed by engine power, after having been first stored in a reservoir. Certain exceptions, in school-children and others who resided where water derived from other sources was used, were easily explained by the fact that those persons spent their days in houses supplied by the high-service water. Now, it was known that this new well was fed not merely by percolation, but by a fissure in the chalk; and that fæcal matter might easily find its way into it was probable, from there being within ten feet of the well a sewer, into which water-closets were drained and cesspools and privies discharged their overflow. These facts were so striking that Dr Buchanan, who investigated the matter on the spot, was at first perplexed when he was told that from the beginning of August, in consequence of the engine having broken down, the high-service water had not been taken from this source at all but from another well, the old well, which also supplied the lower parts of the town where there was no fever. But, on further inquiry, it turned out that on one particular day, the 17th of August, the water-wheel which was used to charge the high-service mains being under repair, they were filled with some water which had been raised from the new well on or before the 1st of August and had in the meantime remained in the high-service reservoir. Subsequently the sewer above mentioned was ascertained to have been leaking in various places, so that the surrounding soil was a quagmire of dark-coloured, fetid slush, which made the men vomit who had to dig it out.

9. At Sherborne, in Dorsetshire, enteric fever became epidemic at the

beginning of the year 1873, and prevailed severely until the middle of April. Dr Bloxall, who went down to inquire into the matter, found reason to conclude that the drinking-water, which was delivered from a reservoir through pipes, and which was believed to be originally pure, became contaminated in a way which would not at first sight have been obvious. In December, 1872, and in January and February, 1873, the water was frequently shut off from the town at a point near the reservoir. Now, it was known that when the water was thus shut off a rush of air would take place *into* certain delivering pipes as soon as their taps were turned on; but many of the mouths of the pipes were situated in the pans of water-closets, consequently if a tap was broken, or if a person forgot to turn it off when he found that it gave exit to no water, the corresponding pipe might continuously suck up sewer gas, or even liquid excrement, supposing the water-closet pan to be full. Then, when water was again delivered, this would wash away whatever particles might have been deposited in the pipes and convey them to be drunk by the people of the town.

10. At Caius College, Cambridge, a local outbreak of the disease occurred in November, 1873, which was traced by Dr Buchanan, with very strong probability, to a precisely similar origin. Twelve out of fifteen cases in students at the college were among the sixty-three residents in Tree Court, a part of the building which had been erected only four years previously with every care as to sewers, drains, and water-pipes. Now, Tree Court had an independent water supply direct from a high-pressure main. This supply was intended to be constant, but there had in fact been a complete intermission of it on two occasions shortly before the outbreak. After such intermissions the water had been noticed to come in with a rush, like "soda-water," evidently in consequence of its having become mixed with air, which had been sucked up into the pipes. Within the Tree Court buildings there were two water-closets, one in the basement of the porter's lodge, the other on the first floor of one of the staircases. The tap of the lower one, or that over an adjoining sink, if left open during the intermission of water supply from the main, would have allowed water to drain from the whole pipe system of the court; that of the upper one would under such circumstances have permitted of the free entrance of air. This air, however, would have been mixed with sewer gas from an unventilated sewer in Trinity Street, which, at the very time under consideration, was receiving the excreta of patients ill with fever in other parts of the town. The effect of recharging the pipes with water must necessarily have been to distribute sewer gas in solution to every part of the building. It was, indeed, positively ascertained that not merely air, but water impregnated with fæcal matter, had been sucked up into the supply-pipe of the upper water-closet, for that pipe was lined with a brownish deposit, containing phosphoric acid and a large proportion of intermixed organic matter. The obvious method of preventing the occurrence of such outbreaks of fever as those at Caius College and at Sherborne is for the sanitary authorities, whenever the water supply is intermittent, to insist upon there being a proper service-cistern for each water-closet.

Thirdly, *milk may be contaminated with the poison of enteric fever.*—This is clearly shown by the following remarkable instances.

1. The first epidemic that was traced to such an origin occurred in Islington in 1870. It was investigated by Dr Ballard. Between July 3rd and September 10th the occupants of sixty-seven houses were attacked, one

hundred and sixty-seven individuals, of whom twenty-five died. It was a most remarkable circumstance that the district affected was included in a semicircle, with a radius of a quarter of a mile, drawn immediately on the north side of the line of the North London Railway from a centre almost upon this line. There was no fever in the area contained in the corresponding semicircle south of the railway, which here passes through a cutting. This, of course, at once suggested that human intercourse was in some way concerned in spreading the disease. The right clue was first hit upon by a lady, whose family was attacked; and a little inquiry convinced Dr Ballard that, far-fetched as such an idea had appeared, there was much probability in it. The milk vendor whose milk was suspected had himself fallen a victim to the epidemic, but his father, greatly to his credit, readily consented when applied to to give a list of the customers. It was then found that the dairy supplied one hundred and forty-two families, a very small proportion of those who lived within the semicircle. In no less than seventy among the hundred and forty-two families there had been cases of enteric fever. The way in which the disease picked out the customers of the dairy in particular streets and rows was most striking. In one long road and a street running from it the milkman supplied three families; two of them were affected. In a crescent with twenty-five houses he supplied four families; they were all attacked. In a new neighbourhood, where there were about seventy houses, he supplied four families; three had the disease. In a square with fifty-nine houses he supplied four families; all had it. On the other hand, there were scarcely any cases among those families who had invariably bought their milk from other sources. As might have been expected, women and children were attacked in much larger numbers than men, who drink comparatively little milk. The source of infection was traced, with much probability, to the water of an underground tank in the cowyard. When this was cleaned out, the woodwork in one corner of it was found to have broken away, leaving a gap, from which a rat-burrow passed into two old drains. Sewer gas had no doubt entered the tank along this channel, and it is quite possible that liquid sewage had taken the same course a few months before the outbreak when the ground had been disturbed to lay a drain-pipe for some neighbouring houses. It could not be ascertained that water from the tank had ever been used to dilute milk, but the pails were washed out with it, and some of it might have been left in one of them by accident. One of the customers had several times complained that the milk was poor, and that when kept it not merely became sour, but stank.

2. In the summer of 1872 an outbreak of enteric fever took place at Armley, near Leeds. This also was investigated by Dr. Ballard, who found that with a single exception all the early cases occurred in families supplied by a particular dairyman. After the first three weeks this rule was less strictly observed, the reason doubtless being that the disease then began to spread in other ways, since the privies, cesspools, and drains in the place were in the most neglected and offensive condition. That water from a pump on the premises of the dairyman had been the means of infecting th milk was rendered almost certain from the fact that, the handle of this pump having been chained up on July 10th, the fever abruptly ceased to appear in fresh families among the customers about a fortnight later. The way which the poison had entered the well beneath the pump was also satisfac torily made out. During the month of May the dairyman himself had bee ill with enteric fever; towards the end of the month there was a good dea

of rain, and this no doubt washed into the well fæcal matters which had escaped previously into the soil from the privy or from defective drains. On inspection of the well black matter was found to be oozing into it, and at the bottom there was a deposit of filth and mud which gave off bubbles of gas when disturbed. A point on which Dr Ballard relies as corroborating his conclusion that the early part of the outbreak was due to infection by milk, is that it was only during that period that multiple cases occurred in the same families.

3. In the summer of 1873 an outbreak of enteric fever occurred in St Marylebone, and in certain parts of St George's (Hanover Square) and of Paddington. It affected chiefly the households of well-to-do people, and among others the family of Dr Murchison, who quickly became convinced that the only probable mode of introduction of the disease into his house was by the milk supply. This was confirmed by a minute investigation made by Mr Netten Radcliffe and Mr Power. It was shown that nine tenths of the two hundred and forty-four cases to which the inquiry extended were in households which consumed milk from a particular service of a particular dairy. Certain ramifications of the same milk supply extended to the east end of Regent's Park, to Belsize Park (Hampstead), and to St Anne's (Soho), and in these districts also enteric fever occurred among the consumers. There was a special incidence of the disease upon women and children, and many striking instances are noted in which those members of a family were attacked who were in the habit of drinking milk, while others escaped who did not do so. The milk which appeared to convey the poison was a special kind, sold as "nursery milk," and taken from three or four cows, set apart for the purpose, at Chilton Grove Farm, in Buckinghamshire. Now, on the 8th of June the occupier of this farm had died in the fourth week of an attack of enteric fever. His evacuations, instead of being thrown into the common privy, were buried in an ash-heap outside the farm buildings. Subsequently, however, it turned out that this was the very worst thing that could have been done with them. For there was a well close by, the water of which was used for dairy purposes, although not for drinking or cooking, as it had been noticed to have a disagreeable taste. Excavations made for the purpose showed that there had been a line of soakage into the well, along the foundations of a wall, of the filth from a pigsty which formed a pool in immediate proximity to the ash-heap above mentioned. If due intervals are allowed for the gradual penetration of the matters containing the poison of enteric fever through the soil, and for the incubation of the disease, the date at which the outbreak in London began—during the last days of June and the first days of July—corresponds exactly with this theory of its origin.

Fourthly, *there is some reason to believe that meat may under certain circumstances convey the poison of enteric fever.* At Kloten, near Zürich, six hundred and sixty-eight persons were attacked in July, 1878, all of whom had partaken of some veal provided for a festival of the choral societies, and derived from various sources, but partly from two diseased calves. Huguenin thinks that one of these calves was actually suffering from an epizootic complaint equivalent to enteric fever. The liver and the brain of this animal fell to the share of persons who did not go to Kloten, and they also fell ill with the fever. A few other outbreaks, also attributed to diseased meat, are referred to by Dr Cayley. It is, however, doubtful whether the lower animals are liable to enteric fever. The so-called typhoid fever of pigs is now known to be an entirely different affection.

It must not be supposed that it is always, or even generally, possible to trace to their source isolated cases of enteric fever, especially when they occur in large towns like London. Probably there are many chances of infection, from sewer-gas in air, and from impurities in water, which no inquirer, however acute, could trace out. The number of cases admitted into the London Fever Hospital varies from year to year much less than might have been expected. Dr Cayley thinks that they are not dependent upon the presence of the contagion of the disease in water taken from the Thames. Although there can be no doubt that the poison frequently passes into the upper part of the river from the towns and villages on its banks, he thinks it is always destroyed by exposure to the air and by other agencies, among which vegetable life is probably one of the most important.

Soil-water.—Before leaving the question of the immediate causes of enteric fever, I must briefly mention a view held by certain German professors, according to which the fundamental point in the ætiology of the disease is its relation to periodic fluctuations in the level of the soil-water. In speaking of cholera I shall have to discuss a similar theory, based upon observations made by von Pettenkofer at Munich from the year 1856 onwards. It was, however, Buhl who applied these observations to enteric fever; he showed that when the soil-water in that city (as measured by the depth of water in the surface wells) is falling, the number of cases of enteric fever increases; when it is rising, the number of cases diminishes. That the facts really are so is generally admitted, but the interpretation which was at first put upon them is, I believe, rejected by everyone qualified to offer an opinion. It was that the falling of the soil-water enables air to penetrate more deeply into the ground than before, and so brings about changes in the organic matters there which result in the giving off of a poison that sets up the disease in persons exposed to it. But this is obviously inconsistent with everything that is known as to the way in which enteric fever spreads; and I cannot doubt that Liebermeister and Buchanan are right in supposing that the soil-water observations simply illustrate its communication by means of drinking-water. Not only is the water contained in surface wells generally more impure when the level of the soil-water is persistently low, but there is far less movement of it in a horizontal direction towards its natural outlets in brooks and streams, so that any noxious matters in it accumulate and acquire an increased virulence. It must also be added that in no other place except Munich has a fixed relation been found to obtain between the soil-water and the spread of enteric fever. In the case of particular epidemics, as notably at Terling in 1868, the disease has broken out with great severity precisely when the wells have been high.

It is certain that *climatic influences* greatly affect the prevalence of enteric fever. In the London Fever Hospital there have been far more admissions during dry and hot summers (*e. g.* 1865, 1866, 1868, 1870) than in damp and cold summers (*e. g.* 1860, 1872); but it is natural to take this in connection with the fact that each year there is an increase of the disease during the four months from August to November, while its frequency falls from March to May inclusive. Similar observations have been made in Berlin and in Basle, and there can be no doubt that the cause is the heat of summer on the one hand, and the cold of winter on the other hand, the effect being, however, not immediate, but retarded by two or three months.* Such conditions play

* At Munich the influence of season seems to be reversed, the maximum prevalence of enteric fever being in February; but Liebermeister suggests that this, after all, may be but the result of a still greater retardation of the same action which obtains elsewhere.

but a secondary and intermediate part in the ætiology of the disease; their effect is merely to favour, or to hinder, the operation of its real causes.

Age.—Certain circumstances remain to be stated, which affect the disposition of individuals to take enteric fever at particular times. Chief among these is age. The disease is far more frequent in persons between fifteen and thirty than in those who are either younger or older. In babies it is very uncommon, but in 1864 Murchison showed at the Pathological Society the intestine of an infant six months old who had been attacked at the same time with her mother. During childhood the liability to the disease increases from year to year, but I think it may be a question whether this does not depend upon an augmented exposure to its exciting cause. After the age of twenty the liability begins to decline, after thirty more rapidly, and beyond forty very few cases occur. However, I remember seeing enteric ulcers in the body of an old woman of seventy, examined by Dr Wilks; and some foreign observers have recorded instances in persons aged seventy-two, eighty-six, or even ninety. Liebermeister gives a tabular statement of the proportion of cases at varying ages at Basle, corrected according to the numbers of persons at the corresponding ages in the population generally. This, of course, diminishes the differences very considerably, and it seems to be possible that what remains of them may be due to the protection acquired by having already passed through the disease.

There does not appear to be any constant predominance of one sex over the other among patients suffering from enteric fever. Liebermeister maintains that it is more apt to attack strong and healthy persons than those who are sickly and delicate; and there seems to be a certain degree of immunity against it among women in pregnancy, after labour, and during lactation. Several French writers have declared that students, servants, and foreigners are especially liable to contract this disease when they first come to live in Paris; and Murchison has shown that more than 6 per cent. of the patients admitted into the London Fever Hospital have arrived in London within three months. This cannot merely indicate, as Trousseau suggests, that such persons are devoid of protection from their not having already passed through enteric fever. But, on the other hand, it may not necessarily prove that an acclimatisation occurs as the effect of longer residence; it may merely show that certain individuals are so very susceptible to the poison as necessarily to succumb as soon as they are exposed to it.

Prognosis.—In giving a prognosis in enteric fever one has to be guided not only by its symptoms, but also to some extent by the age and circumstances of the patient. The mortality does not, indeed, vary at different periods of life to the same extent as in typhus; Murchison's statistics show that until after the age of fifty-five it is almost uniform, at least among cases sufficiently grave to be admitted into the London Fever Hospital; and even in old people it is not much more than twice as high as in young adults. Enteric fever is especially dangerous in drunkards, in those who are very fat, in gouty persons, and in those who are affected with Bright's disease, heart disease, emphysema of the lungs, or phthisis. The death-rate is not augmented by a state of poverty. Murchison says that in private practice it is probably greater among the upper classes than among the very poor. In women the existence of pregnancy does not modify it to any great extent—abortion or miscarriage almost always occurs; but as a rule such patients recover.

The indications afforded by symptoms are exceedingly liable, when they seem to be favourable, to be unexpectedly upset by the supervention of some almost inevitably fatal complication. It is this which has led to the prevalent idea that a prognosis ought hardly to be hazarded at all in enteric fever, and one cannot too strongly insist on the fact that no case, however mild, can ever be declared to be altogether free from danger; but, if complications be left out of consideration, Liebermeister maintains that there is scarcely any other acute disease of which the course can be foretold at an early period with so high a degree of probability. The basis on which one's opinion must mainly rest is afforded by the thermometer. The death-rate is found to be greater in proportion as the maximum temperature—usually an evening temperature—is higher, and by the end of the first week the fever has generally reached its highest point, or nearly so. Still more important are observations on morning temperatures. The more marked the morning remissions the more favourable the case. A considerable fall on even a single occasion is a good sign, unless it is due to hæmorrhage or to some other complication. Fiedler found that the disease proved fatal to more than half of those patients in whom the temperature in the morning reached 105·4°, and to every one, with a single exception, in whom it reached 106·25°. Liebermeister seems to think that morning remissions are of value by interrupting the pernicious action of heat upon the tissues, and that they also show the fever to be less obstinate than if it were continuous. He goes on to say that it is a favourable omen if a cold bath lowers the temperature much and for a considerable time, and that a dose of quinine affords a sort of test as to the gravity of a case, this being less in proportion as the antipyretic action of the alkaloid is greater.

But individuals differ in the extent to which pyrexia is injurious to them, and the best measures of such differences are afforded by the state of the pulse and by the degree of disturbance of the cerebral functions. Liebermeister put together the cases that had occurred at the Basle Hospital, and shows that, the average mortality being 16 or 17 per cent., no fewer than 40 per cent. of those patients died when the pulse reached 120, and 80 per cent. of those in whom it exceeded 140. He also arranged his cases according to the intensity of the delirium and coma, and corresponding variations were found in the death-rate. Fatal complications occur far more frequently in severe cases than in those of which the course has been mild.

Treatment.—But the prognosis of enteric fever also depends upon the treatment to which the patient is subjected. It is a point of extreme importance that from the very commencement of the disease he should be kept at rest, and free both from bodily fatigue and mental strain. Not the least of the advantages which result from the habitual use of a clinical thermometer in daily practice is that the discovery of pyrexia in what might otherwise have been taken for a trifling disorder of the *primæ viæ* gives one grounds for insisting that no work shall be done until the temperature has fallen to the normal level. Men are apt to do themselves irreparable injury by struggling on day after day, even if they do not try to "walk off" their illness, or go away for change of air and a holiday, when they ought to be lying quietly in bed. The members of our own profession show no more judgment than others in this respect; Liebermeister has known medical men to go on seeing patients after having themselves noted their temperature at 104° on the previous evening. Sir William Jenner, speaking at Birmingham in 1879,

declared ('Lancet,' 1879, ii, p. 715) that some of the worst cases of enteric fever which he had ever seen appeared to owe their gravity to the circumstance that the patient had travelled, after having begun to feel ill, in order to reach his home. He added that he scarcely ever allowed a patient to be removed from the place where he was seized with the disease, if his residence was at a distance.

The sick room should be large and airy, so that it can be well ventilated without draughts. I do not know whether Liebermeister is right in asserting that a person whose temperature is much above normal is incapable of catching cold, and that therefore exposure to draught for a short time can do him no harm.

Food.—Another matter which is of great consequence is the diet. This throughout the whole course of the disease should consist of fluids only. The staple ought to be milk, of which two, three, or four pints may be supplied each day; it should be given in regular portions every two hours, or every hour, or even oftener; as the case goes on it soon becomes necessary to persevere with the feeding systematically throughout the night. If curdled milk is rejected by the stomach, or if it appears undigested in the evacuations, recourse should be had to Benger's *liquor pancreaticus;* the conversion of the casein into a peptone prevents all further trouble with it. This may very likely hereafter become a routine practice. Or lime-water may be added in the proportion of one part in three, or the milk may be diluted with barley water, which prevents a curd forming.

Other articles of food which may be allowed are animal broths, beef-tea, and calves'-foot jelly. It is generally said that beef-tea is apt to cause increased diarrhœa.* Jenner advises that some strong essence of vegetables should be added to the broths. He permits a little strained fruit juice to be taken, but does not let his patient suck grapes because of the difficulty of preventing the seeds from slipping down the throat; even particles of the pulp of fruits may cause irritation of the bowel.

Pure water may be taken without stint. Toast and water, or lemonade, or barley water with lemons may be preferred, or red or black currant jelly in water, or strained tamarind tea; but before many days the tongue is furred and dry, the patient can taste nothing, and cold water is the most grateful drink. Iced water generally quenches the thirst less. Ice to suck can do no harm, but I think it rather aggravates than relieves the parched mouth and throat. Painting the tongue with glycerine is a better remedy, and the lips should be well smeared with cold cream or vaseline so as if possible to prevent painful fissures.

Stimulants.—The rules for the administration of alcohol in enteric fever in which most experienced physicians are agreed are as follows, and the same apply to the treatment of typhus. Young and healthy patients may go through well-marked attacks with perfect safety when no stimulants are administered throughout. When, however, the pulse becomes very weak or irregular, when the first sound of the heart becomes faint, or when there is great prostration, shown by sinking in bed and sluggish reflex action, the motions passed unconsciously, the eyes half open, the attention scarcely to be

* Experience fully bears this out. It is therefore well to avoid the routine use of beef-tea. Veal-tea and chicken broth are far less likely to act on the bowels. It must, however, be remembered that in many cases of enterica there is no diarrhœa but constipation throughout the fever. Then beef-tea is rather indicated than not, and its valuable effect on the heart should not be lost. When there is much diarrhœa, and beef-tea is prohibited, we must depend upon stimulants to maintain the pulse.

roused, then, whatever the age of the patient, stimulants must not be withheld. Most patients above twenty or twenty-five years of age will need alcohol before the disease has run its course, but even here it is generally better not to begin its administration at once, but to wait for indications in the circulation, the breathing, or the general condition. In the somewhat rare and dangerous cases of fever in patients above forty-five or fifty, stimulants should be freely given from the first. Since typhus (and to a less extent relapsing fever) is not uncommon after forty while enterica is decidedly rare,* more cases of the former than of either of the latter fevers require free and constant use of stimulants. When the condition of the circulation, heart, pulse, and pulmonary congestion, or the general depression of the patient's powers demand alcohol, it should be given frequently every two hours or every hour, sometimes for a short time more frequently still. Children generally take brandy better than any other form of stimulant, diluted and sweetened. Young adults often do better with wine, especially red wine, such as burgundy and port. Elderly patients are sometimes wonderfully benefited by champagne. Whatever form is found best suited to the case, it should be given as a medicine, *i.e.* in definite doses and at definite intervals.

Drugs.—Almost all observers are agreed that no method of treatment has yet been discovered by which the course of enteric fever can be shortened, but there is no absurdity in supposing this to be possible. We have good reason to believe that the disease sometimes spontaneously aborts at the end of the second week, and there seems to be no reason why the number of cases in which this occurs should not be increased by the administration of some medicine or other. Liebermeister is disposed to believe that by three or four doses of from eight to ten grains of calomel, given within twenty-four hours at an early period of the fever, he succeeded in certain cases in cutting short the disease.

Treatment of the pyrexia.—Of late years, however, the attention of physicians, so far as the treatment of enteric fever is concerned, has been mainly directed to the question whether or not it is advantageous to the patient to have his temperature systematically kept down by *cold baths* or in other ways. And a mass of weighty evidence has now been accumulated in support of this practice, which, although it had been advocated in this country by Currie nearly a century ago, had fallen altogether into neglect. The revival of it was due first in 1861 to Brand, of Stettin, and secondly to Jürgensen, of Kiel, in 1866. Since that time it has been adopted by many physicians, both in Germany and in England; among ourselves no one has studied it more carefully than Dr Cayley, of the London Fever Hospital, who recorded his experience of it in the Croonian Lectures for 1880. A point of great importance is that those who recommend an *antipyretic* treatment in enteric fever appeal, not merely to the vague impressions derived directly from the observation of cases, but to a numerical analysis of the results which they have attained.†

It must be understood that the fundamental proposition upon which the antipyretic method of treating enteric fever is based is that pyrexia is

* The percentage at the London Fever Hospital was as follows. Out of upwards of 3000 cases of typhus, nearly 74 per cent. were under, and more than 26 per cent. over 40; out of 437 cases of relapsing fever the corresponding numbers were 82 and 17·5, and out of 1772 cases of enterica 95 and 5.

† See, for example, statistics of mortality in enteric fever, before and after the treatment by baths was introduced, at Berlin, at Hamburg and in the German army, by Senator, Goltdammer, and others, 'London Med. Record,' Dec., 1886, p. 557.

harmful, not because the bodily tissues undergo rapid waste in the maintenance of febrile heat, but because they are affected injuriously by exposure to a high temperature, especially when it is kept up for a length of time and without intermissions. For, on the former view, which till recently was generally adopted, it would be worse than useless to employ cold, since the effect is undoubtedly to increase for the time the evolution of heat within the body, even if the temperature does not afterwards rise again to its former level.

Notwithstanding the weight which the judgment of those who advocate systematic baths in enteric fever justly carries, many are unconvinced of its advantages. Statistics of results are much open to fallacies. Some epidemics are very slight, others very severe. A case early treated and well nursed and fed will do well when one not seen until the second week will die. The dependence of mortality upon the patient's age is so remarkable that we can only fairly compare those of about the same age. The two most formidable complications, hæmorrhage and perforation, at once change a favourable into a most serious or a fatal prognosis. Lastly, there is no acute disease which varies so greatly in symptoms, severity, and reaction to treatment, as enteric fever.

At present, therefore, it seems desirable (in accordance with the experience of Dr Alex. Collie and many other experienced physicians) to employ means of reducing the temperature, not as a routine method of treating the *disease*, but as a special method of treating the *symptom* of high fever. It is doubtful whether moderate pyrexia—say, under 103° F.—is of itself injurious, and whether its suppression, if possible, is desirable. But there is no doubt that hyperpyrexia is a dangerous condition in itself and should be dealt with energetically. At what point we should interfere cannot be laid down absolutely. A temperature of 104° in a child may be left alone, when one of 103·5° in a patient over thirty must be dealt with. A considerable evening rise which subsided well towards morning may be only watched, while pyrexia which rose high last night and only slightly remitted this morning must this afternoon be checked as soon as it begins its ascent. Moreover, the presence of delirium and of other symptoms of severe pyrexia will show that *for that patient* the temperature is high and must be treated, although the same or even higher temperature unaccompanied by grave disturbance of the nervous system and the circulation may be safely left to itself.

When we have decided on bringing down the temperature, there are several ways of accomplishing it. One is to place the patient at once in a cold bath, of 60° or 65° F. The shock may sometimes serve as a useful stimulus; but it is almost always better to use a tepid bath of 90° to 85° F. and rapidly cool the water with lumps of ice. The temperature must be watched by means of a thermometer *in recto*, and it must be remembered that it will most likely fall considerably after removal from the bath. Another important precaution is to give brandy immediately after, or even before, the bath, so as to stimulate the heart and further the cutaneous circulation. Currie's original plan of cold affusion is best adapted to relieving headache and delirium with pyrexia by a douche upon the head. The practical difficulties of a bath in the case of adult patients, and the serious disadvantage of lifting and moving them, it may be many times in a few hours, speak strongly for applying cold to the surface as they lie in bed. For this purpose the "wet pack" has been often used with good success; it is

usually soothing and sedative as well as antipyretic, but is less effectual than the bath and sometimes is resented. Another plan is placing bladders of ice in the axilla and over the great vessels of the neck and thighs, or fixing a coil of Leiter's tubes in the same regions and feeding them from a receptacle of iced water placed over the patient's bed. But in many, perhaps in most cases, the easiest and safest is also the most efficient method, namely, sponging the surface with cold water, or rubbing the trunk and limbs with pieces of ice, as the patient lies on a blanket with a waterproof sheet under it. Even when there does not appear any call for active interference, sponging the face, arms, and legs, with cold water or spirit lotion is grateful to the patient, and is often followed by tranquil sleep.

Many *antipyretic drugs* have been recently used in enteric and other fevers, sometimes in conjunction with, sometimes instead of, what may be called "the cold-water treatment." And certain of these have been credited with antiseptic power in addition, by which the course of the disease apart from the pyrexia is modified. Of these, quinine in large doses was at one time popular, but, except under special circumstances, it cannot be depended on to bring down hyperpyrexia, and is of doubtful value for any other purpose.* It is believed in this country that salicyl compounds, invaluable as they are in rheumatic fever, are probably worse than useless in pyrexia from other causes.

Kairin (a chinolin compound, the hydrochlorate of ox-ethyl-chinolin hydride) was one of the first artificial antipyretics introduced. It acts efficiently, but the effect is extremely evanescent.

Antipyrrhin (belonging to the same chemical series, dimethyl-oxychinicin) is more lasting in its effects, but it also has to be frequently repeated, and it has a dangerously depressing action on the heart. Moreover, it is apt to produce vomiting, and sometimes rigors.

Thallin (the sulphate or tartrate of tetra-hydro-parachinanisol) is, I am disposed to think, as efficient, or more so, and safer. But it sometimes produces rigors, and is probably not entirely free from danger.

Antifebrin (an anilin not a chinolin compound, phenyl-acetamide) is said to be more useful, more agreeable, and more safe than any. I have found ten grains cause collapse. One great drawback to all such drugs is the difficulty of knowing when to give them so as to *prevent* the rise of temperature.

Apart from special treatment of the pyrexia, or of the particular symptoms to be mentioned immediately, it seems to be grateful to most patients to take what is called 'a simple febrifuge,' such as ten drops of dilute hydrochloric acid in infusion of orange, of calumba or of serpentary. It is probably a mere placebo, but there is every reason to please as well as to cure our patients.

Treatment of other complications.—The most fatal of all, *perforation*, can only be relieved by opium when it has once occurred. Treatment consists in preventing it, by checking peristalsis, and by scrupulous care in feeding after as well as during the attack.

Hæmorrhage should always be regarded as a serious symptom, and stopped, if possible, by gallic acid, by acetate of lead, by ergot, and above all by laudanum, or subcutaneous injection of morphia and ergotine. Turpentine was used by Graves, and is sometimes efficacious, but the great indication is to stop peristalsis by opiates. According to statistics, the practice of systematic cold baths does not appear to favour intestinal hæmorrhage in this disease, but the reverse.

* "Large doses of quinine are as useless and injurious as in typhus."—MURCHISON.

In cases of enterica, which are not cut short by perforation or hæmorrhage, the cause of death is usually failure of the circulation with hypostatic congestion of the lungs. Beside the free use of stimulants, as described above, great benefit is derived from the common senega and ammonia mixture, or carbonate of ammonia alone in three- to five-grain doses, with its pungency removed by treacle, syrup of tolu, or liquorice, and, if needful, laudanum or paregoric added to prevent laxative effects. With this digitalis may be given, and sometimes its effect on the rapid, feeble, and irregular pulse is striking and most useful; but often it disappoints us, and on the whole is less to be depended on in these cases than when a similar condition of the pulse is produced by valvular disease of the heart.

Diarrhœa.—This should be checked from the first. If there are not more than two loose motions in the twenty-four hours, and the patient is young, and the fever not severe, we may wait; but as soon as this is exceeded, the starch and opium enema of the Pharmacopœia should be administered, and repeated after each movement of the bowels. When pain and restlessness are also present, Dover's powder is probably the best form of opiate to give internally, and chalk mixture, catechu, or other astringents are often prescribed with advantage; but in most cases it is better to leave the stomach as much as possible for food and alcohol.

When there is constipation instead of diarrhœa, and especially if there is much flatulence and discomfort, a soap-and-water enema may be administered. Murchison recommends a teaspoonful of castor-oil, and repeats it every three or four days if there is constipation throughout the fever. Knowing, however, that in all cases there is some ulceration of the ileum, and that the degree of diarrhœa is no certain guide to its severity, I venture to think it better to err on the side of caution and to abstain from meddling with the bowels until convalescence is established. I have frequently seen a patient pass through the disease favourably with constipation throughout; I have twice seen such cases die from perforation and been thankful that I had not used even an enema; and again and again I have seen the bowels act naturally and comfortably after the temperature has fallen, without aid from drugs.

No qualified man would think of giving ordinary laxatives in a case of enteric fever, but we often meet with cases in which harm has been done by saline and other purgatives being taken for a supposed "bilious attack" before the true nature of the disease has been recognised.

Tympanites is not only an unfavourable symptom but is also injurious. It may be met by turpentine stupes or enemata containing asafœtida or other carminatives. The distension is chiefly in the colon, and therefore drugs given by the mouth are not of much service.

Bedsores ought to be prevented by extreme cleanliness, by careful drying and dusting after each evacuation, and by daily friction with brandy from the first. When the skin is already red and threatens to break, it is better to apply lead lotion frequently. Careful shifting of the patient so as to vary the points of contact as much as possible is part of a skilful nurse's duties. A water-bed is an important preventive.

Treatment during convalescence.—Stimulants should be at once diminished on the subsidence of fever, and in many cases it is desirable to substitute an ounce or two of wine twice or thrice a day for brandy at frequent intervals. With young patients, however, after a favourable attack there is often no need for stimulants or drugs. No solid food should be given for a fortnight after

fever and diarrhœa have ceased. The patient will bitterly complain of the restriction, but if the physician has once lost a patient from perforation during convalescence he will be inexorable ever after. Progress to health after this disease must be slow if it is to be sure. Leaving the bed, taking meat, going downstairs and out of doors—each stage of convalescence must be carefully considered. And there is no disease, not even rheumatic fever, in which it is so important for the patient to have a long period to recover his strength before returning to his ordinary duties.

FEBRICULA.—When continued fever was no longer regarded as one "disease" with endless varieties, and when the distinction between Typhus and Enteric was established, Relapsing Fever formed a third "species," and a fourth was called Simple Continued Fever, ephemeral fever, or febricula. This was described by Jenner in his well-known Lectures, and admitted by Murchison among the 'Continued Fevers of Great Britain.' But most physicians now believe that a disease without any morbid anatomy, without any known ætiology, and without any definite or characteristic course or symptoms, cannot be admitted into a useful nosology. This was the opinion of the late Dr Tweedie. In some admirable 'Lectures on Fever' delivered by Dr Peacock, of St Thomas's Hospital, he wrote as follows: —"The cases which have been classed under this head embrace a variety of different affections. Some are probably cases of imperfectly developed typhus, typhoid, or eruptive fever; others may be cases of relapsing fever, in which the relapse does not occur; and yet others may depend on common causes—exposure to cold or damp, noxious miasms, or on gastric, intestinal or hepatic disorder. I should rather regard the so-called ephemeral fevers as abortive attacks of one or other of these forms of disease, than as possessing a specific character of their own. You will constantly find that when a series of cases occurs in members of the same family, some are characteristic attacks of typhus or typhoid, while others present only slight febrile symptoms, to which we may apply the terms Febricula or Ephemera. Thus, in the three cases of typhus which I have before referred to as examples of contagion and of the varieties in the form of the eruptions on the skin, the first two, those of the patient and the first nurse, were examples of characteristic and severe typhus; the third case, or that of the second nurse affected, was merely a slight febrile attack in which no eruption appeared on the skin. Again, among the typhoid cases is included one which presented the characteristic features of the disease during life, and proved fatal, and after death extensive intestinal disease was found. The brother of this boy was under treatment at the same time, and in his case the fever was only slight; and numerous similar examples might be quoted. We see facts precisely analogous during epidemics of the eruptive fevers, and especially of scarlatina."

With this view I fully concur. It may have been needful to include in the College of Physicians' Nomenclature (1884) "Simple Continued Fever," with the definition, "Continued Fever, having no obvious distinguishing character;" but if obvious characters for diagnosis fail, we must seek for those which are more latent.

It must, however, be admitted that remarkable cases of fever do occur, which we must at present leave unexplained and without a name.

(1) There are cases of pyrexia and even hyperpyrexia without other

signs of fever. Some of them have been referred to already (*supra*, p. 41 which occur in women between the ages of fifteen and forty-five; but others remain. In 1886 I had a patient in Guy's Hospital, a healthy, decent mechanic, of about forty, who for several weeks suffered (or rather did not apparently suffer) from high temperature, with the normal diurnal variations exaggerated, and sometimes reaching 105·6° F. Yet he ate and slept well, and there was no local lesion to be found. At last the temperature gradually fell, and he went out well. The case was published by my colleague, Dr Hale White.

(2) Children are apt to be affected by pyrexia, and sometimes by a sharp attack of fever, from causes which in adults would cause only insignificant disturbance. Slight gastric catarrh from eating indigestible food, diarrhœa from fruit, catching cold, or exposure to heat or fatigue, will be enough to cause febricula, and the diminutive will often apply to the duration only, not to the height of the pyrexia. The "infantile remittent fever" of Evanson, and Maunsell, and other writers is probably almost always enterica without the characteristic rash.

In young women (who retain the pathology of children much later than youths of the other sex) similar short and sharp fever is not infrequent from "gastro-entérite" due to indigestion or chills, from retention, suppression or disorder of the menses, and particularly from a degree of sore-throat which falls very far short of quinsy.

Here may be mentioned the remarkable cases of gastro-enteritis with fever which are sometimes undoubtedly the result of eating bad meat or fish.

(3) Slight cases of enteric fever have often been called febricula (*v. supra* p. 177). In the absence of the rash, enlargement of the spleen is probably the best diagnostic sign. See an interesting discussion reported in the 'Dublin Journal of Medical Science' for July, October, and November, 1879, by Drs Cameron, Grimshaw and others, and a valuable paper by Prof. Bäumler of Freiburg-in-Breisgau, on the question, "Can the mildest forms of enteric fever be distinguished from acute febrile but non-specific gastro-enteric catarrh?" (*ibid.* November, 1880). See also Jurgensen's paper in 'Volkmann's Sammlung,' translated for the New Sydenham Society.

(4) There remain, however, certain epidemic and probably contagious febrile disorders which cannot as yet be brought under any recognised category; they are neither continued, eruptive, malarious, nor symptomatic. The best observed instance with which I am acquainted is detailed with great care and judgment by Dr Seaton in the Clinical Society's 'Transactions' for 1886 (vol. xix, p. 26). No less than 157 cases occurred during the four summer months (June to September) in a Boys' Orphanage. The attack began suddenly with rigor and severe headache, and the temperature rose rapidly to its maximum, which varied from 101° to 105° or even 106°. There was often severe vomiting, with no diarrhœa, but with scanty urine in which the chlorides were remarkably deficient. In most cases the fever ceased in two or three days, in others on the fifth or sixth, and labial herpes usually appeared at the same time. Earache with otorrhœa and pneumonia were the two complications noticed. Death occurred in seven cases. *Post-mortem* examination threw no light on the disease, but it appears to have been only made in a single case.

SCARLATINA*

History and distribution—Contagion—Modes of transference—Surgical and puerperal scarlatina—Incubation—Onset and Course—The throat—The rash—Abortive and malignant varieties—Complications and sequelæ—Prognosis—Protection—Prophylaxis—Treatment.

THE recognition of Scarlet Fever as a distinct disease began with Sydenham (1675), and was completed by Withering a century later (1778). It had, indeed, been described as far back as 1556 by Ingrassias under the name of Rossalia; but Morton, who was a contemporary of Sydenham, maintained that it was only a variety of measles, and called it *morbilli confluentes.* The designation *febris scarlatina* (Ital. *scarlatto* = *scarlet*), was employed by Sydenham. It is very remarkable that he made no allusion to sore-throat as one of its symptoms.

From the persistence and virulence of its contagion and from the severity of its effects, this disease is justly dreaded. Since the introduction of vaccination it has taken the place of smallpox as the most common and fatal of all the specific fevers.

Its geographical distribution is now very wide, but like smallpox and measles, relapsing fever and typhus, it has been introduced from Europe into other parts of the globe within historical times. Thus it appears to have been unknown in the colonies of North America until the year 1737 and in South America for nearly a century later. It was brought to Iceland in 1827, to Greenland in 1847, and about the same time the first cases were recorded in the Australian colonies.

Ætiology.—The *contagium vivum* is chiefly present in the epidermis which is shed. It is particulate, but although more than one form of micrococcus and bacillus is present in the secretions of the fauces during the disease, none have been identified as pathogenic or even as characteristic.

The channels and mode of entrance of the contagium are not always the same. The dry epidermic dust is probably most often taken in with the air in breathing, or with food and drink in swallowing. It is often conveyed by clothing or other fomites. Attempts to reproduce it in the lower animals by transference of blood or epidermis have not succeeded, but investigation into a remarkable recent epidemic by Dr Power and Dr Klein appears to render it probable that Scarlatina may not only be conveyed from one person to another by means of milk as before ascertained, but that the disease may originate in cattle, and be conveyed from them by their milk to children.†

The *contagion* of scarlatina, though virulent and lasting, is not easily diffusible. Thus, it rarely crosses a street, and epidemics do not spread so quickly as those of typhus, measles, variola or diphtheria. But they linger long and die slowly out.

* *Syn.*—Scarlet Fever—Febris rubra.—*Germ.* Scharlach, *Ital.* Febbre Scarlattina.

† Dr Klein has since succeeded in reproducing the disease in cattle.—'Proc. R. S.,' March 3, 1887.

Although scarlet fever never arises but by contagion, yet, as in other cases, there are conditions which predispose to receiving and developing it.

First of these is the *age* of the recipient. It is rare in infants, most common in early childhood (Murchison found 64 per cent. of cases to occur between the ages of one and five), and rare after puberty; but it may occur even in advanced age.

Secondly, puerperal women are peculiarly liable to take the contagion; and, with somewhat masked features, it forms a considerable proportion of the cases known as puerperal fever. Dr Braxton Hicks found that in eighty-nine cases of this terrible disorder no fewer than thirty-seven either showed a scarlatinal rash or had been subjected to infection from patients with scarlatina.

Thirdly, persons who are suffering from wounds are extremely prone to infection. The sorethroat and fever are frequently present without the rash, or the rash is slight and quickly over. But that the affection is genuine scarlatina is proved by its "breeding true" and by its protecting against future attacks. Moreover, although most of these cases are mild, like inoculated smallpox, occasionally a severe one occurs along with the rest and exhibits the sequelæ as well as the symptoms of the disease. Sir James Paget and Mr Howse, Dr Gee, Dr Eustace Smith and Dr Goodhart have placed the true nature of this "surgical scarlatina" beyond doubt. It is remarkable that according to the observation of the last-named author, antiseptic precautions do not prevent the infection, so that the otherwise probable supposition that the contagion gains a direct entrance through the open wound appears to be untenable.

Incubation.—This period is short compared with that of enterica, typhus, measles, or smallpox. It most commonly lasts between two and five or six days. Occasionally the interval between infection and the first symptoms may be still shorter, twenty-four hours, or perhaps even less. Among the cases with the shortest incubation are those of puerperal and surgical scarlatina just mentioned. Although rare, instances occur in which more than a week has elapsed between contact with a case of scarlatina, and appearance of its first symptoms, so that the incubation may be sometimes as long as that of smallpox or of measles.*

Course.—The onset of the disease is generally quite sudden. In children the first symptom is often vomiting or a convulsive seizure. In adults it is usually soreness of the throat, and there may be chilliness or even a rigor. The patient complains of headache, malaise and prostration.

The face quickly becomes flushed, the pulse remarkably rapid, and the skin hot. For a child's pulse to be at 140 or 160 within a few hours is not uncommon, nor of unfavourable augury; and it may remain high for some days.

The *temperature* may rise to 104° or 105° in the course of the first day, or it may attain the same point more slowly afterwards, while the rash is coming out. It then generally rests stationary, or nearly so, until the rash begins to fade. The extreme dryness of the surface is apt to give one an impression that it is hotter than is really the case; from the time of Addison the pungent heat of the skin in scarlet fever has been spoken of at Guy's

* In one case, under my own observation, a child left his father's house, where three of the family had died from scarlatina, on November the 19th and continued well until December the 2nd, when he developed the disease, just a fortnight after the last exposure to contagion. It is, however, possible either that the infection was from a different and more recent source, or that it was from fomites which were carried away with him from the house.—Ed.

Hospital as comparable only with that which is to be felt in acute pneumonia. Dr Gee describes the fever as frequently ending in a complete crisis; this occurred in two of his cases on the fourth day, in four on the fifth, and in three on the seventh. But its fall is more often gradual, taking from three to eight days for its completion.

The *urine* is scanty and high coloured as in other fevers, an abundant precipitate of lithates forms as it cools, and it often contains a little albumen even at this stage. Urea is in great amount and the proportion of potash salts is increased.

The *tongue* is at first coated with a thick creamy layer, as in other febrile diseases. But before long enlarged fungiform papillæ are seen projecting as shining scarlet points, and after two or three days the white fur clear away from before backwards, leaving a smooth bright red surface; this, with the little prominences that are thickly scattered over it, has an appearance that has been aptly compared with that of a strawberry. Sometimes, however, no such enlargement of the fungiform papillæ occurs. German writers speak of a miliary vesicular eruption as occasionally present, especially on the dorsal surface. The tongue does not regain its normal aspect until convalescence has set in.

On looking at the *fauces* one finds the arches of the palate, the uvula, and the tonsils more or less reddened or purple. Sometimes, but not always, they are swollen; and the redness may extend to the roof of the mouth and to the pharynx. Swelling of the mucous glands often gives a granular appearance to the affected parts; and their secretion may accumulate upon the surface so as to simulate the presence of ulcers. The tonsils may even project inwards until, with the club-shaped uvula, they block up the passage; and after a few days one or both of them may suppurate. There is often swelling of the neighbouring cervical lymph-glands.

The *rash* generally makes its appearance in from twelve to thirty hours after the commencement of the disease. Sometimes it is later; but, as Dr Gee remarks, the proof of this is difficult, not only because the first signs of it are easily overlooked but because it may recede for a time, and then come out again. In some of the more severe cases it is altogether ill developed, and writers say that it may then be delayed until the third or the fourth day. In the great majority of cases the rash appears towards the end of the first day and is fully out on the second.

As a rule, the rash is first to be discovered upon the sides of the neck and the upper part of the chest; and it generally takes twelve or twenty-four hours, or even two or three days, to reach its full development. But sometimes it comes out almost at once over a very large surface. The face often remains free from it. Prof. Thomas, of Leipzig, insists that in most cases it is at first limited to the forehead and temples, the cheeks showing only the ordinary flush of fever, while the parts round the mouth, nearly to the chin, remain pale and afford a striking contrast. The upper arms are often covered with the eruption, and it is exceedingly well marked on the abdomen and on the inner side of the thighs. The colour is usually a bright scarlet, so that Watson aptly compared it with that of a boiled lobster; but sometimes it is of a lighter pink, and sometimes purplish. It may appear to be uniformly diffused, but on careful examination one can generally see that it is made up of very minute red points, which are at first isolated from one another, and which, even when they have coalesced, often leave a little islet of healthy skin here and there. It momentarily disappears on pressure

except where there has been some degree of hæmorrhage or perhaps capillary stasis, in which case isolated red spots remain, with a more or less general yellow discolouration. As Watson long ago pointed out, on the forearms and the legs, as well as on the backs of the hands and of the feet, the rash of scarlet fever consists of larger and more prominent papules than elsewhere; the palms and the soles, however, show only a faint diffused blush. The skin of the affected parts is slightly turgid or even swollen; the eyelids and the cheeks look a little puffy. Trousseau remarks that tumefaction of the fingers often prevents the patient from closing his hand.

Löschner discovered exudation-cells in the rete Malpighii, and probably they were seen by Dr Fenwick also, who further observed that the basement membrane of the sweat-glands was thickened, and their channels were obstructed by an overgrowth of epithelium, or by extravasated blood. That the scarlet fever eruption bears no definite relation to these glands, nor to the hair-follicles, seems to follow from a case (cited by Thomas on the authority of Landenberger) in which it did not fail to develop itself over an immense cicatrix, the result of a burn which was said to have destroyed the whole thickness of the skin.

Certain modifications of the eruption of scarlet fever are sometimes observed. Instead of being punctiform, it may in rare cases consist of large, irregular, slightly raised maculæ, more or less like those of measles, or it may be markedly papular. Or, again, it may be associated with immense numbers of miliary vesicles, especially upon the neck and chest, when there has been much perspiration.

At a variable period after the subsidence of the rash—which usually takes place from the fifth to the seventh or eighth day of the disease, but sometimes earlier—the superficial layers of the cuticle begin to peel off, or, in technical language, to *desquamate*. This is sometimes observed within a few days, sometimes not until two or three weeks have elapsed. Its amount is not necessarily proportionate to the intensity of the cutaneous affection; it may be well marked where the rash was so slight as to have been overlooked, and so may be of service in clinching a doubtful diagnosis. In some cases it assumes a furfuraceous form; a mealy powder, or a fine scurf, becoming detached from the surface, especially from the face. But more generally it is "membranaceous;" distinct flakes come away, and may be some inches long. The first step towards the throwing off of the epidermis at a particular spot is often the formation of a little opaque raised vesicle, very like those which are characteristic of eczema, but dry; this breaks at the summit, leaving a free edge in the shape of a ring, which gradually becomes larger and larger. The cuticle of the hands is now and then shed *en masse*, like a glove; and the same may be the case with the feet; in some cases not even the nails are left behind. So complete a desquamation as this necessarily takes several weeks for its completion. Much more often, however, the fact that the exanthem interferes for the time with the nutrition of the nails, is shown, not by their exfoliation, but by the formation of a transverse groove upon them: to this Dr Wilks has especially drawn attention. It is seen upon several of the nails at equal distances from their roots, and of course it ultimately becomes lost at their distal extremities. Such grooves on the nails are not, indeed, peculiar to scarlet fever, being sometimes seen after other acute diseases; but, even with this qualification, their presence sometimes throws valuable light upon the origin of sequelæ, the real nature of which might otherwise have remained undetermined.

The hair comes off with the cuticle; but, so far as I know, not more than after any other fever of equal severity.

Desquamation follows the same course as the rash, beginning on the chest, shoulders, and neck, then spreading over the arms and back, and then over the lower extremities. It is often delayed in the hands and feet until long after the rest of the skin is clear.

Aberrant forms.—A striking feature of scarlet fever, and one upon which all writers lay stress, is the great variability of its symptoms and of its course; so that, indeed, there is much difficulty in describing all the different forms it may assume.

In some cases the disease is *rudimentary* or *abortive.* Thus, during epidemics, it often happens that adults become more or less feverish, complain of a slight sore-throat with redness of the mucous membrane, and have a little pain and swelling of the cervical glands, but are at no time really ill. This is especially apt to be the case with those who have already had an attack in childhood; medical men have told me that it has again and again occurred to them, when attending patients suffering under scarlet fever. But I lately saw an instance in which a father so affected gave to his children a disease of such severity that one of them died. Again, the chief symptom may be feverishness, so that the complaint passes for a mere febricula, there being little or no affection of the throat, and no redness of the skin beyond what might be attributed to the pressure of clothes or to some other accidental circumstance. Lastly, the sole indication may be an eruption, and this perhaps limited to a small part of the surface. The real nature of this affection is often first shown by the occurrence of desquamation, or even by the unforeseen supervention of dropsy or of some other sequela.

In a very different class of cases, the symptoms are incomplete because the end comes too quickly for their full development. In several recorded instances the patients have died in from eight to fifteen hours; the chief phenomena have then been delirium or coma, convulsions, or even tonic spasms with trismus, incessant vomiting, and diarrhœa, with extreme rapidity of pulse and of breathing. Sometimes there has also been hyperpyrexia, in which case one would be inclined to regard this as determining the form of the disease. A temperature of 115° F. was observed by the late Dr Woodman in some patients. The only thing that could render a diagnosis possible would be the occurrence of other cases of scarlet fever in the same neighbourhood.

The cases just referred to constitute the most extreme examples of what in England is commonly called *malignant* scarlet fever. But there are other varieties of it which are less rapidly fatal. Sometimes, with early severe constitutional symptoms of a "typhoid" character, the rash comes out late and imperfectly, and its hue is not bright scarlet, but rather a livid violet. In some instances the disease assumes a *hæmorrhagic* form; at least, almost all writers say so except Dr Gee, who is evidently inclined to think that the early hæmorrhagic roseola of smallpox may have been mistaken for it.

Sometimes, again, the eruption develops itself in the regular way; but the febrile disturbance, which from the first is high, runs on for two or three weeks instead of subsiding after a few days. The patient then falls into a typhoid state, with muttering delirium, a dry brown tongue, and sordes upon the lips. Many of these cases end fatally. In a large proportion of

them the affection of the throat is unduly severe. Either the fauces are covered with a diphtheritic exudation, or they become gangrenous and slough away, leaving a horribly offensive cavity. The inflammation often spreads to the lining membrane of the nose, and an acrid fluid flows from the nostrils, which excoriates the upper lip. The nasal bones may even become necrotic. Still more frequent is extension to the ear along the Eustachian tube; the tympanum then fills with pus, which is discharged by perforation of the membrane. Here, again, there is often great destruction of the tissues; Sir Thomas Watson mentions a case in which, every time that the child swallowed, some of the fluid food ran out immediately at one of its ears; and it is no uncommon thing for fatal hæmorrhage to ensue from perforation of the carotid artery.

In other cases, the chief local mischief is outside the throat, in the cervical lymph-glands and the adjacent structures. A large swelling may form on each side of the neck near the angle of the jaw; or the whole space from the chin to the sternum may be occupied by a brawny shining mass, which has been termed a "collar." It does not appear that the salivary glands, whether parotid or submaxillary, take any part in this affection. The induration of the connective tissue may spread backwards between the pharynx and the spine, or downwards into the mediastinum. If suppuration occurs, the pus may be discharged by a series of fistulous openings burrowing and undermining the skin in all directions; or a post-pharyngeal abscess may be formed, which, pressing on the larynx, may cause suffocation. Extensive sloughing often takes place, so that the muscles are laid bare, as in a clean dissection; and dangerous or fatal hæmorrhage may ensue, from perforation of one of the arteries or veins of the neck.

Complications and Sequelæ.—Some of the affections just described ought perhaps to be reckoned under complications of scarlet fever, rather than any other head; but I do not see where the line is to be drawn. We may, however, certainly give that name to one morbid condition which is comparatively seldom observed, and unrepresented in the normal course of the disease, but which is by no means confined to cases of great severity. I refer to *synovitis*, a painful swelling of some or all of the joints, which generally sets in while the skin is peeling. This affects sometimes the smaller, sometimes the larger articulations; it generally subsides quickly, and Thomas says it is even more fugitive than that which occurs in acute rheumatism. In some cases, however it "settles" into one particular joint, leading to chronic effusion, or even suppuration.* Probably most cases of synovitis following scarlatina are true rheumatism.

A more frequent and more important complication of scarlet fever is acute *nephritis*, accompanied with albuminuria. Whether any affection of the kidney is present in ordinary cases of scarlet fever, which end in recovery, appears to be still doubtful. Thomas remarks that the question could be settled only by the microscopical examination of the organs from a patient killed by an accident during the course of the disease. It is certain that nephritis has been found after death in some exceptional cases of scarlatinal dropsy in which the urine, although scanty, contained neither albumen nor casts during life. This fact obviously suggests the possibility that renal

* Dr Ashby, of Manchester, finds synovitis in the course of the fever to be very rare and unaccompanied by evidence of cardiac inflammation. Subsequent rheumatism is less rare in young adults than in children. Endocarditis (rheumatic or nephritic) is less common as a sequela than dilatation of the heart ('Lancet,' May 22, 1886).

changes may be present in cases which do well, without any evidence being discoverable. It is also certain that the more assiduously one tests the urine throughout the whole course of the disease, the more numerous are the cases in which a trace of albumen is detected at one time or another. Thus some German writers look upon a renal catarrh as bearing to scarlet fever the same relation which bronchial catarrh bears to measles. But albumen and occasionally even tube casts are found in the urine in other febrile diseases, so that their presence is not necessarily to be taken as part of the action of a specific poison upon the kidneys. Scarlatinal *dropsy* I shall leave to be discussed with the similar condition that results from other acute forms of renal inflammation. Anasarca without albuminuria is occasionally met with after scarlet fever.

Serous inflammation.—Pleurisy and pericarditis are of rather frequent occurrence, especially in connection with the rheumatoid affection of the joints. This may also be attended with endocarditis, and so may doubtless be the starting-point of chronic changes in the valves of the heart; but whether such changes ever arise when the joints have remained free appears to me to be doubtful. In making *post-mortem* examinations of children who have recently had scarlet fever I have always found the valves healthy. Pleurisy and pericarditis, or pneumonia, may also accompany scarlatinal nephritis.

Chronic enlargement of the *tonsils* is very common in children who have passed through this exanthem. Far more important are various affections of the *ear*, which are apt to be left behind by it, so that indeed it is almost always responsible for the occurrence of deafness acquired in early life. The most frequent of these is acute suppurative tympanitis produced by extension of the faucial inflammation along the Eustachian tube. It often leads not only to perforation of the membrane, but to necrosis of the petrosal bone, and subsequent fatal pyæmia. Necrosis of other bones is not very uncommon, and glandular abscesses sometimes follow during convalescence.

An important complication, sometimes occurring in the first week, sometimes not until convalescence, is *diarrhœa*. It occasionally leads to fatal exhaustion.

Prognosis.—The prognosis of scarlet fever cannot be altogether devoid of anxiety: the disease is never so mild but that some dangerous complication may arise. Sporadic cases are sometimes quite as severe as those which form part of an epidemic. Indeed, different epidemics differ very widely in their gravity. I have known one to prevail for many months in a country town without there being a death, and without dropsy ever supervening. Graves has recorded the fact that whenever scarlet fever appeared in Dublin between 1805 and 1833, it was always mild, so that on one occasion eighty children were attacked in the same public institution, and all recovered; but during 1834 and 1835 the city was the seat of a very malignant and fatal epidemic. Thomas says that a mortality of from 13 to 18 per cent. is very common, but that it not infrequently rises to 25 and sometimes even to 30 or 40 per cent.

I have already indicated most of the inferences which are to be drawn from particular symptoms or combinations of symptoms. But it may be added that, according to Trousseau, the occurrence of convulsions during the first or second day is always a sign of danger, whereas in other exanthemata it is not of evil omen. Another point which is made by this writer is that in scarlet fever, as in smallpox, the more intense the rash the more severe is the disease. Here, however, he differs from most other observers;

and I think that the statement can only be admitted to be correct in so far, that those cases in which a bright red eruption covers the whole of the body and lasts for an unusually long time, are generally grave ones.

Diphtheria is happily a rare complication, for it is extremely fatal.

Protection.—This is less complete than in the case of typhus, enterica, smallpox, or perhaps measles. Second and even third attacks are not very uncommon. But they occur after a considerable interval, and relapses are seldom or never seen.

We have no means of *prophylaxis* but isolation. Belladonna has been fully tried and conclusively proved to be useless. Arsenic has been credited with a similar power, probably on insufficient grounds.

Treatment.—We have no means of checking scarlatina when the contagion has been once taken in, and must be content to treat it on the same general principles as those indicated in the chapters on Typhus and Enteric Fever.

In mild cases little is needed beyond careful isolation and free diluents.

When the temperature is very high, 104° or upwards, we must check it by cold sponging, rubbing with ice, or cold baths, as described above (p. 204).

The angina causes much pain and difficulty in swallowing. Sucking ice gives great relief, and if the patient will allow it without too exhausting a struggle, it is useful to clear the throat with a large camel-hair brush or with cotton-wool firmly tied on to a penholder. Older patients may use Condy's fluid or chlorate of potash as a gargle, or borax and honey as a linctus, with advantage. In severe cases nutrient enemata may be tried, and if they fail, as they unfortunately often do, the patient must be fed by the nose; not with a catheter—a child's nasal passages are too small and the process would be too long—but by inserting a glass funnel, of the kind used for filtration in laboratories, into the opening of one nostril, closing the other, and pouring milk and egg, beef-tea or other fluid nourishment into the pharynx.

Brandy must be given if the pulse requires it. Wine is often useful during convalescence.

As soon as the eruption is fully out, inunction with carbolic oil (1 in 30) should be begun, and when the fever has subsided and desquamation has begun, the hair should be cut short, and the whole body well washed with hot soap and water, followed by inunction. A warm bath with soap may then be given daily. The oil is not only pleasant to the patient but prevents the desquamating scales from flying about.

Great care should be taken during the stage of peeling to keep the patient from draughts. The body should be clothed in flannel, and any chill carefully guarded against. The urine should be daily tested and while it contains albumen the patient should not be allowed to leave his bed or to take meat.

If, in spite of precautions, nephritis and dropsy follow, the case is one of acute Bright's disease, and must be treated by purges and other means, as subsequently detailed in the second volume.

Rheumatism as a sequela is amenable to salicyl compounds, another proof of its true nature.

During convalescence tincture of steel is the most useful drug. Even in mild cases the child should keep his bed for three weeks and his room for a month at least from the beginning of his illness (Eustace Smith).

The rules for disinfection given on p. 34 must be thoroughly carried out. The patient must not associate with other children until the desquamation has ceased, except perhaps that on the soles of the feet, which sometimes lasts for weeks after the rest of the surface is clear.

MEASLES*

History and Nomenclature—Ætiology—Incubation—Onset—Course and Eruption—Varieties—Complications—Sequelæ—Protection—Diagnosis—Prognosis—Treatment.

Two centuries only have passed since the writings of Sydenham (about 1675) first led to the separation of Measles and Scarlatina from one another. The celebrated Arabian physician Rhazes† described Smallpox and Measles in a treatise which was translated into Syriac, Greek, and Latin, and is still extant; he distinguished these two diseases, but probably confounded Scarlatina with Measles. Measles was long regarded as a minor form of smallpox.

The name Morbilli (Ital. = a little plague) seems to have been constantly applied to it until the middle of the eighteenth century, when Sauvages first called it Rubeola. This designation was unfortunately adopted in Great Britain by Cullen, and by Willan; and their authority led to its general acceptance. But within the last few years most English writers have reverted to the use of the term "morbilli." The English word measles means spots or pimples.

Origin and propagation.—Measles is a strictly contagious, specific, febrile disorder, and has been propagated over temperate, warm and cold climates, so that it is now almost pandemic. Its introduction into Iceland, the Faröe Islands, and the Fiji Archipelago, took place in recent times. Like smallpox and syphilis it was much more severe and fatal when thus imported into a virgin soil.

The contagion, though active and sure, is not nearly so persistent as that of scarlatina, and it is readily dissipated so as to become innocuous by ventilation. It is probably conveyed by the secretion from the affected mucous membranes by the breath, and perhaps also by the skin.

The contagium has not been isolated, and it is unknown whether it is a microphyte. Messrs Braidwood and Vacher observed minute glistening particles in the mucus of measles which resembled those seen in vaccine lymph ('Path. Trans.,' 1878, vol. xxix, p. 421).

It is not known to be inoculable by the blood, nor is it certain whether measles occurs among the lower animals.

Like scarlatina, measles is a disease of children. Most persons above puberty have already had it. But cases occur at any age, and unprotected adults take it very readily and severely. Whooping-cough almost all writers agree to be a decided predisposing cause for measles.

Incubation.—This has been carefully observed in cases which have occurred on board ship, and in the remarkable epidemic which invaded the Faröe Islands in 1846. Panum (afterwards the eminent professor of physiology at Copenhagen) found that a period of thirteen or fourteen days elapsed

* *Synonyms.*—Morbilli—Rubeola.—*Fr.* Rougeole, *Germ.* Masern, *Ital.* Rosolia fersa, *Arabic* Hasbah.

† Abubekr Mohammed ar-Razi, born at Rai (whence his surname) a town of Irak Ajemi in Persia, about 850 A.D.

between exposure and the appearance of the rash, *i. e.* the incubation period was ten or eleven days.

Further exact observations have shown that although variations occur, they are less than in most other cases of contagion, so that the disease rarely manifests itself before the tenth or after the thirteenth day. When designedly inoculated by mucus from nostril to nostril, the period of incubation is shorter, as with inoculated variola—eight or nine days.

During this period the child continues apparently in perfect health.

Onset.—The *prodromal* stage of measles presents more than one peculiarity. In the first place, although the patient is taken suddenly ill in the ordinary way with anorexia, headache, and malaise; although vomiting may set in, or diarrhœa, or epistaxis, or a rigor, or (in children) a convulsive seizure; and although by the evening of the first day the temperature rises rapidly so as to reach 102°, or more, yet the course of the fever during the next two or three days is altogether uncertain. On the second or the third day the thermometer may fall to normal; and, as the appetite may at the same time return, the idea of an impending illness may be almost abandoned. In other cases, however, the temperature remains at about 102°, with only trifling oscillations backwards and forwards.

In the second place, there are catarrhal symptoms from the first; the patient is troubled with coryza, sneezing, intolerance of light; fluid secretion pours from his eyes and nose, his face and eyelids are swollen, and his conjunctivæ are injected; he may be hoarse, he may have a cough, sibilant sounds or rhonchi may be audible on auscultation over his chest; and the cough may even be harsh and "croupy." By the second, or at latest by the third day, one finds on looking at the fauces that besides a general injection of the soft palate, there is an eruption of scattered points and spots over its mucous membrane. Ringer attaches some importance to the presence of thin, opaque, white patches on the gums, and the inside of the lips. In the pharynx one can hardly recognise anything more than a diffused redness, and the same has been observed within the larynx by the aid of the laryngoscope. Some writers have regarded all these appearances as indications of a "mucous exanthem," and have proposed to term it an "endanthem." But I agree with Thomas in thinking that such a view is certainly tenable only for the affection of the fauces; although it may be of clinical value in enabling measles to be recognised among the dark races of mankind, in whom no cutaneous exanthem is visible.

The tongue in this disease is coated with a white fur, through which a few red papillæ may perhaps be seen projecting. The faucial affection and the early catarrhal symptoms in general continue into the eruptive stage and then subside. According to Thomas, indications of the cutaneous rash may sometimes be seen on the face during the prodromal stage, in the form of minute puncta, around which the characteristic papules afterwards develop.

Course.—The *eruptive* stage which succeeds begins generally on the fourth, but sometimes on the third day of fever, and from the fourteenth to the seventeenth from infection. In exceptional cases it is postponed until, according to Trousseau, six, seven, or eight days have elapsed; or even, according to Watson, until the tenth day. There is now a further rise of the temperature, which in thirty-six hours (as a rule, on the sixth day) reaches its acme, this being generally 104° or 105°. Afterwards it may either begin at once to decline, or may remain at or near the same point for a day or two. The fall,

when it does occur, is rapid, and the normal temperature is reached by the eighth or the ninth evening at the latest.

Prof. Thomas seems to have studied the thermometric changes in measles with more care than any previous observer; it will be noticed that his conclusions accord perfectly with those of the older physicians, who laid stress on the fact that "the fever does not, as in smallpox, cease, nor even abate, upon the emergence of the eruption, but sometimes increases in intensity" (Watson). The rash itself first appears on the face; Ringer says that the earliest traces of it are seen on the forehead close to the scalp. It spreads over the face (not avoiding the parts about the mouth) and the trunk; on the limbs, especially the lower limbs, it generally comes out rather less freely, but it shows no decided predilection for the flexor rather than the extensor surfaces, and it may be well marked upon the palms and the soles. In several of these minor points it differs from the eruption of scarlet fever. The rash commonly takes three days for its complete development, but sometimes not more than a few hours; the later it is in beginning, the more quickly it diffuses itself over the body. Thomas says that before it has existed at any one spot for twenty-four hours it always begins to decline, so that when it comes out slowly it fades upon the face and neck, while it has still to make its appearance over the more distant parts; but according to Watson it may remain three days at least on the face before its subsidence commences. The old doctrine that a rapid retrocession of the rash was apt to be associated with the onset of a dangerous complication is disputed by Thomas, in common with many other modern writers. On the other hand, he says that there is sometimes a short-lived recurrence of it when from any accidental cause there is an exacerbation of the fever.

The colour of this rash is a more or less deep rose or crimson, inclining to purple rather than to scarlet. It consists of spots of irregular form, of all sizes up to that of a split pea. They are at first isolated, but afterwards coalesce into patches, the margins of which are sharply defined, and here and there present rounded notches, or (to use the current expression) are "crescentic" in outline. They are slightly raised; indeed, even the earliest papules can be distinctly felt with the finger, corresponding, as Thomas believes, with the hyperæmic mouths of sebaceous follicles. However, he himself alludes to the investigations of Gustav Simon, who, having excised a portion of the skin from a patient affected with measles, examined it histologically with negative results, so that he could only attribute the swelling to hyperæmia of, and serous exudation into, the tissues. When there is much sweating a few vesicles of miliaria are sometimes to be seen. Before it subsides, the eruption acquires a yellowish tint, which is particularly well marked when the blood-vessels are temporarily emptied by the pressure of one's finger. This is no doubt the result of the diffusion of altered blood-pigment in small quantity into the substance of the cutis; for even in cases which are doing well, it is no uncommon thing for actual hæmorrhage to occur; so that, after the rash has faded, purple stains remain, which afterwards become brown and yellow, and do not finally disappear for two or three weeks. Desquamation takes place to a slight extent, especially from the skin of the face. No large scales are ever detached, but only a fine mealy powder, which (as Trousseau remarks) is often best seen when one brushes the skin of the patient with one's coat sleeve, or (according to Ringer) when the surface is stretched and viewed sideways. This writer says that it begins on the sixth or the eighth day, and continues for eight or ten days.

Varieties.—In many cases, however, measles runs an irregular course, and, like those of other exanthemata, its aberrant forms are of two kinds: some *benign*, being shorter and more favourable than usual, others *malignant*, attended with very grave symptoms, and ending more or less rapidly in death.

Each of these, again, has to be subdivided into two varieties at least. In one form of benign measles there is a rash, but no catarrh ; in another it is the rash which is absent. Of *morbilli sine catarrho*, Thomas remarks that it is especially apt to occur in very young infants, and that it is almost unattended with fever. There is reason to suppose that many cases of rötheln were formerly assigned to it, notwithstanding the slight affection of mucous membranes which accompanies the exanthem in question. Perhaps this is one reason why Sir Thomas Watson says that the incomplete form of the disease confers no immunity against recurrence. In other cases, as he goes on to say, the failure to protect is shown by the fact that the patient is soon afterwards seized with regular measles, a true relapse probably, such as may take place after other mild varieties of the complaint. Dr Eustace Smith, however, thinks it questionable whether *morbilli sine catarrho* is measles at all.

Of *morbilli sine morbillis*, Thomas observes that this form is in all likelihood diagnosed more often than it really occurs : but surely the only cases in which one would think of suspecting its presence would be if during an epidemic an unprotected person should suffer from catarrhal symptoms without any apparent cause, or should even go through the ordinary prodromal stage and then get well. Some writers have asserted that, as in the case of latent scarlet fever, all doubts may ultimately be removed by the occurrence of desquamation, but this being so slight even when the affection of the skin is intense, one would hardly expect to see it when there has been no eruption at all.

Of the malignant varieties of measles, there is one which is attended with hæmorrhages from the mucous surfaces, and with an intensely *purpuric* form of eruption. At the present day, however, this is very infrequent : it is observed chiefly in young and sickly children. Thus, when one finds the older writers laying great stress upon "black measles," one is almost inclined to suspect them of having mistaken cases of hæmorrhagic smallpox for this disease ; and it is interesting to notice that Sydenham speaks of an unusually bad kind of measles as prevailing in London in 1670 and 1674, at a time when variola also was remarkably malignant and fatal. The hæmorrhages are said sometimes to begin before the ordinary morbillous rash comes out, sometimes afterwards, in which case it quickly fades or turns of a livid purple colour. Petechiæ and vibices cover the skin, while blood oozes from the mucous membranes of the nose, kidneys, intestines, uterus, as well as into the substance of the deeper tissues and internal organs. Death generally takes place within a very few days.

In other instances measles becomes fatal by the severity and prolongation of symptoms which are not in themselves unusual. The fever is from the first intense and persistent ; the rash, although it may come out early, is generally of a livid purple colour, and often develops itself very imperfectly ; instead of the temperature falling on the eighth or ninth day, it remains high throughout the second week ; the pulse is very rapid and feeble, the patient becomes delirious and drowsy, and passes into a "typhoid" condition with a dry brown tongue and sordes on the lips. Death is ushered in by prostration and collapse.

One of the most important clinical uses of the thermometer is in drawing

attention to the fact that cases of this kind deviate from the ordinary course of measles, and, according to Thomas, it is no less unfavourable for the temperature to be low when the eruption is coming out than for it to continue high at a time when it should be falling.

Complications.—However, it is a rule to which there seems to be scarcely any exceptions, that in all but the most rapid among fatal cases of measles some *complication* or other is really the cause of death. Even when the symptoms are not sufficiently marked to lead to its recognition during life, a local lesion is sure to be discovered at the autopsy; and in cases that recover similar affections are very frequent, and often seriously modify the symptoms.

Broncho-pneumonia, with consolidation of scattered lobules throughout the lungs, is the most frequent complication, particularly in certain epidemics; indeed, it occurs so often that some writers have been disposed to include it in the regular course of the disease. But this cannot be admitted: and since the effects of catarrhal inflammation of the pulmonary tissues are the same as when a like affection arises from other causes, I may leave it to be described elsewhere. In some instances a lobar pneumonia is found, and sometimes, perhaps, there is simply a capillary bronchitis. Collapse usually follows in young children.

Another very dangerous complication is the formation of a layer of plastic exudation upon the fauces or within the larynx, a secondary "membranous croup" or "*diphtheria*," according to the view which is taken of such morbid changes. Again, in many cases *diarrhœa* becomes so severe as to bring about a fatal issue, especially when the evacuations assume a dysenteric character. I believe that little more than redness of the intestinal mucous membrane is found under such circumstances.

Epistaxis is not infrequent, and occasionally serious. Among the less serious complications, so far as the life of the patient is concerned, must be mentioned certain affections of the eyes and ears. *Ophthalmia* is common, and often runs on for a long time after recovery from the exanthem. I have once seen a diphtheritic membrane form again and again on the conjunctivæ. Sometimes iritis occurs, and sometimes a destructive corneitis. In other cases the morbid action extends from the fauces along the Eustachian tube, causing *deafness* and pain in the ear. As a rule, it seems merely to lead to an accumulation of mucus in the tympanic cavity; but now and then suppuration takes place, and even necrosis of part of the temporal bone. Trousseau relates one instance of this, and another has come under my own observation.

Sequelæ.—Almost any of the complications of measles may be so prolonged as to have a fair claim to be regarded as sequelæ, but there are some other affections which have a still better right to that title, since they do not begin until after the patient has recovered from the primary disease. Among these is a form of gangrene, attacking the mouth (*cancrum oris*) or the female genitalia, and commonly known under the name of *noma*. According to Thomas it does not arise spontaneously, but is preceded by some slighter lesion of the same parts, such as a decayed tooth, or an infantile leucorrhœa respectively. It is generally, but not always, fatal. In other cases measles is followed by *necrosis* of a portion of the upper or lower jaw, or by abscesses in the neck.

Again, children often remain for many months after recovery in a state of ill-health, for which no obvious cause can be found, and during this time they are very apt to be seized with bronchitis, or even by lobar pneumonia,

especially in the cold seasons of the year. The swollen bronchial lymph-glands may become caseous, and thus *tuberculosis* is not unfrequently set up. Indeed, this is one of the commonest and most fatal evil results of measles. An intercurrent attack of measles is said greatly to accelerate the downward course of a pre-existing phthisis. On the other hand, certain cutaneous diseases, such as eczema, seem often to be favourably modified by the supervention of the exanthem, at least for a time; and, according to Rilliet and Barthez, the same thing happens in some cases of epilepsy, chorea, or incontinence of urine.

Dr Goodhart finds that whooping-cough is often a sequel of measles, more often than its predisposing cause.

Protection.—Measles often fails in preventing a second attack. Even relapses are not unknown, and second or third invasions in a lifetime are as common as with scarlatina. It is more severe in adults than in children.

Diagnosis.—This depends on the late appearance of the eruption, the coryza, and the colour, distribution and form of the rash. It has been mistaken for smallpox, but the symptoms are far less severe, the papules look and feel different, and the non-development of pustules decides the point. It is most frequently mistaken for scarlatina, but the eruption is later, it is purple not scarlet, blotched not punctate, and appears first and is most marked on the face. It is often confounded with Rötheln or Rubeola notha, to be described in the next chapter.

Prognosis.—The mortality of measles varies widely in different epidemics. It has sometimes been so low as 2 to 3 per cent. of those who have been attacked, sometimes as high as 50 per cent. Malignant cases killing within the first week by prostration, with or without a hæmorrhagic rash, are rare—much rarer than the corresponding malignant scarlatina. Petechiæ are, however, often seen in ordinary benign cases. Trousseau relates that in 1845 and 1846 he lost from broncho-pneumonia twenty-two out of twenty-four children under his care in the Necker Hospital. According to Thomas the few cases which occur during the first six months of life are generally mild; but with these exceptions the disease is more severe in infants than in older children, and especially during dentition. Thomas says that he has several times found teeth just protruding from the gums when death had been preceded by cerebral symptoms. Convulsions, occurring after the rash has appeared, are of ill-omen.

Among adults, measles is most dangerous to women who are pregnant, or who have recently been confined, and to the very few old people who are susceptible of the disease. It is, however, almost always more severe in grown persons than in children.

Treatment.—The general plan of treatment is the same as in scarlatina, for in neither have we any specific method of dealing with the malady itself. Apart from the febrile condition the coryza must be managed as a bad cold and cough of common origin. A few drops of ipecacuanha wine and paregoric, with syrup of tolu, are better than senega and ammonia. Poultices may be used to the chest, but with little children a cotton-wool jacket is better, since it interferes less with breathing and does not get cold or slip off. Stimulants are occasionally needed. During convalescence quinine is generally found a better medicine than steel.

RUBEOLA*

Recognition and nomenclature—Its characteristics—Discrepancies and probable explanation—Incubation—Onset and course—Diagnosis—Prognosis.

HARDLY had scarlet fever become universally recognised as distinct from measles, when physicians in Germany began in the latter half of the last century to try to isolate a third member of the same group of diseases, and adopted for it the term "Rubeola," for which they had no other use, since they adhered to "morbilli" as the Latin name for measles. And ever since a controversy has been smouldering in that country as to the existence of this complaint, which is commonly known there as Rötheln, the corresponding term for measles being Masern.† But in France and in England the question has until lately attracted little notice, one reason being, in all probability, that the word rubeola was otherwise appropriated. Dr Paterson, of Leith, however, described the supposed third exanthem in the 'Edinburgh Medical Journal' for 1840, and among English writers who have since expressed their belief in its existence may be mentioned Copland, Aitken, Squire, Bristowe, Murchison, and Robert Liveing. A suitable name has yet, I think, to be discovered. Copland and Aitken, having reverted to the use of morbilli for measles, call it "rubeola." The term "epidemic roseola," adopted by Squire and by Bristowe from Trousseau, would naturally convey the impression that the disease bears some relation to the other eruptions which have been known as species of roseola since the time of Willan.

On the whole it seems best to use the word Rubeola. Measles in English, and *morbilli* in Latin, are satisfactory names, distinctive and unambiguous. It is a pity to lose a word which has already been applied to a disease that wants a name, and which, if not so used, must be abandoned altogether. Moreover Rubeola is the accepted equivalent of Rötheln with German authors.

Rubeola, then, is a specific and infectious febrile disease, occurring in epidemics, arising from contagion and breeding true, accompanied by an exanthem, and protecting against itself.

Its most essential features are that with a rash like that of measles, it resembles scarlet fever in having a very short prodromal stage and in being attended with a marked sore-throat.

A favourite idea with some of the older writers (Schönlein among others) was that it was a "hybrid" between these two diseases; they even spoke of the symptoms as alternating in different cases, so that sometimes the rash of measles would be associated with the local lesion of scarlet

* *Synonyms.*—Rubeola notha, bastard measles (Babington); German measles; Rubella.—*Fr.* Roséole epidémique, *Germ.* Rötheln.

† Rötheln is a local or dialectic name for ordinary measles in many parts of Germany, according to Seitz.

fever, whereas sometimes the reverse would be the case. But this was purely fanciful, and whatever may be said as to the possibility of the specific causes of two exanthems combining so as to produce in a single patient a modified affection, one can scarcely conceive that such an affection should be epidemic, still less that its contagion should protect against the recurrence of itself, while affording no immunity against either of the constituent diseases.

In the first edition of this work, the author contrasted the account given by Dr Paterson and accepted by Aitken and other systematic writers with that of German authors, and especially with Prof. Thomas's article in Ziemssen's 'Cyclopædia,' based on two epidemics at Leipzig in the years 1868 and 1872. In the former account we read of marked disturbance at the invasion, high fever, severe catarrh, with œdema of the glottis, bronchitis, severe angina with dysphagia, and a raised blotched eruption appearing on the third or fourth day. Death was not infrequent from suffocation or from convulsions.

The discrepancy of this account from those given by Thomas and other German writers must be admitted, and I think that the probability is that Paterson's epidemic was not rubeola at all, but measles—occurring perhaps after a long interval of freedom in the locality. I remember when a student at Guy's Hospital that the crew of a Peruvian vessel in the Thames were attacked by measles, and the patients were taken into our wards. The disease was exceedingly severe, and several died from it, in almost every case from bronchitis or pneumonia. The same unusual malignancy marked the introduction of measles into the Fiji Islands.

However this may be, there can be little doubt, as it appears to the Editor, that the disease described by Niemeyer, Thomas, and other German writers, by Murchison, Bristowe, Liveing, Squire, Cheadle, Eustace Smith and Goodhart,* is one and the same, different both from measles and from scarlet fever.

Rubeola occurs in the British Isles and on the Continent, in Egypt, in India,† and in America.

Origin and incubation.—All writers agree that rubeola arises strictly by contagion from other cases. The most important predisposing cause is age; it is more frequent about puberty, is rare in young children, and not common in adults. Boys are said to escape in a family oftener than girls.

The incubation stage is generally said to be somewhat shorter than that of measles. Bristowe says about a week, sometimes five days only; Squire about ten days; Liveing ten to fourteen days; Douglas fourteen or fifteen days, never more, and rarely less; while Thomas gives a fortnight or even three weeks. Dr Clement Dukes, whose opportunities as medical officer of Rugby School were exceptional and exceptionally used, states that out of twenty-four carefully-observed cases, the incubation lasted twelve days in two and from fourteen to twenty-two in the rest. The infection is apparently less active than that of measles and less persistent than that of scarlet fever, for more escape in a house or school during an epidemic of rubeola than during one of measles, and cases seldom occur after an interval of cessation. Nothing

* I may also refer to a succinct but full account of an epidemic of fifty cases by Dr Douglas, of Newbury ('Brit. Med. Journ.,' May 26, 1877), and to a short paper by Dr Ryle (ibid., July 24, 1886).

† Surgeon-Major McLeod read a paper before the Epidemiological Society (Feb. 11, 1885) on an epidemic rose-rash, which visited Calcutta in 1881, and which he rightly decided to be Rubeola.

is exactly known of the locality, the structure, or the mode of conveyance of the contagion.

Onset and course.—The first symptoms are usually not severe. There is little or no catarrh before the rash, and the fever is not high. The eruption appears early; often it is the first symptom noticed, and it is probably never delayed beyond twenty-four hours after the temperature rises. It is more like the rash of measles than that of scarlatina, but its colour in the cases I have seen is a brighter rose; it is patchy, but less mottled and more diffuse than measles, less vivid red, less diffused and less punctate than scarlet fever. Its distribution is earliest on the forehead and cheeks, and the trunk, particularly the back. It is not so markedly developed on the face as measles. The upper arms are affected, the nates and the thighs, the hands and feet rarely. It spreads more rapidly and less regularly than the other two exanthems.

The eruption lasts three days or more, usually longer than measles, but not so long as scarlatina. Desquamation is present, but needs careful looking for, and never resembles that of scarlet fever.

With the rash there is, in probably every case, angina; the fauces are injected and swollen, but there is never sloughing, and the lymph-glands at the angles of the jaw are usually not enlarged. On the other hand, the glands which run along the hinder border of the sterno-mastoid muscle can often be felt. There is lacrymation with some photophobia, and usually slight nasal catarrh, accompanying but not preceding the exanthem. In exceptional cases this is followed by bronchitis. But catarrhal symptoms may be absent, and they are rarely so marked as in even mild cases of measles.

Pyrexia on the second day is sometimes rather high, with quick pulse, restlessness, and occasionally delirium. It subsides with the rash on the fourth day.

Traces of albumen are often to be found in the urine, and Dr Liveing has in exceptional cases seen it persist and anasarca follow. The tongue is moderately furred, and does not resemble that of scarlatina. The pyrexia rapidly subsides as the exanthem fades. Dr Douglas informs me that, in an epidemic in 1883–84, he twice saw "rheumatoid" synovitis affecting the wrists and lasting three or four days.

Sequelæ appear not to coexist. "If after an attack of German measles a child remains thin and feeble, or has a discharge from the ears, the disease was either scarlatina or measles" (Goodhart).

Diagnosis.—The first cases of an epidemic may often be doubtful, but after seeing a few the eye recognises the different aspect of the rash. Between slight cases of scarlatina and rubeola the distinctions are often slight—the desquamation in the former case is probably the most helpful point to look to. The early appearance of the exanthem, usually with the fever, is the best distinction from measles.

It is often difficult to distinguish rubeola from "ordinary rose-rash," whether set up by gastric disturbance or by definite poisons, or caused by local irritation. Dr Dukes says that the roseola produced in some persons by handling the hairy larvæ of certain moths (the "woolly bears" which schoolboys collect) is sometimes exactly like rubeola. There is no doubt of the existence of a generally diffused, bright red erythema, which, though not contagious, appears in groups of cases in hot weather or under unknown conditions. The name roseola is suitable for this, and it is distinguished from true erythema multiforme (to be described in the second volume under

Diseases of the Skin) as well as from rubeola (notha), from morbilli and from scarlatina. I mention it here because there is no doubt, from the absence of catarrh and sore-throat, from the severe itching, and from the proneness to relapse, besides many other characters, that this affection, graphically described as Roséole by Trousseau ('Clinique Medicale,' tome i, p. 161) and as Erythème scarlatiniforme by Hardy ('Leçons sur les Mal. de la Peau,' 2me partie, p. 35) is not—as the late Dr Tilbury Fox ('Skin Diseases,' p. 93) and other writers have supposed—identical with the rubeola of German authors. Probably the papules described by Vogel and Borsieri as *ardentes et prurientes*, and named Essera Vogelii, were of the same nature. Essera is defined by Blanchard ('Lex. Med.,' ed. 1702) as *tubercula parva, ad rubrum vergentia, duruscula, cum insigno pruritu subito universum corpus occupantia.*

Beside two or three scattered cases which I have seen, there was an epidemic among the nurses at Guy's Hospital a few years ago which satisfied, I think, all who saw the cases of the reality of the disease. The cases differed considerably in severity and in the character of the rash. Sore-throat was always present, and catarrh much less frequent, so that it was very distinct from measles, and the absence of the characteristic signs of scarlatina and of any severe cases prevented confusion in that direction. Some of the mildest cases, when the patients were about, and therefore the rash not obvious, might have been set down as "epidemic sore-throat."

Second attacks are rare, probably rarer than in the case of true measles. Statements to the opposite effect no doubt refer to epidemic roseola, not to Rubeola. It does not protect from measles or from scarlatina. Among sixty-three cases of rubeola seen by Dr Dukes, thirty-nine had previously had measles. Among thirty cases observed by Dr Cheadle, twenty-two had had measles, and ten of these cases had been under his own care. There seems no reason to suppose that measles predisposes to rubeola, but measles attacks as a rule younger, and rubeola older children. There are, however, several instances of an epidemic of rubeola following one of measles.

Prognosis.—Most authors agree in saying that in epidemics much more severe cases than the rest may occur, and that death may occasionally happen. If, however, we exclude the description by Paterson, these cases must be extremely rare. A favourable prognosis may probably always be given.

No special *treatment* is necessary. The patient need not in many cases be confined to bed after the nature of the complaint is clear, but he should be secluded from others for a fortnight, if infection is to be prevented.

SMALLPOX*

Incubation—Onset—Early rashes—Specific eruption—Varieties of the Exanthem—Course and symptoms—Discrete, confluent, and modified Smallpox—Complications and sequelæ—Prognosis and treatment.

VACCINIA—*Inoculation—Introduction of vaccination—Course of the eruption—Its protective power—Its drawbacks—Its relation to Variola.*

THIS terrible disease—before the introduction of vaccination the most fatal of all epidemics—was first described by Rhazes about 900 A.D., under the name *Jadari*, translated into λοιμική, *i. e.* the pestilential eruption, by the Greeks. It is, in some respects, the most typical of the class of exanthemata.

The *incubation* of smallpox is, as a general rule, twelve days. On the thirteenth day from that on which the contagion entered the patient's body, he is seized with more or less severe symptoms of malaise and fever. Most writers allow that the interval may sometimes be longer; Curschmann extends it in exceptional cases to thirteen, Bristowe to sixteen days; while Marson affirms that he at least has never found the eruption fail to appear in fourteen days after infection. He admits, however, that the incubation is sometimes shortened by three or four days, and Curschmann alludes to one instance in which it did not last more than five days. Zülzer believes that in the hæmorrhagic form of variola it is constantly from six to eight days. During this period patient generally feels well; but occasionally he complains from the very first of a vague malaise, with gastric disturbance, headache, and giddiness, and towards the end there may be a little pharyngeal catarrh, with reddening of the uvula and tonsils.

When inoculated the incubation was somewhat shorter, usually ten days, but even then there were slight occasional variations in time; eleven days was not uncommon.

Prodromal stage.—This usually sets in with a severe rigor or with a succession of slight chills. The temperature at once begins to rise, and may reach 102° to 104° within twenty-four hours, and 105° or even 107° by the second day (*fièvre d'invasion*). In some cases the patient becomes rapidly prostrate, and totters if he attempts to walk or to stand. Curschmann remarks that whereas a working man attacked with enteric fever will often come on foot to the hospital at a time when his temperature is already high, one who has smallpox must be carried. Such a patient, while he is out of bed, has cold limbs, a pale sunken countenance, and a small pulse; so that he is hardly to be recognised a few hours later, when he has become warm,

* *Synonyms.*—Variola.—*Scottice*, the Pocks, *Fr.* la petite Vérole, *Germ.* Blattern, Menschenpocken, *It.* Vajuolo, *Mod. Gr.* Εὐλογία, originally coined as a translation of the Arabic *Hasbah*, *i. e.* measles. The English word refers to the pockets or little pocks, pokes or bags, in which the matter is contained, and so marks the pustular form of the eruption. It also marks the distinction between this and the still more dreaded Great Pox, *i. e.* Syphilis. The Latin word *Variola* is a diminutive formed from *Varus* (with a short *a*, although this did not prevent a Ciceronian pun between it and *varius*), a pustule or pimple, used as a translation of ἴονθος. There is no classical word for smallpox, since the disease was unrecognised by the ancients.

and when his face is red and turgid. The pulse is much accelerated, varying from 100 to 120 in men, while in women it may reach 130 or 140, and in children 160. The breathing is quick, short and laboured. The skin is generally dry, but sometimes moist or even sweating. Some medical men, among whom was the late Mr Stocker, believe that it already emits a peculiar odour. There is no doubt that the breath is fœtid, and the tongue is often very foul. Anorexia and thirst are present. The patient almost always complains of pain across the forehead or over the whole head; and this is sometimes intense. Children are not infrequently attacked by epileptiform convulsions; or they may become delirious or comatose. They often have diarrhœa; but in adults constipation is usual. In the more severe cases enlargement of the spleen may be detected.

So far, there is little to distinguish the early period of variola from that of other fevers. A more characteristic symptom is *vomiting*, attended with violent retching and pain at the epigastrium, and sometimes so severe and persistent that a case has been sent up to hospital as one of ileus. Another symptom, still more characteristic, is *pain in the sacrum and loins*. This, however, is not constant; Curschmann says it occurred in rather more than half of his cases, and that in only one third of them was it sufficiently intense to give rise to spontaneous complaints. It appears to be most frequent in severe cases. Women are apt to suppose that it merely indicates the approach of menstruation; or, if they are pregnant, that labour is about to set in. And, as a matter of fact, the catamenia do frequently appear during this stage; sometimes prematurely, but often in their regular course. In men the pain in the loins is likely to be mistaken for that caused by gravel or some other affection of the kidney, or for lumbago.

Another set of symptoms, which are of importance because they may cause the disease to be mistaken for measles, are those of common *catarrh*, sneezing, epistaxis, intolerance of light, lacrymation, sore-throat, and hoarseness. The tonsils and the palate may be seen to be reddened.

Lastly, the initial stage of smallpox may be attended with two distinct forms of cutaneous rash, which differ in their characters and still more in their significance.

Early roseola.—One of them is, I think, best described as *roseola variolosa*, a name employed for it long ago by Rayer, and in 1853 by Eimer. To attempt to trace back the history of the knowledge of this remarkable eruption is, indeed, almost useless, because until recently most observers regarded it as a proof that one of the other exanthemata was present, in addition to variola. Reinbold, in 1840, seems to have originally propounded in express terms the doctrine that it is a preliminary symptom of that disease alone. In this country Dr Wilks, I believe, deserves the credit of having first drawn attention to it, in the 'Guy's Hospital Reports' for 1857 and for 1861. Watson, however, also mentions it in 1857. But Simon, of Hamburg, is the writer who has most fully described it, and has recorded the largest number of cases. As a rule, it comes out on the second or the third day; but sometimes it immediately follows the initial rigor, and occasionally is the earliest sign that anything is amiss with the patient. There seem to be two varieties of this early roseola. One is maculated, and is more or less like measles; it may come out all over the body, perhaps appearing first upon the face. The other is an evenly diffused red blush, resembling scarlet fever, but (according to Simon) darker and of a more purplish tint. This often has a distribution peculiar to itself, which was, I think, first pointed out by Hebra:

it is then limited to a triangle having for the base a horizontal line drawn across the abdomen about the level of the umbilicus between the two iliac spines, while the apex is formed by the contact of the two thighs. Or it may also be visible in the axillæ, and on the adjacent parts of the upper arms and of the chest. And on the limbs it may affect the extensor surfaces of the elbows and of the knees, the backs of the hands and fingers, as far as the first phalangeal joints, and the back of each foot in a line corresponding with the course of the extensor tendon of the great toe. Its occurrence in the pubic and inguinal regions is so characteristic that it enabled me several years ago to diagnose correctly a case in which there was no other reason to suspect smallpox. Even if no other eruption should afterwards develop itself, one ought probably still to maintain such a diagnosis, for Simon has recorded an instance of this kind in which the disease aborted, notwithstanding that its real nature was proved by the patient's sister suffering from an attack of variola at the same time. Sometimes, however, the seat of *roseola variolosa* is irregular. We have in our museum at Guy's Hospital models of a case in which it affected only the flexor surfaces of the forearms; and in women it may be limited to the skin about the nipples. It often lasts for a day or two and sometimes not longer than a few hours. But in a great many cases it assumes a petechial form, and leaves green or brownish stains which may remain visible the whole course of the disease. A point of importance is that, even when it thus becomes hæmorrhagic, the prognosis need not be modified in an unfavourable direction. Hebra and Trousseau seem to have independently remarked that the parts affected by *roseola variolosa* afterwards remain free from the proper smallpox rash. All subsequent observers have confirmed this statement, reserving, however, some few cases as exceptions to it.

Early purpura.—Very different is the second form of initial eruption, which in fact constitutes the most fatal of all the varieties of the disease,—the *Variolæ nigræ* of older writers, now generally known as *Purpura variolosa*, *Malignant smallpox*, or *Hæmorrhagic smallpox*.

After the usual early symptoms have been present during from eighteen to thirty-six hours there appears on the trunk and the limbs a diffused scarlet redness, which at first can be made to disappear by pressure with the finger. This soon becomes the seat of most extensive effusions of blood, in spots and patches of all sizes and shapes. Large black rings now form round the eyes; the conjunctivæ are ecchymosed (this, according to Marson, is often seen very early in the case and is always a most dangerous symptom); the whole face may be red and swollen. The tongue is thickly coated. The breath has a horrible fœtor, from the presence of a diphtheritic affection of the throat. Hæmorrhages may occur from any or all of the mucous surfaces, Bloody liquids are coughed up or vomited, or discharged from the uterus or the bladder; or there may be epistaxis, or a sanguineous flow from the eyes or the ears. The urine is extremely fœtid, and it is albuminous even when it contains no blood. The patient generally complains of severe pain at the præcordia, and suffers severely from vomiting. Yet, according to Curschmann, the temperature is seldom over 104° until just before the fatal termination. Both he and Marson remark that consciousness is generally retained almost to the very last; "few patients are so fortunate as to become quickly delirious or comatose." Anæsthesia or hyperæsthesia of certain parts of the surface, and paralytic affections of the limbs, are said to have been observed by Zülzer. Before death the whole body becomes black or of a leaden grey

colour. According to Marson, the smallpox eruption is nearly always confluent in cases of this kind; but they often end too rapidly for such an affection to appear. I have, however, seen at least two or three instances in which, although the disease was prolonged for several days, not even papules could be discovered. The first case that impressed this fact upon me was one which occurred in the clinical ward of Guy's Hospital. The patient lived several days, and as no sign of a proper smallpox eruption could be detected, I came to the conclusion that it was not an example of *purpura variolosa;* but a few days afterwards the clinical clerk who watched the patient fell ill with a mild form of smallpox. It has often been remarked that strong muscular men are particularly apt to be attacked with fatal hæmorrhagic smallpox, but it is also seen in drunkards, and in women recently confined, or in those who are pregnant, and who of course at once abort. According to Marson, the blood in this variety of smallpox is "poisoned from the very first, and is rendered very fluid and watery." German pathologists, however, lay some stress on the fact that in such cases the liver, the spleen, and the kidneys are found after death to be neither swollen nor softened, and that the substance of the heart is firm and of a dark red colour.

Slight and abortive cases.—Hitherto I have said little or nothing about variations in the intensity of the symptoms during the normal initial stage. Occasionally they are very slightly marked, or even absent; the smallpox eruption then appears after a few hours' malaise, or is the first sign that the patient is ill. In such cases the disease itself is always very mild; so that Trousseau is not quite right in saying that the greater the duration of this period the less serious is the attack. Unlike a slight initial stage, one which is severe is no guide as to the ultimate progress of the case. In women and in children it often happens that the most alarming symptoms are present during the first day or two, notwithstanding that the rest of the disease is to be of a mild character.

The most extreme instances of this are afforded by a form of smallpox which was long ago described by Sydenham as *Febris variolosa*, and by de Haen as *Variolæ sine variolis.* After a well-marked initial stage the disease aborts, and the patient is well in three, four, or six days at the latest. I have already referred to one instance of this kind, which was attended with a characteristic roseola. Another example of it is thus recorded by Marson. A lady walked with a person already affected with smallpox. Twelve days afterwards she was taken ill; she was for a few hours delirious, but her illness passed off without eruption; twelve days later still, her sister, who had not been out of the house for three months, was attacked with the same disease, which ultimately assumed a confluent form. Curschmann tells of a woman who was seized with shivering, fever, headache, and pain in the back, so that, as variola was epidemic at the time, she seemed without doubt to be passing through the initial stage; but on the fourth day defervescence occurred, no rash could be detected, and by the tenth day she felt perfectly well; however, she gave birth to an infant, which was covered with the eruption of smallpox.

The eruption.—As a rule, the *third* day is that on which the eruption comes out; but in children it is often the second day. Sometimes, however, nothing is to be seen until the fourth day; and then the prognosis is comparatively favourable. A postponement until the sixth or the seventh day

seems to occur only when there is some complication, as in a patient of Trousseau's who had choleraic symptoms.

The proper variolous exanthem is first papular, then vesicular, and finally pustular. The papule is round and smooth ; at first it may be scarcely redder than the rest of the skin ; and as it is hard, like a small shot, it can often be felt better than it can be seen. But after twenty-four hours it is always decidedly reddened, besides having increased in size. Unlike the papule of measles and of most other exanthemata, it depends upon a definite change in the superficial and middle cells of the rete mucosum, which from the very commencement of the morbid process are swollen and opaque. And in their midst exudation quickly takes place, so that by the end of two days the horny layer of the epidermis is raised to form a minute conical vesicle. By the fourth or fifth day of the eruption (seventh or eighth of the disease) the vesicle is generally as large as a split pea, hemispherical in form, and opaline in appearance. As a rule, the *pock*, as it is termed, has a central depression or umbilicus. The origin of this has been much discussed ; it generally seems to depend upon the fact that the original papule was developed round the mouth of a hair-sac, or else round that of a sweat gland, either of which structures may afterwards form a *retinaculum*, tying down the roof of the vesicle in the middle. Rindfleisch gives a drawing of a preparation in which a sudoriparous duct is plainly seen in this relation to the pock, and he says that he has many such specimens in his possession. However, it would seem that this explanation is not always applicable, since the pock does not necessarily bear a definite relation to any of the canals which traverse the cuticle. In all probability a similar function may be discharged by one of many other bands which cross the upper part of every vesicle in a direction more or less vertical, dividing it into a number of separate chambers. This *loculated* character of the pock attracted notice long before its nature was understood ; it affords the reason why only a small part of the fluid is evacuated when a needle is introduced into the roof at a single spot. But the idea of the older observers was that the septa between the chambers were new formations, consisting of fibrin. Sir Thomas Watson, for instance, speaks of a "central whitish disc," consisting of "several little cells ;" and this, he says, may by careful dissection be taken out entire with the "lymph" which it contains, even when the rest of the fluid in the pock has become yellow and purulent. Auspitz and Basch, however, showed several years ago that all the septa in question are in reality formed out of the original cells of the rete mucosum, small bundles of which cohere together, and become stretched out into filaments and bands, as the exudation accumulates around them. In this fluid leucocytes are present in small numbers from the very first ; they go on increasing, and thus transparent serum gradually passes into opaque pus ; the change is complete in about six or seven days from the first appearance of the papule,—that is, in the earliest part of the eruption, by the ninth or tenth day of the disease. The pustule, when at its height, is often hemispherical, the umbilicus having disappeared in consequence of the rupture of the retinaculum which formed it.

But while the roof and the cavity of the pock are thus being developed out of epidermic structures, a morbid process is at the same time going on more or less actively in its floor, which consists of the papillary layer of the cutis, with at least the lowest cylindrical stratum of the rete Malpighii. Whether a swollen state of the papillæ has any share in the formation of the original papule appears to be doubtful ; but there is no doubt

that its redness is due to their hyperæmia; and (according to Bärensprung) this extends down through the whole thickness of the skin. The exudation which fills the vesicle, and afterwards the pustule, is necessarily derived from these vascular tissues. But Curschmann says that, so far from the papillæ being always enlarged at this stage, they are often rather flattened by the pressure to which they are subjected. In some cases, however, they become the seat of an infiltration of leucocytes, which is so intense that it obliterates their blood-vessels, and, indeed, destroys their structure completely, converting them into a white or ash-grey substance. German histologists, in accordance with their usual terminology, describe this form of pock as *diphtheritic* (*vide supra*, p. 62). The contrast is the greater because the surrounding skin is of a bright red colour for a considerable distance, making what is termed the *halo* or *areola*. The infiltration of leucocytes into the floor of the diphtheritic pock may extend to a varying depth in the derma, or even through it into the looser tissue beneath. Rindfleisch gives a drawing from an injected preparation, in which the affected area had failed to receive any of the colouring matter.

Many of the pustules of smallpox undergo destruction almost as soon as they are fully formed. They break, or are ruptured, giving exit to a honey-like matter, which collects in drops upon the face or other exposed parts of the patient's skin, and saturates his shirt, his pillow, and his sheets. Others, however, remain uninjured, and ultimately dry up without discharging their contents. First a yellow-brown spot appears in the roof of the pock; this sinks in, so as somewhat to resemble the earlier umbilicus; gradually it enlarges and extends to the periphery. The process of desiccation, both in ruptured pustules and in those which are entire, begins about eight or nine days after their first appearance in a papular form. The crusts vary in colour from yellow to brown, or even black, as more or less blood is mixed with the pus of which they are formed. It is now that the extent to which the cutis vera has been involved in the inflammation affects the result. If it has escaped, the crusts fall off in four or five days (about the fifteenth or sixteenth day of the disease), leaving purple-red stains, but little or no permanent cicatrisation. But if pus-cells have infiltrated the papillæ, or the whole thickness of the skin, these structures, to whatever depth they may have been affected, slough away and become detached, as shreds, adhering to the under surface of the crusts. Their separation takes longer, and may not begin until the eighteenth or twentieth day. Moreover, when it occurs, granulating surfaces are exposed which may take a considerable time to heal. The resulting cicatrices are for a time of a brown colour, but ultimately they become whiter than the skin around them. They may either be so faint as to be scarcely perceptible, or more or less deep and pitted. It often is not until many months after recovery from the disease that they become depressed below the surface to the full extent.

Distribution.—So much for the appearance and structure and course of the individual pocks of variola; the characters of the eruption as a whole have still to be considered. Like the rest of the exanthemata, it does not develop itself over the whole of the body at once; it appears first upon the face and scalp, especially on the forehead and about the eyes, the nose, and the upper lip. A few hours afterwards it is to be seen upon the trunk and the arms. Marson mentions the wrists as among the earliest parts affected, and says that on the legs and the feet it is generally two days later than elsewhere. He also lays stress on the fact that the papules come out

"in threes and fives, forming crescents," or even a complete circle if two crescents happen to coalesce. During the first day or two, fresh spots keep appearing, even on those parts which are already more or less thickly covered; but by the end of this time the eruption is complete, for even if a few more should afterwards show themselves they soon abort and die away. A curious circumstance is that smallpox specially affects any parts of the skin which may happen to have been recently irritated; the red patch from a mustard plaster applied during the incubation or shortly before will present many more pocks than the regions adjacent. Curschmann mentions the case of a man who was infested with clothes-lice, and whose body was consequently covered with scratches; on his becoming affected with a mild form of variola, these scratches were found covered with pustules, which were closely arranged in lines, like strings of pearls, some of them three or four inches in length.

Certain *mucous membranes* take part in the eruption of smallpox, but not in quite the same way as the skin. There first appear raised spots, whitish or grey in colour, contrasting with the reddened state of the surface around them; but these quickly pass into excoriations. They are sometimes present in large numbers on the inside of the lips and on the cheeks; sometimes they rather affect the tonsils and the palate, which may then become greatly swollen and the seat of deep phlegmonous inflammation, ending in abscess. On the other hand, the tongue very seldom presents any traces of vesicles, but is sometimes enormously enlarged, protruding from the mouth, rendering the patient unable to close his jaws, and apparently helping to bring about a fatal issue. The air-passages are said to show more or less distinct pustules as low as the bifurcation of the trachea, or even (according to Wagner) down to bronchia of the second or third order; there may also be deep ulcers in the larynx, with perichondritis and necrosis of cartilages, and œdema of the aryepiglottidean folds. In the alimentary canal nothing resembling a pock can be seen below the upper part of the œsophagus, except perhaps in the rectum close to the anus, or at the entrance of the vagina. Marson speaks of twenty-six cases at the Smallpox Hospital, out of a total of over fifteen thousand, in which the conjunctiva showed a single pustule, which was generally situated midway between the cornea and the internal canthus. It did not in any way affect the sight or lead to any ill result.

Varieties in the eruptive stage.—Before I attempt to describe the general symptoms of the eruptive stage of smallpox, I must distinguish certain varieties of the disease which have long been known to physicians by separate names, and the recognition of which is of no little practical importance. Roughly speaking, the severity of variola depends upon the number of the pustules, and in the relation of the pustules to one another we have a criterion which, while it is itself based upon their number, affords a natural division of the most marked kind. Whenever they are so crowded that they run together the disease is said to be *confluent*. This may either be the case over the whole of the body, or only over certain parts of it, or even on but a single region; this is always the face, since the eruption is never more abundant than upon the face. On the other hand, if the pustules remain everywhere distinct from one another the smallpox is described as *discrete*. It will be noticed that what constitutes the boundary line between the two forms is in reality the state of the face alone; I believe that there is very seldom any difficulty in drawing it sharply and decisively. Watson, however,

speaks of some cases in which the pustules stand just thick enough to touch one another without coalescing, and would then call the disease *cohering;* and Trousseau alludes to instances in which a few isolated patches are found confluent, but which he would term discrete.

In ordinary discrete, as well as in confluent, smallpox, the pustules run through all their stages, unless indeed the disease should happen to be cut short by the patient's death. But there is a third variety, consisting partly of discrete and partly of confluent cases, which is characterised by the premature subsidence or abortion of the eruption. For this, unfortunately, there is no satisfactory name. German writers* describe it under the name of "*variolois,*" which is etymologically incorrect; since it is not like variola, but is true, though modified, and capable of generating the severer variola forms by contagion. In this country it is generally termed *modified smallpox,* because it is comparatively seldom seen except in persons who have already passed through a former attack, or who have been vaccinated. But it is important to know that variola sometimes passes off in exactly the same manner in a patient who is altogether unprotected. Probably this depends upon some peculiarity in his predisposition, whereby his susceptibility to the contagious principle is diminished without being annulled. Trousseau mentions a case which goes far to establish such a view. A person had been vaccinated (as I presume, for the first time), but the operation did not take effect. Some time afterwards, when the doctor was thinking of repeating it, he was called to see the patient, whom he found to be suffering from a discrete smallpox, which ran a modified course. I do not know that the *confluent* form of the disease ever runs this modified course in those who have not been vaccinated and who have not previously had variola.

1. In *discrete smallpox* the eruption presents in their most typical forms the characters which have been described as belonging to the individual pustules. Its amount, of course, varies infinitely in different cases, from a few spots that can almost be counted on the fingers up to many thousands. At the time when they are acquiring their areola there is often a considerable degree of swelling of the surrounding parts, especially when the subcutaneous tissue is loose. Even three or four pustules upon the eyelids may cause them to be puffed out like bladders, so that on the ninth and tenth days the patient may be unable to open his eyes. Trousseau cites a case of Van Swieten's in which a single pustule on the prepuce produced phimosis and great difficulty in micturition. He remarks that the absence of a corresponding degree of tumefaction of the rest of the face renders the swelling of the eyelids more conspicuous in some cases of discrete smallpox than in the confluent variety of the disease. At about the same time the pustules first begin to be painful.

As regards the general symptoms of discrete smallpox, the first thing to be noticed is that as soon as the eruption has fairly begun to develop itself, the fever which has existed during the initial stage subsides, and the tem-

* Strictly speaking I think that this is not quite correct. It is true that German writes give *variola modificata seu mitigata* as a synonym for their "*variolois.*" But in practice they confine the use of the latter term to cases of a certain moderate degree of severity, and apply it to all such cases, whatever may be the course of the individual pustules. Thus Curschmann speaks of *variolois* as sometimes leading to destruction of the papilla and to the formation of deep cicatrices, "so that what distinguishes the case from one of *variola vera* is merely the very much smaller number of pustules." We should call such a case one of very mild discrete *un*modified smallpox. On the other hand, I do not find German writers mentioning under *variolois* the examples of modified confluent smallpox which will presently be described.

perature falls within thirty-six hours to normal, or even lower. Its decline is not always quite continuous, being perhaps interrupted by a slight evening exacerbation. At the same time the pain in the back and the sickness disappear, and the patient often feels perfectly well, with as good an appetite as when in health. This lull in the symptoms commonly lasts for three days; at the end of it he is, in all cases except those in which there are but very few pustules, again attacked with shivering and with febrile disturbance, which is known as the *secondary fever*, or the *suppurative fever*, or the *fever of maturation*. The date at which this sets in seems to be differently stated by writers; Trousseau puts it at the seventh or eighth day of the disease, Watson at the eleventh. It is generally remittent in type, the daily variations amounting to one or two degrees Fahr. The height to which the thermometer now rises varies with the extent of the inflammation in the cutaneous tissues, and therefore roughly with the number of the pustules; even in severe cases it is seldom above 102° or 103°. The pulse is quickened, being at from 110 to 120. The patient at the same time complains of headache, and is restless and sleepless. He is not unfrequently delirious, especially during the first night or two.

It must not be supposed that discrete smallpox is always unattended with danger. Trousseau relates the case of a girl, aged twenty-one, who had passed through a remarkably mild attack, but who was one evening suddenly seized with cerebral symptoms and difficulty of breathing, and in an hour she was dead; and he remarks that when this form of the disease does prove fatal, death occurs at an earlier date than in the confluent form, namely, about the eighth or the ninth day.

2. In *confluent smallpox* the eruption necessarily fails to some extent to present its typical characters, at least upon those parts of the body where an actual fusion of its elements has taken place. Even before any definite papules can be recognised, there is often a diffused redness of the face, which in itself could hardly be distinguished from the rash of measles. Watson mentions a case in which the appearances of urticaria, with its characteristic sensations, were at the outset so intermingled with the papules of variola that for twenty-four hours he doubted which of the two diseases was developing itself; and in other instances smallpox has been mistaken for erysipelas. Trousseau remarks that even where there are, in fact, multitudes of papules, the complete absence of intervals between them may render one almost unable to detect any unevenness of the surface by passing the hand over the patient's forehead or cheek. The papules are always smaller than in the discrete form; they coalesce so as to form the most irregular figures and patterns. At a very early period a milky fluid collects in their summits, giving rise to minute flat vesicles. The whole of the face has then a whitish-yellow colour, and its appearance is exactly like that of a mask of parchment, with which substance Morton, in his 'Pyretologia,' long ago compared it. By this time, however, there is already a good deal of swelling, and it goes on increasing up to the ninth day of the disease, and until the features are so altered that it is impossible for the patient to be recognised by his nearest relations. The parts about the ears and the sides of the neck become enormously bloated; the eyelids cannot be opened. This condition is very painful, and the scalp is often so tender that the pressure of the head upon the pillow can hardly be borne.

Somewhat later, about the eleventh or twelfth day, the hands and the feet become red, swollen, and painful. Trousseau, following certain of the

old writers, attached a high prognostic value to this symptom; according to him its absence is almost always followed by death.

The *mucous membranes* generally suffer severely in confluent smallpox. The interior of the mouth not only presents the eruption already described, but the fauces may become covered with a diphtheritic membrane. Laryngitis often renders the voice very hoarse, and it sometimes causes a sudden fit of suffocation, which proves fatal in a few seconds before anything can be done to relieve it. Trousseau mentions three cases of this kind, in each of which death seems to have occurred unexpectedly on the eighth day of an illness that had previously run a normal course.

Another complication, perhaps due to extension of inflammation to the parotid gland, is *salivation*. Trousseau speaks of this as a characteristic feature of confluent smallpox. It begins, he says, about the fourth or fifth day, and goes on increasing until by the ninth or the tenth day one or two quarts may run from the patient's mouth within the twenty-four hours. Even during sleep there is a constant flow of saliva. Again, ophthalmia is of frequent occurrence, which (unlike the harmless discrete pustules of the conjunctivæ) is often attended with sloughing of the cornea, and leads to permanent blindness.

From the very commencement of the eruptive period, the general symptoms of confluent smallpox run a course which differs from that of the discrete form of the disease. Instead of the temperature falling when the papules come out, it remains at 103° or 104°. There is often violent *delirium*, even during the daytime. It may be quite impossible to keep the patient in bed, except by tying him down with a folded sheet across the chest. If free, he must be most carefully watched, or he is very likely to throw himself out of a window or over the stairs, or to cut his throat with a razor. In those who have been intemperate, the nervous disturbance often assumes the form of delirium tremens.

Retching and vomiting commonly run on throughout this period of the disease; and diarrhœa is often obstinate and troublesome. The urine contains albumen in a large number of cases.

When suppuration is established, there is a further rise of temperature. Indeed, it becomes higher than in the corresponding stage of discrete smallpox although from the patient having all along been feverish, the development of a fever of maturation is less conspicuous.

This is the most dangerous period of variola. Few patients die before the eleventh day; the most fatal days are the twelfth, the thirteenth, and the fourteenth. Towards the last there is generally coma; sometimes hyperpyrexia is present, as in a case of which Wunderlich gives a chart, and in which, before its termination on the eleventh day, the thermometer registered 109°. In many instances the immediate cause of death is bronchitis, pneumonia, pleurisy, or pericarditis. Even under the most favourable circumstances the fever runs on for at least ten days longer. Not infrequently, during the third week, the patient falls into a typhoid condition, with sordes, a dry brown tongue, muttering delirium and subsultus; and he is then very likely to die of exhaustion.

When recovery is to take place, whether from the discrete or the confluent form of the disease, the redness and the swelling of the face subside, as the pustules undergo conversion into crusts. The pain from which the patient has been suffering passes off, but only to be followed by the most intolerable itching. His fever also declines more or less rapidly, being now

proportionate to the amount of inflammatory action still going on. He regains his appetite, and becomes once more able to sleep. He opens his eyes, and gradually his features begin to reassume their natural outlines. At the time when the crusts are falling off, or a little later, he generally loses almost all his hair, and if the deeper layers of the skin of the scalp have been involved in the morbid processes there is always reason to fear that permanent atrophy of the hair-sacs may result.

3. I have still to describe two minor varieties of smallpox, which on account of their dangerous tendencies deserve to be ranked with the confluent rather than with the discrete form of the disease. One of them is characterised by the occurrence of hæmorrhage into the pustules. This must not be confounded with the *hæmorrhagic variola*, which proves fatal at an early period before the proper eruption has begun to develop itself. By way of contrast Curschmann calls it *variola hæmorrhagica pustulosa*. According to that writer the pustules generally attain their full size before any blood is effused into them; but sometimes this begins even during the papular stage. As a rule, those pustules which are seated upon the lower limbs are the first to show a purple colour. Bleeding presently takes place from the nose, kidneys, intestines, and other mucous surfaces. In women menstruation sets in, or abortion occurs if they are pregnant. The gums become spongy and bleed, as in scorbutus; the fauces show a kind of "diphtheritic" affection, which is attended with a terrible fœtor. The constitutional symptoms are in most respects severe; but although the pulse is very rapid the temperature during the eruptive stage is seldom above 102°, and it not rarely falls to 95°, or even lower, before the patient's death. Curschmann says that this variety of smallpox occurs chiefly in persons over forty years of age and in such as were previously out of health. He describes it as being almost always fatal. He points out, however, that in patients who have, in consequence of delirium, got out of bed and walked about during the early part of the eruptive stage of smallpox, the pustules on the legs may become filled with blood, without there being anything particularly serious about the case; and I think I recollect at least one case in which a similar appearance of the pustules on the forearms has brought with it no evil consequences.

4. Another and a very rare variety of smallpox is that which is called *corymbose*. I have never seen an instance of it. Marson describes it as generally presenting two or three patches or clusters, of the size of the hand, upon which the eruption is as thickly set as it possibly could be, while the skin around is for some distance almost, if not entirely, free. They are often developed symmetrically upon corresponding parts of the limbs. Sometimes there are numerous clusters, about as large as half-crowns, seated on different parts of the body. Elsewhere the pustules may be but sparsely scattered, and one would have expected the disease to be attended with little danger. The contrary, however, is the case. Marson collected from the register of the Smallpox Hospital one hundred and four cases of this kind which had occurred during a period of thirty years; and he found that there was a mortality among them of no less than 41 per cent. Even if only a single cluster was formed, the disease yet manifested its fatal character, and, what is perhaps most remarkable of all, it was scarcely less destructive to vaccinated persons than to those who were unprotected. In many cases the patient died from some complication when he had appeared to be doing well; and, at the best, his convalescence was long and tedious.

5. In *modified smallpox*,—which, as we have seen, is not exclusively confined to protected persons,—the course of the eruption is much less regular than in the ordinary forms of the disease, whether discrete or confluent. During the initial stage, however, no distinction can be drawn between it and them; no doubt there is a larger proportion of cases in which that stage is but little marked; but in some instances, at least, it lasts the usual time, and is attended with severe symptoms. But when the papules begin to appear, it is said that the characteristic irregularity is often shown by their being visible upon the trunk or elsewhere, before there are any of them on the face; and it is also said that the period during which fresh ones develop themselves is variable, the number being sometimes complete in a few hours, while in other cases they keep coming out even when those which were first formed have already become pustules. And what appears to be still more indicative of this variety of smallpox is the course of the fever at the commencement of the eruptive stage. The temperature falls, as in unmodified discrete variola, but with still greater rapidity, and without any interruption in its downward path, so that in less than twenty-four hours it is normal. Trousseau and others, however, speak of confluent smallpox running its normal course for ten or twelve days, and then suddenly subsiding, so as to show that it really belongs to the modified form of the disease.

It is thus evident that no definite period can be fixed at which the modified character of a given case of smallpox first becomes apparent. Sometimes the eruption dies away before it has passed out of the papular stage. Sometimes the papules undergo a partial conversion into vesicles, which then rapidly dry up. A special modification of this variety of the affection has long been known as the "horn-pox" (*variola verrucosa*); but writers by no means agree in their descriptions of it, for while Trousseau speaks of the vesicles as drying up and leaving in their places small, hard, corneous projections, which fall by a sort of desquamation between the tenth and the fifteenth days, Curschmann says that after the scabs have become detached, the solid bases of the pocks remain for a considerable length of time as warty elevations, especially on the face, to which they give an ugly appearance.

Again, in yet other cases of modified smallpox, the vesicles pass on into pustules. The change then appears to take place unusually rapidly, and the pustules themselves are often extremely small, and probably never reach so great a size as that to which they may attain in the unmodified forms of the disease.

Complications and results.—The complications of smallpox, besides those already mentioned, are few. They mostly concern the respiratory organs; and bronchitis is frequent. Œdema of the larynx has occasionally proved fatal. Salivation is frequent and distressing.

The *sequelæ*, beside the pitting, are chiefly various forms of local suppuration, like those which follow enteric and scarlet fevers; abscesses in the skin and deeper organs, furunculi and ecthyma, local gangrene and phlegmonous erysipelas. In severe cases the cornea may ulcerate, and the eye be lost.

Convalescence is, however, usually rapid, and the health and strength are not impaired as after enteric fever.

The *protection* afforded by smallpox is probably the most complete known.

Relapses never follow, and although second attacks undoubtedly occur, they are more rare than in the case of measles or scarlatina, at least as rare as in that of typhus and enteric fever.

Prognosis.—The points upon which this depends have been already indicated. They are (1) the number and quality of vaccination marks. The statistics of the Smallpox Hospital for twenty years show that where four distinct and pitted scars were present, the mortality was only one in two hundred; where three equally good were found, less than 1 per cent.; where three indifferent marks, more than 3 per cent. Two good marks kept the mortality down to 2·3 per cent., two poor ones only to 8 per cent. With one good mark, less than 4 per cent. of the patients died, and with one indifferent mark nearly 12 per cent. At the same hospital the mortality among unvaccinated patients was 37 per cent. (Marson).

(2) The second point for prognosis is the abundance of the eruption. Dr Marson's statistics give the mortality among 2654 of unvaccinated patients as 4 per cent. in cases of discrete variola; 8 per cent. in cases of semi-confluent, including coherent, variola; and 50 per cent. in cases of confluent variola. A corymbose eruption is, we have seen, particularly fatal.

(3) More than half the unvaccinated patients above thirty years old who are attacked by smallpox die, and of those above sixty nearly 80 per cent. To children under five it is as fatal as to adults between thirty and forty; about half attacked die. The most favourable age is from eight or ten to twenty; even then, about one in four die.

(4) A purpuric eruption is of most serious omen; most so when it precedes the true exanthem, but hemorrhagic pustular smallpox is also extremely dangerous.

(5) Delirium and convulsions, previous drinking habits, and want of sleep are indicative of danger. "Children who grind their teeth hardly ever recover" (Marson).

(6) Pregnancy is usually a fatal complication; abortion usually takes place and the mother dies soon after. Cases, however, are recorded in which both the mother and the child have survived.

Treatment.—There is no special means of treating smallpox apart from treatment of the fever and of the local complications. The most rigid isolation is of course necessary for the sake of others. Free ventilation and the utmost cleanliness are the first essentials. Diarrhœa should be checked. Laudanum or morphia are admissible and valuable as hypnotics. Ammonia and senega are needed for bronchitis, and stimulants during the suppurative stage, especially when boils or abscesses appear.

Vaccination in unprotected persons exposed to contagion is imperative; and even if performed after the first symptoms of the disease have appeared, it probably has some effect in diminishing its severity.

The true treatment for smallpox is prophylactic, and will next be considered.

VACCINIA.—The terrible mortality from unmodified smallpox, and the scarcely less terrible results it left behind—the scarred and hideous features, the sight injured or lost, permanent deafness, and other disastrous results—can only be appreciated by those familiar with the general as well as the

medical literature of the last century. Treatment of the disease had been much improved by Sydenham, but its ravages were still almost unchecked until the introduction of the practice of *Inoculation*. This has been carried on in China from time immemorial, but it is not known how it was introduced into Turkey. An account of inoculation as a prophylactic for ordinary smallpox was first published in the 'Philosophical Transactions' for 1714, but it became popular seven years later by means of the letters and example of Lady Mary Wortley Montagu, the wife of the English ambassador at Constantinople. By inoculation with the lymph from a variolous vesicle the disease is reproduced with a shorter incubation period, with far less severe symptoms, and with a far smaller mortality than when contracted in the ordinary way.

Variola, however, thus produced was still variola, and as contagious as ever, so that the effect of inoculation was to make more cases of smallpox, along with a generally milder form of the disease. Thus, although the mortality was reckoned at only 3 *pro mille*, the total deaths from smallpox were increased. The Smallpox and Inoculation Hospital was founded in 1746 and inoculation was practised there down to 1822, but after 1840 it was prohibited by Act of Parliament. The operation was performed in China by putting variolous crusts into the nostrils, but in Europe in exactly the same way as vaccination except that the lymph was variolous instead of vaccine.

The practice of *vaccination*—the greatest achievement of medicine—was the fruit of the scientific temper and indomitable perseverance of the illustrious Edward Jenner. He observed that the milkmaids in the great dairy farms of Gloucestershire were subject to a particular eruption of the fingers which was derived from similar pustules on the udder of cows. He ascertained the accuracy of a current belief that those who were so affected did not suffer from smallpox. After long investigation he was so satisfied of the fact that in 1798, after inoculating a boy with the matter from a cow's udder, *i. e.* with the cow-pox instead of with smallpox, and finding that this inoculation was as good a protection from the dreaded variola as the other, he made known his discovery.*

The practice of inoculation with this "vaccine lymph" rapidly spread. It was adopted by the best physicians abroad as well as in England, and the process of "vaccination," thus begun, was at last made compulsory in all civilised countries. Vaccination does not, and Jenner did not pretend that it would, protect absolutely from smallpox. Inoculation did not, and an attack of smallpox by ordinary contagion does not, absolutely protect. But variola, like other exanthems, protects in the vast majority of cases against a second attack; inoculated variola is a mild and comparatively safe form of the disease, and likewise protects from further attacks; vaccination is a still milder and perfectly safe form of disease and likewise protects from future smallpox.

The statistics given on the last page show the truth of this assertion. It is also proved by the following example, quoted by Dr Eustace Smith. Dr Gayton had 1574 children under observation in the Smallpox Hospital at Homerton (1871–78). Of these 211 showed good vaccination marks, and only one of them died; 396 showed incomplete vaccination, and 39 of them died; 179 had been vaccinated but showed no scar, and 46 of them died; while of 788 who unhappily had never been vaccinated at all, no less than 385 died.

Similar evidence on a large scale is afforded by the compulsory vaccination carried out in the great Continental armies, by the results in our own army, in the military school at Chelsea, and by observations in Sweden, in Germany, in France, in the United States, and in India.

It is certain that vaccinated persons are to a small extent liable to smallpox, especially if the scars are few and slight, or if the time since vaccination has been very long. Efficient vaccination is necessary to obtain the full benefit of Jenner's discovery. A second vaccination is desirable about the time of puberty, and repeated vaccination is prudently submitted to by those who are going into contact with the disease, as doctors and nurses taking duty at a smallpox hospital.

With delicate persons the local inflammation attending vaccination may be severe and cause some febrile action and several days' discomfort. With feeble infants the effects may be more serious. And it is now certain that vaccination from a syphilitic child may introduce the virus of syphilis as well as that of vaccinia, especially if blood as well as lymph be inoculated. This is, however, an occurrence so excessively rare that while every precaution is rightly taken against it by choosing healthy infants from whom to take vaccine lymph, the practical risk is infinitesimal.

A much more common but less certainly ascertained drawback to vaccination is that the slight febrile disturbance which attends it may call forth local or general disorders, especially eczema and catarrhal inflammations. Swelling of the axillary lymph-glands of the vaccinated arm and erythematous œdema of the arm itself are frequent, and sometimes, especially in adults, troublesome. Erysipelas or pyæmia are possible results, but not more frequent than after any other slight puncture.

It has been suggested that these drawbacks, slight as they are, might be avoided by vaccination direct from the udder of a heifer affected with Vaccinia instead of from a vaccinated child. And this plan has been extensively carried out. But its disadvantages are greater than its advantages. The operation is less certain of success, the febrile disturbance is usually more marked, and the maturation of the pustule and falling of the scab is considerably delayed. Hence "arm-to-arm" vaccination, from a healthy child to a healthy child, is probably the most efficient, least disturbing, and most practically eligible method.

Course of vaccinia.—When vaccine lymph has been introduced into the lymphatic spaces of the skin, an incubation period of two days follows. On the third a papule appears, which in two days more becomes a vesicle. Towards the end of the first week a large umbilicated vesicle forms and attains its full development on the eighth day, when a congested halo surrounds the pustule. The lymph then becomes turbid, its contents gradually dry up, the areola fades again, and by the end of the second week a scab has formed. This falls off about the end of the third week, and leaves a pitted depressed scar, at first darker but finally whiter than the surrounding skin. Meanwhile, slight pyrexia has begun on the fourth or fifth day, increases at the beginning of the second week, and falls with the maturation of the pustule and the disappearance of the surrounding inflammation.

This process is sometimes slower, as when the vaccinated child is already in the incubation stage of another febrile disorder like measles; and it is sometimes quicker, or otherwise disturbed, as when the child is already affected by some inflammatory affection like eczema.

The course of a second vaccination is almost always rapid, and the

constitutional disturbance usually greater, owing perhaps to the patient's age.

Nature of vaccinia and pathology of vaccination.—Jenner himself believed that cow-pox was modified variola. This was long disputed, but numerous experiments, particularly those by Mr Ceely (1839) and Mr Badcock (1840), have proved the point. Variolous lymph inoculated on a cow's udder produces vaccinia; this inoculated in a human arm reproduces itself and protects from subsequent vaccination, as well as from subsequent smallpox. Cows only, not oxen, are subject to vaccinia, because the contagion is conveyed to them from human variola in the process of milking. The disease is only derived by cattle from human beings, and dies out when not thus reinforced. It may also affect horses and probably sheep, though the variola ovina is in certain respects different. It is not related to "grease" in horses.

Vaccinia, therefore, is true variola modified by "cultivation," and is the first and hitherto the most successful example of the method of protection from specific diseases by previous inoculation with an "attenuated" virus.

For further details on the subject of vaccination, so interesting as an example of scientific experiment and reasoning applied to medicine, so important in its bearing on general pathology, and so deeply affecting the lives and happiness of the human race, I may refer to Dr Seaton's article in 'Reynolds' System,' to Mr Simon's Reports to the Privy Council relating to the history and practice of vaccination, and to Mr Ernest Hart's excellent popular pamphlet.

VARICELLA*

History of the recognition of Chicken-pox—Its distinction from Smallpox—Incubation and onset—Characters of the eruption—Symptoms and course—A disease of children—Diagnosis—Prognosis.

At the end of the seventeenth century, soon after the final separation of measles from smallpox by Sydenham, English writers mention a variety of the latter disease popularly called "chicken-pox." In all probability the allusion in that name is to *chickpease* (French *chiche*, Latin *cicer*). The same affection seems to have been described in the sixteenth century by Vidus Vidius, and by Ingrassias, under the designation of "Crystalli." Vogel (1764) is said to have introduced the name of Varicella. In 1730, Fuller asserted that chicken-pox and smallpox were really distinct diseases; but Heberden, in the first volume of the 'Medical Transactions of the College of Physicians' (1767) first indicated fully the differences between them. He also pointed out the chief reason which made the recognition of chicken-pox a matter of importance, namely, that those who had it might otherwise be deceived into a false security, "which might prevent them either from keeping out of the way of the smallpox or from being inoculated."

After the introduction of vaccination, it became of great consequence to distinguish the two diseases, since every case of varicella occurring in a vaccinated person would otherwise have been regarded as an instance of the failure of the operation. But it is to be regretted that some of the earliest advocates of Jenner's method, being anxious to show that the protection afforded by it was absolute, referred every suspicious-looking eruption in persons who had been vaccinated to chicken-pox. This, in its turn, led to a reaction, since it necessarily involved the giving up of all the distinctive characters of the affection, and the consequence has been that its claim to be regarded as an independent disease has been disputed by several eminent physicians, from Dr John Thomson, of Edinburgh (1820) to Hebra, of Vienna. Unfortunately, too, the great German dermatologist has helped to throw the subject into confusion by employing the term varicella in an entirely new sense, namely, for all very mild cases of smallpox. Thus when he says that "varicella" may generate variola, he really means nothing that would not be admitted by everyone. It is, however, perfectly certain that the varicella of other writers is altogether unconnected with smallpox.

The proofs are (1) that it occurs in those who have been vaccinated, or who have had variola, just as readily and with the same characters as in those who are unprotected; and (2) that a person who has passed through it remains as susceptible as before to the vaccine virus or to that of smallpox. In the 'Lancet' for 1877 a case is recorded of an unvaccinated child, who

* *Synonyms.*—Variola crystallina, spuria, volatica, Chicken-pox.—*Fr.* Varicelle, *Germ.* Windpocken, Wasserpocken. The word varicella is evidently intended as a diminutive of variola.

was admitted into St Thomas's Hospital for chicken-pox, but who was placed on the floor containing the smallpox wards, because the diagnosis was at first uncertain. Two days afterwards vaccination was performed, which succeeded. Eight days later still the child fell ill with modified variola. According to Thomas, Czakert vaccinated with success a boy in whom the inoculation of the cow-pox had failed on three previous occasions, by introducing the lymph into the interior of the vesicles of a varicella with which he happened to be attacked. Indeed, if it were not that at the present day almost everyone is vaccinated in infancy, we should have in the cow-pox virus an almost infallible test, which we could apply at our own discretion, and which would soon clear up all doubts as to the nature of any individual case that might be difficult of diagnosis. I think it is not too much to say that we have really more positive grounds for declaring that smallpox and chicken-pox are distinct, than for making the same statement about almost any other two acute diseases. What makes the case the stronger is that, whereas the fluid from even the mildest eruption of variola is capable of conveying the disorder by inoculation, almost all of those who have tried to inoculate varicella in the same way from its vesicles have failed. Early in the present century Bryce performed this experiment upon children who had never had either smallpox or cow-pox, to the number of thirteen, without any result. It is true that in a long series of cases collected by Hesse in 1829, there was a small minority in which inoculation appeared to succeed; but according to Thomas there are reasons for thinking that some error crept into these observations. In order to negative one of Dr Thomson's principal arguments on the other side, most writers cite Möhl's statement that from 1809 to 1823 chicken-pox was annually observed at Copenhagen without concomitant smallpox, "and that afterwards both diseases prevailed at intervals epidemically, but always under circumstances which convinced the physicians of the town that their sources were distinct." But it seems to me that evidence of this kind, based only upon opinion, tends rather to weaken a position which is otherwise altogether impregnable.

Nor are there wanting numerous points of difference from variola in the characters of the eruption of varicella, and in the way in which it develops itself.

Incubation.—This seems to be of variable duration, so far as has been ascertained, but I believe that in general it has been calculated upon the very precarious basis of the interval between the dates at which different children of the same family have been successively attacked. Dr George Gregory stated it at from four to six days; Heberden at eight or nine. Bristowe says that in some cases it lasts exactly a week, but perhaps more commonly a fortnight. According to Thomas, it may be from thirteen to seventeen days; according to Gee, about a fortnight; according to Liveing, variable, about thirteen days with eruption on the fourteenth. Eustace Smith gives seven to fourteen days; Trousseau, from fifteen to seventeen days; Dukes, fourteen to nineteen days.

Onset.—This is usually simultaneous with the appearance of the eruption, so that there is no stage of invasion of the fever before the exanthem appears, as in typhus, enterica, scarlatina, measles, and smallpox.

Thomas took the temperatures of children who afterwards developed varicella (no doubt where the disease was already in the family), and always found them normal or not raised above half a degree (Fahr.), which in childhood is of common occurrence in health.

Even where there are slight prodromal symptoms, as headache or cough, with loss of appetite and feverishness, they only last a few hours before the characteristic rash appears, so that the period of incubation must, as in the case of Rubeola (*sc.* notha), be reckoned up to that of eruption.

In some instances, however, Thomas found that the child was feverish for a few hours before the disease made its appearance, and sometimes the fever lasted two or three days, and was accompanied with delirium or (as in a case which I saw some years ago) with convulsions. Once this author observed a transitory general erythema, with a temperature higher than 106°.

The exanthem.—This begins as a series of small, slightly-pointed red spots, which Trousseau compares with the rose-rash of enteric fever; Dr Gee says that they disappear when the skin of the part is stretched, this being a proof that there is no exudation into the tissue of the cutis, but only hyperæmia. In a few hours they pass into as many transparent tense vesicles, round or oval in form, and about as large as split peas. These sometimes have a red base; sometimes they are seated upon a perfectly colourless surface, so that the patient looks exactly as if he had been sprinkled with drops of clear water. They are generally scattered quite irregularly, but it is said that they may occasionally be somewhat clustered together, as in herpes. A chief distinction between them and the vesicles of smallpox is their superficial position; they have no thickened floor, and they do not consist of a series of separate chambers; thus they are not umbilicated,* and when they are pricked they almost completely collapse. Thomas, however, says that when they first appear this is not the case, and that they are at that time intersected by delicate septa. They appear in crops, which come out in succession during the first, second, and third nights of the disorder. At length they acquire a yellowish appearance, and the fluid in them turns slightly opalescent, or may become puriform. After from twelve to twenty-four hours they begin to dry up and become flaccid; they comparatively seldom escape being ruptured, either by the nails of the patient (for there is often considerable itching) or in some other way; but they often fall in first at their centres, so as to acquire a sort of spurious umbilicus. Ultimately they form thin brownish-yellow scabs, which in a few days crumble away, leaving reddish pigmented spots.

The eruption of varicella generally appears first on the upper part of the back or on the chest, rarely on the face. Formerly it was said to spare the face; and although this is incorrect, it certainly does not come out more thickly there than elsewhere, as is the case with variola. On the scalp it is almost always present. Afterwards it spreads to the limbs. One of its most important characters, however, is that even on the body the vesicles come out, not in a single crop, but in a succession of crops, which may be prolonged over three or four days or even a whole week. Thus one sees papules and vesicles and crusts side by side at the same time; in this it is very different from smallpox. The total number of vesicles is sometimes not more than from ten to thirty, but according to Thomas it is generally two hundred or two hundred and fifty; as many as eight hundred are said to have been observed. Only a few come out on the first day; perhaps a score. Then a hundred or more fresh ones are seen the following morning.

* So many authors: but it is certainly not always the fact. Most of the vesicles of Varicella are rounded, but in almost every case some may be found umbilicated and occasionally most seem to be so.

The scabs which form as the vesicles dry up fall off, if left to themselves, in a week or ten days, and leave reddish marks which gradually disappear; but sometimes a few small white cicatrices remain behind; and these may occasionally be depressed and even pitted.

The *mucous membranes* take part in varicella. Thomas says that complaints of pain in micturition have often led to his observing vesicles upon the labia of girls, but that on the prepuce in boys they are less commonly to be found. On the palate they often persist for some time, having slightly reddened bases, but on the lips, the tongue, and the cheeks they are to be recognised only in the form of excoriations or small superficial ulcers.

Its varieties.—Some of the papules which, as we have seen, constitute the earliest stage of the eruption of chicken-pox, not infrequently abort and disappear, especially those which come out towards the end of the disease. Thomas has recorded a case—the nature of which was established by the fact that the patient's sister had just before had varicella—in which every single papule died away after thirty-six hours, so that no vesicles were formed. On the other hand, the vesicles sometimes increase in size until they deserve the name of bullæ; such an enlargement of them does not always begin until they have already become scabbed over, and they may go on spreading at the periphery of the crusts for a considerable time. Indeed, it would seem that in exceptional instances varicella lasts much longer than any other exanthem. Trousseau speaks of an epidemic in the Necker Hospital, in which, during from fifteen to forty days, blebs like those of pemphigus kept appearing on different parts of the patients' bodies, leaving ulcerations which lasted for six weeks or two months. Mr Hutchinson ('Lect. on Clin. Surgery') saw a rash exactly like fading chicken-pox, which was said to have been out during a month, and which vanished spontaneously almost immediately afterwards.

Its relapses and sequelæ.—Some writers speak of relapses of varicella, and Thomas, although he denies that the disease is ever followed at once by a second complete attack, admits that he has seen a few vesicles appear as late as a month from the commencement of the eruption. The question is of some importance, on account of its relation to a remarkable cutaneous affection which Mr Hutchinson believes to arise out of chicken-pox, and which he therefore terms "varicella-prurigo." He has recorded notes of sixteen cases, all of which are said to have begun with the characters of varicella, most of them having in fact been diagnosed by medical men as examples either of that disease or of modified smallpox. But it is curious that it never affected several children in the same family; indeed, there is only one instance out of the whole number in which more than one child suffered from the original disease; and I must confess that my doubts as to the correctness of Mr Hutchinson's views are strengthened by the fact that he gives twelve other cases in which the same skin disease was supposed to have been caused by vaccination. In fact I believe it to be an exaggerated form of strophulus. There is no doubt, however, of the validity of Mr Hutchinson's observation of gangrene as a complication of varicella, though it is happily a very rare one. Instead of the vesicle running the usual course it becomes tinged with blood, and the scab is very large and black. The slight areola is more marked and of a dusky hue. Under the crust is an ulcer, and this may penetrate deeply. These multiple gangrenous ulcers may prove fatal in young children.

Course.—The general *symptoms* of varicella are very slight. In some

patients the thermometer fails to rise above normal during the first twenty-four hours, while the vesicles are coming out. In others there is slight fever of two or three days' duration, but it often passes off in a single day. Moderate fever may last four days. It is particularly to be noticed that the temperature in chicken-pox does not fall, as it does in variola, when the eruption begins to develop itself. Trousseau, however, speaks of successive onsets of fever, occurring regularly at night, and accompanying the different crops of vesicles; and Thomas seems to have observed something of the same kind, although he speaks of such exacerbations rather as tending to interrupt the nocturnal rise and the morning fall of temperature which would generally occur in this disease, as in most others. The maximum temperature is about 102°. In some slight cases there is said to be complete absence of fever throughout the course of the eruption. The defervescence, when it occurs, is usually rapid.

All that need be said about other symptoms is that the patient may be a little restless or drowsy, but that he seldom complains of headache or wishes to be kept in bed. The tongue is clean, but there may be some thirst and loss of appetite. In a child who was previously suffering from chronic laryngitis, Dr Gee noticed a decided increase of dyspnœa during an attack of varicella.

Sometimes, however, the symptoms are more marked, perhaps more in children above six or seven years old, or in adults.

In a case seen by the Editor in 1886, the patient, a lady twenty-two years old, felt unwell one day, but went out to dinner and was then obliged to leave the table, and fainted. Next morning the rash appeared on the face and shoulders, and continued in successive crops. The highest temperature was 102° F., and it did not rise with the appearance of the eruption. There were good vaccination marks. The febrile disturbance lasted about three days. One scar remained on the forehead close to the hair, where a scab had been repeatedly scratched off. There were other cases of chicken-pox among children in the village, one of which I saw.

Predisposing causes.—I have throughout spoken of *children* as the subjects of chicken-pox, and in truth the disease is almost confined to them. Dr Gee gives a table drawn up from the records of the Great Ormond Street Hospital, according to which infants under six months of age were often attacked, while there was absolutely a much larger number of cases among children between six and twelve months old than during any other period of equal length. Most patients are under six, and after ten years of age it very rarely occurs. Most writers say that they have never seen it in grown-up patients, but Heberden relates one case in which a mother caught it from her children, and another instance of it in an adult female was observed by Gregory.

Varicella is sometimes sporadic, sometimes epidemic. Thomas remarks that in large towns epidemics are not separated by intervals of several years (as is the case with measles and smallpox), but occur once every year or once every half year—in Leipzig regularly a short time after the opening of the infant schools.

This disorder does not affect any particular season, although Heberden speaks of it as occurring chiefly at the end of summer, and Gee thinks it is most common in the fall of the year, in September and October. It is highly infectious. The contagion is probably communicable through the air, and is also capable of adhering to solid bodies. That it possesses comparatively

little tenacity may perhaps be inferred from the fact that the extent of an epidemic is not generally very large.

Varicella protects efficiently against itself, but not against vaccinia or variola. Trousseau is almost the only authority who has seen second attacks of chicken-pox.

Diagnosis.—The diseases with which variola has been confounded are acne, pustular syphilide, and molluscum contagiosum, to distinguish which needs only knowledge, and care—and variola, especially variola in vaccinated persons, and here the distinction is sometimes difficult. The slightness of the early symptoms, the close sequence of the exanthem, the softness of the papules, their appearance in successive crops, their distribution, and rapid course will generally decide the question.

The *prognosis* is always good except in the rare cases of subsequent gangrene. There are no other complications and no proper sequelæ; but occasionally children are some time in regaining strength, and both Gee and Eustace Smith have seen tuberculosis follow chicken-pox.

The vesicles should as far as possible be protected from injury by scratching, lest the resulting scars be made deeper than they otherwise would be.

No *treatment* is needful, except to ease the troublesome itching.

ERYSIPELAS*

Definition of the disease—Its contagion—A specific febrile exanthem—Relation of the phlegmonous form to facial idiopathic erysipelas—Incubation and onset—The eruption—Its histology—The symptoms and course—Sequelæ—Recurrent erysipelas—Diagnosis—Prognosis—Morbid anatomy—Treatment.

ERYSIPELAS is one of those diseases which date back to Hippocrates. There have, however, been great differences of opinion as to what affections should be included under this designation.

Some physicians, especially in France, have been anxious to separate from the cases which are seen in surgical practice, of erysipelas attacking the limbs and the body after injuries, a "medical erysipelas" of the face and head; in England this distinction received Sir Thomas Watson's support. Another distinction, which at one time caused much discussion, was that between erysipelas and erythema. The latter term is applied to various forms of superficial dermatitis, which will be described in the last section of this work; but there can be no doubt that it has sometimes been held to include cases which really were slight cases of erysipelas. But if the scope of the word erysipelas has thus been unduly narrowed in one direction, in another direction it seems to have been made far too wide. Many English surgeons regard as "erysipelatous" almost every form of spreading inflammation of connective tissue, including even such a diffuse affection of the retroperitoneal structures as that which is apt to arise after operations on the rectum or on other pelvic organs. This view is strongly objected to by German writers, among whom Volkmann, in the 'Handbuch' of Pitha and Billroth, must be specially mentioned. They will not even allow that we are right in describing under the name of "phlegmonous erysipelas" cases of suppuration or gangrenous inflammation, attended with intense redness of the skin, but affecting also the subcutaneous and even the intermuscular textures.

Ætiology.—It is clear, I think, that the settlement of all these questions must ultimately depend upon ætiological considerations. Erysipelas is a contagious disease. Of its propagation by contagion several examples were recorded many years ago by Dr Wells, of St Thomas's Hospital, the celebrated author of the 'Essay on Dew.' For a long time Continental observers disputed the possibility of such an occurrence, but Volkmann cites more than a dozen instances of it, and it is now universally admitted. Perhaps the most striking series of cases that have been recorded was brought before the Paris Academy in 1864 by Dr Blin. One of the surgeons at the Lariboisière Hospital had under his care two patients suffering from erysipelas when he was himself seized with it. A medical friend from Guise visited him and fell ill after returning to that place, where no other case of the disease then existed. That gentleman's servant was attacked, and also a relative who came to see him, and who lived in the neighbourhood. The

* *Synonyms.*—Febris erysipelatosa, Ignis sacer, Rosa, the Rose, St Anthony's fire.—*Germ.* Rothlauf. Ἐρυσίπελας, *i. e.* red skin, is a classical Greek term.

latter gave erysipelas to his wife, and three members of another family who were repeatedly in contact with them during their illness suffered in their turn. From this family the disease spread to two sisters of mercy, and they carried it to their home and gave it also to a medical man who attended them, and lastly it passed from him to his daughter.

By far the simplest view of the matter is to regard erysipelas as not only contagious, but *specific*, in the sense of being always dependent upon the entrance into the body of a definite *virus* from without, and in support of such an opinion very much may be said. It is a great mistake to suppose that the disease, like pyæmia, necessarily becomes prevalent wherever surgical patients are crowded together under unfavourable conditions. In the Crimean war, in the Austro-Prussian war of 1866, and in the Franco-German war of 1870 repeated examples presented themselves of hospitals which it was impossible to keep healthy, and yet erysipelas failed to appear. On the other hand, there have been many instances in which it has prevailed epidemically, sometimes in a single ward or in several wards of some one hospital, sometimes in various institutions of a city or town. Whether it ever spreads as an epidemic over the inhabitants of an entire district, like the exanthemata, is more doubtful. Between 1841 and 1854 this is said to have been the case in various parts of the United States, but both Volkmann and Hirsch are of opinion that the disease was really different from erysipelas and allied rather to diphtheria. In a hospital the poison of erysipelas often clings to particular wards, and even to particular beds, with extreme obstinacy. Mr Savory, in the 'Brit. Med. Journ.' for 1873, remarks that at St Bartholomew's, during a small epidemic which occurred there, the disease almost always, in passing from one person to another, attacked the nearest patient who had an open wound. And the late Mr de Morgan, in 'Holmes' System of Surgery,' cites, on the authority of Dr Goodfellow, a most extraordinary instance in which it spread in regular order throughout a ward of thirteen beds to almost every patient in turn, going down one side of the ward and then up the other side. Even where the poison seems to adhere most closely to a particular spot, it may be that it really comes from a little distance off. A case in point is related by Mr de Morgan. It having been found at the Middlesex Hospital that patients occupying two adjacent beds with a window between them were particularly apt to be attacked, the suspicion arose that this might be due to the presence of a dustbin in the area below. It was cleaned out and there were no further cases. But two years later the disease reappeared, whereupon it was found that the dustbin had again become foul, although no unpleasant effluvium from it could be detected. The adoption of the same measures as before rendered the beds again healthy. In this instance it might perhaps be doubted whether the impure air actually conveyed a poison to the patients, or whether it merely predisposed them to be attacked. But no such doubt seems to be admissible in regard to another set of cases, recorded by Dr König, of Rostock, in the 'Arch. d. Heilkunde' for 1870. In the hospital of that town a small epidemic was clearly traced to infection from the cushion of the operating table, which cushion had become deeply discoloured by dried blood from former patients; from the day when it was removed no fresh case of erysipelas occurred. The cushion was now soaked in water, and a brownish solution was obtained which was inoculated upon two rabbits, with the result that one of them had an affection which closely resembled erysipelas. Dr J. Orth, of Bonn, has since recorded in the 'Arch. für experim. Path.' for 1873, a

series of experiments by which he has demonstrated the possibility of infecting rabbits by the subcutaneous injection of fluid taken from an erysipelatous bulla in man; he also transmitted the disease from rabbit to rabbit by inoculating with liquid from inflamed and œdematous parts of the skin and even with blood from animals already infected. That erysipelas can be conveyed in a similar manner from one human being to another seems to be established by an old observation made by Doepp, who vaccinated nine children with lymph from a child who on the following day fell ill with erysipelas; all were attacked by it. Orth found motionless micrococci both in the fluid with which he began his experiments on rabbits, and in that which he subsequently made use of. He even succeeded in producing erysipelas by inoculating animals with micrococci that had undergone artificial cultivation. The presence of such organisms in erysipelas had, indeed, been previously demonstrated by Hüter, not only in tissues affected with the disease and in the œdematous fluid which saturates them, but even—in small numbers—in the blood. Subsequent investigations have confirmed the presence of *Micrococcus erysipelatosus*, and it has been employed to produce erysipelas of a cancerous tumour with the hope of checking or destroying the new growth.

Thus a good case seems to be made for the view that erysipelas is a specific disease, and for my own part I am strongly of the belief that this will be the conclusion at which pathologists will ultimately arrive. At present, however, many observers hold a different opinion with regard to it. Mr Hutchinson, as I have remarked at p. 26, considers it to belong to the class of diseases which he terms "contagious inflammations." He is convinced that it is often set up by a draught of cold air blowing upon a wound, as when a hospital ward has its windows kept open all night.

Phlegmonous erysipelas.—We must now revert to the question of the relations between the various forms of disease that have been included under the name of erysipelas. As regards the "spreading inflammations" first mentioned, Volkmann himself admits that acute diffused suppuration, severe diphtheria, or even puerperal fever, when introduced into a hospital free from erysipelas, often forms the starting-point of an outbreak. He cites a case by Pujos, in which a man who had a severe gunshot wound of the foot died of erysipelas, or, as he says, "pseudo-erysipelas," which in several places had passed on to gangrene. A brother, who attended him, was attacked with erysipelas of the face, and so was a nurse; and several other persons became affected with phlegmonous inflammations. Volkmann adds that those who attend upon cases of puerperal fever often become the subjects of facial erysipelas. Such occurrences are the more striking because it is admitted on all hands that contagiousness is a far less conspicuous feature of erysipelas than it is of many specific maladies. But, after all, it is quite possible to account for them without giving up the view that erysipelas is a specific disease. It may be that there are certain cases of puerperal fever, or of cellulitis, or of diffused phlegmonous inflammation, which owe their origin to the poison of erysipelas, and that these cases alone are capable of reproducing it in their turn. Or it may be that just as in cultivation experiments there is great difficulty in keeping fluids free from accidental contamination with other bacteria besides those which one wishes to develop, so the discharges from diseased surfaces in living patients afford a favourable nidus for the growth of various kinds of microzymes which may have no relations to the complaints from which these persons are suffering.

Until recently it was thought that erysipelas of the face and head, as physicians see it, differed altogether from the surgical affection in one very important respect, namely, in appearing upon the unbroken skin, and independently of any wound or abrasion. I believe that Trousseau was the first to point out that it very often has a starting-point in some slight breach of surface, as from a suppurating pimple at the angle of the eye, which the patient may have scratched, or from a trifling eczematous eruption on the nose, or from a fissure at the corner of the mouth, or from a herpetic affection of the fauces, or even from a sore gum due to a decayed tooth. This view has since been adopted both by Volkmann and by Zülzer. The most careful record that I have met with of observations made for the purpose of testing its correctness is by König. Among twenty-nine cases of erysipelas of the face or scalp which occurred in the inmates of a prison at Ziegenhain, fifteen were traceable to previous injury of the affected parts; in the remainder no such starting-point could be found, but in all of these the face was the seat of the disease, and in many of them it was so much swollen when the patient was first admitted that no complete examination could be made.

The supposition, of course, is that the contagious virus of the disease settles upon the spot which is afterwards to become the seat of the erysipelatous inflammation, and that infection of the blood is secondary. The complete want of symmetry in the distribution of the cutaneous affection may be mentioned as affording a strong argument in favour of this view.

Incubation.—Watson mentions cases in which the incubation of erysipelas appeared to last a week, and Murchison states that in his experience it was from one to three or four days. There is reason to believe that during this time local changes are actively going on, although there is no obvious inflammation of the skin. For it had long ago been noticed by Frank and by Chomel that the development of erysipelas of the face is often preceded by pain, tenderness, and swelling of the cervical glands; and the late Mr Busk, after close observation of a large number of cases, was convinced that this was an invariable occurrence. The most probable explanation of it seems to me to be that inflammatory exudation is really taking place, but that the lymphatics carry off the effused matters as fast as they appear. The possibility of such a modification of the inflammatory process is expressly admitted by Cohnheim. Doepp's vaccination cases, too, seem to show that the infective microzymes of the disease must at the same time be multiplying themselves locally, for it will be remembered that the lymph which conveyed erysipelas was taken from the arm of a child in whom the disease did not appear until the following day. Nor is there any difficulty in imagining that the blood may even at this early period be contaminated both with microzymes and with inflammatory products, whether by direct absorption through the veins or by transmission through the lymph-glands and onwards through the thoracic duct. And thus it is quite easy, without supposing a primary infection of the blood, to account for the production of constitutional symptoms, which seem to be premonitory of the local affection but are really not so. Watson says that before the outbreak of erysipelas the patient often feels ill, shivery, feeble, languid, and often drowsy; that his pulse is very frequent; that he may suffer from nausea, vomiting, and even diarrhœa. It must be added, however, that he speaks of sore-throat "as an early, and almost a constant, accompaniment of the complaint." Volkmann declares that in the many cases in which he has watched the development of

erysipelas in patients, already in hospital, whose temperatures had been systematically observed for some time previously, he has never seen any prodromal symptoms whatever. In his opinion, whenever they seem to be present, the fact really is that the local affection has already begun ; but in some deeply-seated part, so that it remains undiscovered. Now, Trousseau has maintained that erysipelas of the face often starts from the mucous membrane of the palate and fauces, and reaches the skin by passing outwards through the nostrils. It is obvious that such an affection might well account for the early swelling of lymph-glands to which I have already alluded. The matter is therefore one which needs further investigation.

A case in point, which interested me very much, came under my observation in 1882. A man who had had jaundice for some time was admitted under my care on June 24th. His temperature was then 105°. He said that his febrile symptoms had begun with a slight rigor on the evening of the 22nd. I could not make out that there was any pylephlebitis or other local affection of the liver to account for the pyrexia. On the 26th the end of the nose was found to be red, and a blush of erysipelas soon spread over the face. It was then remembered that when he first came into the hospital he complained of sore-throat, and was hoarse, and that on the 25th the clinical clerk had noticed the submaxillary glands to be swollen.

In some cases severe febrile symptoms may set in and last two or three days before the rash appears. This period between the incubation and the exanthem is exactly like those of scarlatina, measles, and smallpox.

Onset and exanthem.—The onset of erysipelas is usually sudden, and it is often marked by rigors, but in persons already suffering from a febrile illness the increase of pyrexia may be comparatively trifling. The late Dr Woodman (in his translation of Wunderlich's work) remarks that even in adults epileptiform convulsions are not uncommon. The temperature may rise in twelve hours to 104,° and usually reaches its *fastigium*, which may be at 105° or even 106° within the first two or three days.

Soon after the fever sets in, some part of the skin, usually near the angle of the eye or the ala of the nose, begins to burn and tingle and becomes red and swollen. The redness rapidly deepens into a crimson blush, which under pressure momentarily fades, but returns as soon as the pressure is removed. Its extent increases until in a day or two it perhaps covers the whole of the face. Where it is spreading, it has a sharply-defined, raised border, beyond which, however, projecting processes advancing into the subcutaneous tissue may be felt with the finger. On the other hand, where it is stationary, its edge fades off gradually into the healthy skin beyond. There is extreme swelling, especially where the skin is loose, as upon the eyelids ; these become converted into rounded prominences, and it is impossible to separate them, so as to get a view of the eyeballs. The features are so altered that the patient cannot be recognised. The surface is tense and shining, though it may be made to pit by keeping up gentle pressure on it. There are often a few vesicles scattered here and there, or even blebs, which may reach a large size. Moreover, Volkmann confirms a statement originally made by Sanson that with a lens minute miliary vesicles can always be seen.

For three or four days the disease may go on spreading, until, if it began upon the face or the head, it may cover the whole surface down to the root of the neck. Volkmann, however, remarks that the chin always remains untouched by it. The conditions which determine its advance in one line

rather than another have lately been carefully studied by Pfleger, whose views are cited by Zülzer with approval. It would seem that this depends mainly upon the arrangement of the subcutaneous connective-tissue bundles; they everywhere interlace, so as to form rhomboidal meshes, but these are usually horizontal or oblique, whereas upon the chin their direction is vertical. Pfleger also maintains that wherever the skin is tied down to the deeper parts the spread of erysipelas is retarded or arrested, as, for example, along the crest of the ileum and Poupart's ligament. Erysipelas of the face and head seldom extends far upon the chest; but when the disease begins upon the trunk or upon a limb, it may spread until it has covered the whole body. If it goes on advancing, however, it subsides in the parts first attacked while it is springing up elsewhere; hence it is never a universal or even a symmetrical exanthem. Beyond an affected area small islets of redness may not infrequently be seen, but these are always connected with it subcutaneously. Volkmann declares that erysipelas never gives rise to two or more patches at a distance from one another: it may, indeed, happen that in a case of double amputation, both stumps are affected; or that in a case of erysipelas of the head, the disease breaks out a few days later round a pimple on the leg; but such cases should be regarded as multiple attacks of the disease. I have notes of the case of a child in whom after a wound of the temple, redness first appeared there, and subsequently upon the chest, the right arm, and the left wrist in turn; but I cannot say that the morbid process might not have been continuous over the back of the body and limbs.

Erysipelas faucium.—During an epidemic of erysipelas in a hospital, it is no uncommon thing for cases of sore-throat to occur, which are evidently of the same nature, but in which the skin remains unaffected. An account of this form of the disease has been given by Cornil, based upon a study of eighteen cases. He describes a shining, purple-red, œdematous swelling of the fauces, sometimes accompanied with the formation of bullæ. The tonsils often take no part in it. The lymph-glands below the jaws and in the neck are much enlarged. There is considerable pain in swallowing, and sometimes a profuse flow of saliva.

Histology.—The minute anatomy of erysipelas was first studied by Biesiadecki, and more recently by Volkmann and Steudener. In the dead body the disease is so little marked, from the redness and swelling having almost completely disappeared, that Volkmann evidently was surprised at not finding the deeper strata of the cutis, as well as the subcutaneous tissue, infiltrated with enormous numbers of granular leucocytes. He gives a drawing of the microscopical appearances, in which the cells are seen packed closely side by side. In the more superficial strata, they are, however, scattered very sparingly. The structure of the bullæ was investigated in 1868 at Vienna by Dr Haight, of New York. He found them to be divided into loculi by irregular septa which are made up of cells of the rete, drawn out into long, spindle-shaped and branching processes. The fluid of the bullæ of course contains numerous leucocytes, and is often converted into pus.

Symptoms.—While the local process is thus running its course, the pyrexia continues, the temperature rising and falling irregularly, or remaining at nearly the same level. According to Dr Reynolds it is frequently lower in the evening than in the morning. The pulse is quick, and it is generally soft and feeble; it may be dicrotic or intermittent. There is usually more or less delirium at night, and sometimes violent maniacal excitement. The patient often complains of headache, sleeplessness, and irritability of sight and hearing.

He has no appetite, and is thirsty; the tongue is thickly coated; he has nausea and may vomit repeatedly; there is often diarrhœa with extremely fœtid stools. The urine is scanty and commonly albuminous, and may even contain casts and blood.

The duration of erysipelas is variously stated by different writers; it is, in fact, very uncertain. Billroth says it seldom reaches fourteen days; Volkmann puts it at about six or eight days. When the disease wanders over the body and limbs it may run on for weeks or even months. The final defervescence is usually sudden, the temperature falling to the normal point in a few hours.

The subsidence of the cutaneous affection is also rapid, and then, four or five days after the commencement of the redness and swelling, the skin becomes pale and flaccid, and shrivels. At the same time, other parts of the surface may be at the height or the beginning of the morbid process. Volkmann and Steudener have investigated the histology of this stage of the process; they find that in the subcutaneous tissue the leucocytes disappear with extraordinary rapidity, breaking down in a few hours into a granular *débris;* in the superficial layers of the cutis they ramain visible a little longer, but within a day or two all signs of tissue-changes vanish. In the meantime the vesicles or bullæ have dried up into yellowish crusts. The cuticle subsequently desquamates, and is detached either in flakes or as a branny powder. When the scalp has been the seat of the disease, the hair falls out for a time, but is soon reproduced.

Sequelæ and recurrence.—But it does not always happen, even when a case of erysipelas ends in recovery, that the local affection subsides thus favourably. Delicate parts, such as the eyelids, the prepuce, or the labia sometimes slough, in consequence of the tension to which they have been subjected. In other cases, when the swelling has gone down, abscesses form here and there beneath the skin and need opening with the knife. Suppuration of the swollen lymph-glands is a very exceptional occurrence.

Erysipelas seems to have no tendency to protect against its own recurrence in the same individual. In surgical wards it used to be no uncommon thing for a patient to have two or even three successive attacks while a large wound was healing. Women sometimes have the disease once or oftener every year; I think that in such cases it is generally directly excited by cold, as by exposure to an east wind; but Zülzer and other writers say that it almost always has a starting-point in some local affection of the face, of the nasal mucous membrane, the ear, or the lachrymal passages. In course of time this "habitual" erysipelas leads to a persistent thickening and induration of the nose, ears, or eyelids which greatly deforms the countenance.

On the other hand, a remarkable effect of a single attack of the disease is that its subsidence is sometimes followed by the disappearance of long-standing cutaneous affections. This seems to have been first noticed by Cazenave in cases of chronic eczema or lupus. More recently it has been found that even sarcomatous growths of considerable size may vanish in a similar way. Volkmann gives copies of photographs taken from a woman under the care of W. Busch, who had several tumours on the face, varying in size from a hazel nut to a pigeon's egg; a portion of one of them was excised, whereupon she was attacked with erysipelas, and this led to the absorption of all the rest. In two other patients, enormous tumours of the cervical glands underwent a great decrease of size under similar circumstances;

symptoms of collapse, however, set in, which in one case proved fatal, while in the other case as recovery took place the growth rapidly regained its former dimensions. This patient had been intentionally exposed to erysipelatous infection, in the hope that it might act beneficially upon her disease. The tumour from the former patient, who died, was examined histologically by Rindfleisch, who found that almost the whole of it had undergone fatty degeneration, so that only in certain portions could the structure of a round-cell sarcoma still be recognised.

Diagnosis.—The recognition of erysipelas is easy, if we leave out of consideration the theoretical doubts as to the relation which it bears, on the one hand, to phlegmonous dermatitis and diffused suppuration, and, on the other hand, to certain forms of erythema. Zülzer speaks of the œdematous form of anthrax as being sometimes mistaken for it. There is another disease, about which I have repeatedly seen blunders committed, and that is zoster of the forehead and face. Only a few weeks ago I found lying in the hospital a youth whose face was covered with flour, through which a diffused redness was visible, while the eyelids were enormously swollen. The first glance, however, showed that the affection scarcely, if at all, passed the middle line of the forehead ; and according to the account which the patient himself gave, it had begun with an eruption of vesicles. I therefore made a confident diagnosis that the case was one of zoster, and dropped atropine into the eye, on account of the danger of iritis. At my next visit, the redness and swelling had disappeared without desquamation ; and the vesicles had dried up into characteristic dark brown eschars, embedded in the skin.

Prognosis.—This is generally favourable for the cases of erysipelas that come under the care of physicians, except in very old people and in infants : in the latter it not infrequently appears at the umbilicus, and proves rapidly fatal. Even in surgical practice recovery so generally takes place that it is difficult, as Dr Wilks has remarked, to understand how it came to pass that the common form of certificate submitted to a magistrate in cases of slight wounds used to be that there was no danger, "unless erysipelas should ensue." It may be, as he suggests, that what really was pyæmia was often set down to erysipelas. There can be no doubt, however, that erysipelas itself was once far more fatal, at least in certain institutions, than it is at present ; thus Volkmann says that in the old Hôtel Dieu at Paris, it would often happen that the majority of surgical cases attacked by it ended in death. But probably much of the difference lies in the fact that patients suffering from erysipelas are now well supplied with beef-tea and wine and brandy, instead of being bled and leeched and kept upon low diet.

Even in adults a fatal termination is apt to occur when the patient has chronic disease of the kidneys, or has been imtemperate, or is otherwise broken down in health. Before death the temperature usually rises to a great height, and sometimes it goes on rising for a short time afterwards.

Post-mortem appearances.—When there have been severe cerebral symptoms, it has often been supposed that inflammation has extended from the scalp or face to the membranes of the brain. Examination after death however, has very rarely verified this suspicion. Volkmann says, however, that he has seen the disease spread inwards from the orbit through the sphenoidal fissure, the orbital fat being found swollen and infiltrated with pus, as had been indicated during life by slight protrusion of the eyeball.

It is not uncommon for the disease when it affects the fauces, to extend

onwards to the larynx, and so to destroy life,* unless tracheotomy succeeds in obviating the danger. The folds at the entrance of the larynx are then found intensely œdematous, or infiltrated with pus.

In other cases the immediate cause of death is pneumonia or pleurisy. In the 'Guy's Hospital Reports' for 1861, Dr Wilks recorded two cases in which erysipelas of the surface of the abdomen appeared to have set up fatal peritonitis.

Another complication of which two instances have been observed in France by Larcher ('Arch. Génér.,' 1864) is ulcer of the duodenum; it is of great interest, on account of the occurrence of a similar affection as the result of burns and scalds. In a third example, reported by Malherbe ('Arch. Génér.,' 1865), there were ulcers in the lower part of the small intestine. Bayer has related in the 'Arch. d. Heilkunde' for 1870, a case in which severe hæmorrhage from the bowels preceded death; but the only lesion found at the autopsy was intense congestion of the ileum.

It has been questioned whether pyæmia is frequently associated with erysipelas in the same patient. Zülzer says that this is not the case, at least if the phlegmonous form and those attended with diffuse suppuration of the connective tissue be excluded from the definition of erysipelas; and our observations at Guy's Hospital seem to bear out this statement. Volkmann, indeed, declares that metastatic abscesses were present in from one third to one half of all his fatal cases; but he adds that every one of his patients also had a severe wound.

Treatment.—In this country we believe that the tincture of steel has a marked influence in checking erysipelas. Mr de Morgan, from his experience at the Middlesex Hospital, speaks most positively of its efficacy in reducing the duration of the disease, so that it subsides in from two to four days, instead of lasting a week or ten days. He gave at least a drachm or a drachm and a half daily, and sometimes as much as an ounce and a half or two ounces. On the Continent it is more usual to give quinine.

When a case comes under observation at the very commencement of the disease an emetic is believed by many to be useful. Dr Ringer believes that aconite administered at this period may cut short the attack. Given after the inflammation has appeared it usually brings down the temperature, but has no useful effect and possibly may do harm.

Rather more than fifty years ago Mr Higginbotham introduced the practice of applying nitrate of silver round the circumference of a patch of erysipelas, in the belief that its spread might in this way be arrested. This was termed the "ectrotic" method, and there are still some who have faith in it, including Volkmann, who prefers the silver salt to the tincture of iodine, which has since been suggested with a similar object. The part must be first carefully washed with soap and water, or with a solution of soda or potash, so as to remove all fat from its surface. It may then be brushed over with a solution of the nitrate in from eight to ten parts of distilled water for a distance of some inches round the reddened area, on all sides of it. Sometimes the disease ceases to spread, and on the following morning defervescence occurs, as is shown in charts given by Volkmann. The application of collodion all over the affected surface is said to be useful as a palliative. I have, however, generally allowed the nurses to carry out the traditional plan

* This was the immediate cause of death in the case of John Stuart Mill, who died at Avignon of facial erysipelas.

of dusting flour upon it. When there is severe smarting pain, or when violent delirium accompanies erysipelas of the head, an ice-bag is said to give great relief, and to be altogether harmless. If the swelling is so great that gangrene seems likely to occur, a series of minute incisions affords the most likely means of preventing it.

It is not often necessary to employ cold baths in the treatment of erysipelas; the pyrexia, although reaching a great height, usually subsides too quickly to be in itself a source of danger. But if a very high temperature is maintained for more than a week, one must carefully consider the question of adopting measures to reduce it. Volkmann gives a chart of a case in which a cold bath was given on the ninth day, the thermometer then indicating 105·8°; in the course of the following week this procedure was repeated thirteen times and the patient recovered.

If death by collapse seems to be impending, it may be well to give turpentine, as was recommended by Copland. At least it is difficult not to accept a case, of which he has recorded the details in his 'Dictionary,' and in which a woman who was already comatose, with a black tongue, and with a pulse that could not be counted, was apparently saved by this drug, administered in doses of three drachms in an electuary of castor-oil and honey.

DIPHTHERIA*

History of the recognition of the disease—Its definition—Its pathology—Relation to croup and to "diphtheritic inflammation"—Micrococci and bacteria—Ætiology—Contagion—Course—Varieties—Complications and Sequelæ—Albuminuria—Paralysis—Prognosis—Treatment.

About the year 1857 attention was first forcibly drawn throughout England to the prevalence of an epidemic disease, resembling scarlet fever in being generally attended with an affection of the fauces, but differing from it in the character of that affection, and in the absence of a rash. Many accomplished physicians, including Addison, declared that they had never seen this disease before; and although a search into medical literature brought to light several instances of its occurrence, both sporadically and in an epidemic form, yet the fact remained that, at least within the present century, it had never previously spread over any considerable part of the country at the same time. But in France it had been well known for several years, and had been carefully studied by Bretonneau, of Tours, who had given to it the name of *diphthérite* (διφθέρα = leather or membrane). In fact the epidemic of 1857 was introduced from France, and was known as the "Boulogne sore-throat."

It is impossible to distinguish, in the accounts of the ancients, between the angina of scarlatina and that of diphtheria, or even of syphilis. But undoubted epidemics of diphtheria occurred in Spain in the seventeenth century, and were described in 1614 by Mercatus, physician to the Kings Philip II and III, and in 1670 by Heredia, physician to Philip IV. It spread to the Spanish provinces in Italy, and was epidemic at Naples in 1618, when it was described by Cortesius (1625), Aetius Cletus (1636), and the celebrated Danish anatomist Bartholinus (1646). It probably appeared in Edinburgh in 1733, according to Fothergill, from whose Treatise on 'The Putrid Sore-throat attended with Ulcers' I have taken the above references. In 1746 there was an epidemic at Bromley, in 1747 one at Greenwich, and in 1749 one in Cornwall, which was described in the 'Philosophical Transactions' by Dr Starr.

Ever since, it has constantly prevailed with more or less severity, springing up here and there in different districts of England, as well as in other countries. Numerous investigations have been made as to its nature and mode of propagation, but in regard to many points doubts still remain, and particularly about its relations to certain other diseases. The designation which is now almost universally applied to it is Diphtheria. Senator, of Berlin, has indeed recently proposed to term it "Cynanche contagiosa;" but this name seems to have no chance of being generally adopted, although its use undoubtedly eliminates many sources of error and confusion, as will be

* *Synonyms.*—Ulcera Syriaca? (Aretæus, c. 70 A.D.), Ulcera pestifera in tonsillis? (Paulus Aegineta, c. 680 A.D.), Epidemica gutturis lues, Angina puerorum epidemica (Bartholinus, 1646), Putrid sore-throat attended with ulcers (Fothergill, 1748).—*Fr.* Mal de gorge gangréneux (Chomel), Angine couenneuse (Louis), Diphthérie (Bretonneau)—*Germ.* Bösartige Rachenbräune—*Ital.* Morbus strangulatorius (Cletus, 1636)—*Sp.* Angina maligna (Heredia, 1673), Garrodillo.

found by anyone who may read Senator's paper in the second series of German Clinical Lectures, published by the New Sydenham Society.

Definition and pathology.—As the term diphtheria implies, the essential character of the throat affection is the presence of a membranous substance, which is spread more or less extensively over the tonsils, the uvula, or other parts. By Bretonneau, and the observers who followed him, great stress was laid upon the circumstance that this substance could be detached from the mucous membrane beneath, and that the latter was then found to be simply reddened and ecchymosed, or perhaps slightly excoriated. They pointed out that the adventitious layer, being of an ash-grey colour, often simulated very closely the appearance which would have been produced by sloughing of the parts covered by it; and, indeed, that the disease had long been known under the name of gangrenous angina (malignant sore-throat). But they declared that such appearances were misleading, and that no considerable loss of substance occurred, still less any extensive sloughing. Finding that in many cases the pellicle extended down into the larynx, they formed the opinion that the complaint known as membranous croup was only a form of diphtheria.

By German writers the subject has been developed in a very different direction. Virchow, in the first volume of his well-known 'Archiv' (published in 1847), distinguished between a "croupous" form of inflammation in general and a "diphtheritic" one. In the former, he said, the exudation lay free upon the surface of the mucous membrane; but in the latter it was seated within the superficial layer of that membrane, which generally underwent sloughing as the result of its presence. These definitions, being followed by subsequent writers without a due comprehension of what probably was Virchow's real meaning, have led to great confusion, which is only beginning to be cleared up. The best description of the characters of "diphtheritic inflammation," in the sense attributed to it by the great leader of the Berlin school, appears to me to be that given by Rindfleisch in his 'Pathological Histology.' He makes it consist of an infiltration of newly-formed cells into the subepithelial connective tissue of the mucous membrane; and, following an idea originally suggested by Buhl, he teaches that the abundance of this infiltration compresses the blood-vessels, and so arrests the circulation through the affected parts, brings their nutrition to a standstill, and deprives them of life. As examples of such an affection, he cites the putrid inflammation of the urinary bladder that is set up by decomposition of stagnant urine, the more severe forms of dysentery, and the dangerous inflammation of the uterus and vagina which may occur immediately after parturition; and he points out that a similar condition may be met with upon other free surfaces; upon the skin, as in the more destructive kinds of variolous eruption, which lead to permanent pitting; and upon wounds, as in hospital gangrene. It must be admitted that all these affections have characters in common, and deserve to be known by a special name.

But, if this view is to be taken of the distinguishing features of "diphtheritic" inflammation, one sees at once that the disease "diphtheria" finds no place in it. And, accordingly, Rindfleisch describes the latter under croupous inflammation, and by the name of "pharyngeal croup." He gives a very full account, which I can confirm by my own observations, of the histological characters of the pellicles which are found upon the surface of the mucous membranes in that complaint. On the palate and tonsils he says they consist, not of fibrin, but entirely of cells, which have undergone

a peculiar glassy change in their protoplasm, and have become fused together, so that a series of little fissures alone indicates the original interspaces between them. According to E. Wagner, these cells are all derived from the original epithelial elements of the affected part. In the air-passages, on the other hand, Rindfleisch describes the pellicles as laminated, and consisting of layers of cells which alternate at tolerably regular intervals with layers of a homogeneous substance, apparently fibrin.

Some writers have attached special importance to the fact that in different affections of the throat there are great variations in the degree to which false membranes adhere to the mucous surface on which they lie. But Rindfleisch points out that these variations depend mainly upon the *seat* of the lesion. The lining membrane of the air-passages possesses a smooth basement-membrane which is wanting in the fauces, and it is suggested that this prevents adhesion. As between the several diseases the distinction is valueless.

The fact that in the very same case of diphtheria the membrane on the tonsils and that in the larynx differ essentially in their histology is of itself sufficient to show that our views in regard to the disease are not to be based on its pathological anatomy alone. And, in the fauces themselves, the morbid changes present wide variations of intensity. There is, in the first place, a "*diphtheria sine diphtherâ*;" a variety in which the tonsils and uvula are merely reddened and affected with catarrhal inflammation, but which can be plainly recognised as diphtheria, because it occurs in members of the same family simultaneously with the more severe forms. And when membranes are present, they differ greatly in extent and thickness in different cases. Rindfleisch, indeed, speaks of the morbid process in the pharynx as being always insular, and as consisting of circumscribed milk-white spots, on an intensely hyperæmic base, which are never raised more than half a line above the level of the mucous surface. But no physician who has had much experience of the disease can be ignorant that in many instances the whole fauces, tonsils, and uvula are covered with a thick tough layer, which looks like wash-leather, and may ultimately be detached *en masse*. In 1858, Dr Fuller exhibited to the Pathological Society a cylindrical cast of the pharynx four inches long, which had been ejected by a girl aged eleven. Indeed, if such false membranes are removed, or become detached, they may be renewed again and again. This is, I think, well known to all who have observed severe epidemics of the disease; and Dr Sanderson vouches for it as the result of his own observation in 1859. "At Crowle," he says, "I had an opportunity of seeing an example of extreme rapidity of reproduction of faucial and pharyngeal concretion; and I have notes of a case, in a robust adult at Hertingfordbury, watched with the greatest care and attention, in which on some of the mucous surfaces the pellicle must have been renewed from twenty to thirty times in the course of three or four weeks." I quote this statement because Rindfleisch expressly denies that false membranes on the palate or tonsils ever recur in the same place, unless they have been stripped off before their spontaneous maturation; and Oertel (in his article in Ziemssen's 'Cyclopædia') likewise limits the possibility of their reproduction by conditions which I cannot fully comprehend. Another point on which Sanderson speaks very positively is the frequent occurrence of ulceration, which, in two cases that he observed, led to perforation of the palate; and he mentions one instance in which the tonsil and the arch of the velum were destroyed, so that a continuous sloughing surface extended to the

pharynx. Bretonneau observed that in cases which presented exactly the appearance of sphacelus,—the uvula and tonsils in a state of "putrid dissolution," and almost entirely detached,—the fauces were found after all to be intact, when the false membranes at length came away; but all recent writers, both French and German, admit that Bretonneau went too far in denying the possibility of gangrene.

Microphytes.—Within the last few years some observers have endeavoured to cast the whole subject of diphtheria into a new shape, in consequence of the discovery that the structures concerned in the disease are permeated by living organisms. This discovery, which seems to have been made by Buhl in 1867, and by Hueter and Oertel independently in 1868, is one of which the real value is still doubtful; but I must briefly recapitulate the main facts which have been brought forward. The organisms in question are among the most minute that are known to exist. They are usually spherical in form, *micrococci*, single, as dumb-bells, in chains or as zooglœa; but mixed with these are some which are rod-shaped, and which are known by the name of *Bacterium termo*. To distinguish the former from mere granules of albumen or fat is very difficult; some observers think that the way in which they are massed together shows that they have been developed by a rapid process of germination. The latter present a more characteristic appearance, especially as they are often arranged in long chains; but then precisely similar bodies exist in abundance in all putrid fluids.

According to the writers who lay stress upon the existence of these parasites in diphtheria, they are found not only in the false membranes (both within the epithelial and exudation cells, and outside them), but also in the subjacent structures. They are described as being present from the very commencement of the morbid process, and as extending beyond the limits which appear to the naked eye to separate the affected from the healthy tissues. They are said to block up the lymphatic channels, to penetrate into the interior of muscles, cartilages, and bones, and to be carried by the blood to distant organs, such as the kidneys, where they germinate afresh.

Now, there is, I believe, no question that micrococci are really present in the tissues primarily affected by the diphtheritic process. But many authorities doubt the validity of the observations in which they have been supposed to be detected in the blood. And the investigations of Dr Creighton have shown how great caution is required in the interpretation of such appearances as those which have been held to indicate the occurrence of organic forms in parenchymatous organs, such as the kidneys.

But an important distinction must now be drawn. The first observations of Hueter were made, not upon cases of "diphtheria," as we define the disease, but upon those of hospital gangrene. More recently Heiberg has discovered micrococci in the vegetations of ulcerative endocarditis. And Virchow met with similar organisms in the kidneys of women who had died of puerperal fever. Now, no clinical observer will maintain that these affections are clinically identical with diphtheria. In other words, the parasitic theory is applicable rather to "diphtheritic inflammation" in the wider sense of that term than to "diphtheria."

The conclusion just stated appears to me to be also fairly deducible from a large number of experiments, made by different observers, in which animals have been inoculated with "diphtheritic" products. Portions of false membrane have been inserted beneath the skin, or introduced into the

trachea of the dog or the rabbit; or the cornea of the eye has been punctured with a poisoned needle. The result has been a disease which proved fatal in two or three dogs. The tissues in the neighbourhood of the spot to which the infective material was applied have been found full of masses of micrococci; and Oertel says that he detected these organisms even in distant parts. But Recklinghausen succeeded in generating a "diphtheritic keratitis" in a rabbit by inoculating the cornea with matters from a case of hospital gangrene. In Oertel's series of experiments, he passed on the infection from one animal to another, choosing sometimes the trachea, and sometimes the muscles of the neck or chest, as the seat of the induced disease; and after six transmissions he obtained a product capable of giving rise to the formation of a false membrane in the air-passages of the last animal experimented on.* One may reasonably suppose that this result was due to some specific property of the material used for inoculation, other than its mere power of infecting the tissues with micrococci.

Indeed, were it not for the experiments just referred to, we should be strongly disposed to think that the constitutional disturbance caused by this so-called "diphtheritic" inflammation, excited by inoculation in the lower animals, is perhaps after all nothing but a form of septicæmia; and that the substances employed act merely as other decomposing substances might act. In taking such a view of the matter we might suppose that the presence of micrococci in the affected tissues in cases of diphtheria simply indicates the unhealthy character of the inflammation. This notion accords well with the statement of some recent writers that the false membranes in the larynx and trachea contain scarcely any living organisms; for, as we shall presently see, those cases of diphtheria in which the air passages are affected commonly end fatally before a septic process begins.†

Is diphtheria primarily local?—One important question in regard to the pathology of the disease has still to be considered, namely, whether it is from the first constitutional, or whether Oertel is right in maintaining that it is originally local and infects the system secondarily. His chief argument is based upon a contrast between the results obtained in the experimental transmission of diphtheria to the lower animals, and those which follow the inoculation of horses with glanders. In the former case, he says, the affection always develops itself at the spot to which the poison is applied; in the latter it invariably affects the nasal membrane, even though the contagious principle should have been inserted beneath the skin. But it is to be observed that even this view of the matter leaves diphtheria in the same position as syphilis and variola. I think there can be no doubt that the disease has really a special tendency to attack the fauces, and that the frequency of throat-diphtheria does not merely mean that the poison comes into contact with this mucous surface more frequently than with any other. At any rate, it is certain that when the affection has begun elsewhere the throat often suffers secondarily, and apart from any direct extension of the morbid process. Thus a man was admitted into Guy's Hospital with an affection of the prepuce of a doubtful nature, but before his death the palate

* "Experimentelle Untersuchungen ü. Diphtherie," 'Deutches Arch. f. klin. med.,' 1871, Bd. viii.

† On this difficult subject, compare Cohn's statements as to the distinction between *Micrococcus septicus* and *M. diphthericus*. 'Beitrage zur Phys. der Pflanzen,' 2te Heft, p. 164, *et seq.*) Eberth ('Zur Kenntniss bact. Mycosen,' 1872), and the 'Report of the American National Board of Health for 1882' are also referred to by Klein.

and tonsils became covered with a false membrane, so as to clear up the nature of the case. And in epidemics of conjunctival diphtheria, of which several have occurred in Berlin, the fauces have occasionally been attacked.

Ætiology.—The first point to be insisted on is that diphtheria is contagious. This, indeed, is not always a very obvious feature of the disease as it is seen in private practice; for if several cases occur in succession in the same house, one cannot say whether they all depend upon a common cause, or whether one has given rise to another. But when a patient suffering from diphtheria is admitted into a hospital, it often happens that persons occupying beds in the same wards are subsequently attacked. And Sir William Jenner relates several instances in which patients sent into the country infected those with whom they came into contact. The epidemic in East Kent in 1856 was ushered in by a striking instance. "No case of diphtheria had ever been seen in Folkestone," says Mr Eastes of that town, "during my time, until Isabella W—, aged $4\frac{3}{4}$, arrived from Boulogne on the evening of July 2nd, being then in an advanced stage of the disease. She died on the following day. On the 6th, her sister, aged ten, was attacked, who had always resided on the East Cliff; another case occurred in the same house three days after, and they all terminated fatally." But the most conclusive of all, perhaps, are the instances in which a medical man has fallen sick with diphtheria, immediately after having had phlegm coughed into his mouth or nose by a patient whose fauces he was examining; or after having used his lips to inflate his patient's lungs, or to blow through a tracheotomy tube. Oertel mentions by name five physicians whose lives were thus sacrificed, among whom was Valleix, the writer on neuralgia. A recent lamentable case of death from such self-devotion occurred in the person of a house surgeon at King's College Hospital, and it was believed that the late Princess Alice contracted the disease by kissing her child who was ill of diphtheria. A case belonging to the same category is that of Dr Wiessbauer's child, who was attacked shortly after having put into his mouth a cannula that had just been removed from the throat of a patient suffering from diphtheria.

But even when the conditions seem to be altogether favourable to the spread of diphtheria, it sometimes fails to propagate itself. Those in attendance upon patients suffering from diphtheria have been known to blow through tracheotomy tubes for the purpose of cleaning them over and over again without suffering any ill-effects, and Trousseau and Peter actually inoculated themselves on the palate and tonsils with diphtheritic matter, and were none the worse. There is not, indeed, anything very surprising in these facts, for persons exposed to contagion from other diseases not infrequently escape. But many writers seem to think that diphtheria is infectious in a much lower degree than the exanthemata. Thus Oertel says that it diffuses itself over a district much more slowly than they do. However, I have long thought that the doctrine of the comparatively innocuous character of this complaint is in great part based upon the fact that so many physicians include under diphtheria what I regard as a non-infectious malady, namely, Croup. The question is one of great difficulty, and it can more conveniently be discussed hereafter. But I may here remark that unless one allows that many cases in which the air-passages become lined with false membranes, even when the fauces are likewise affected, are distinct from diphtheria—one must recognise and account for the fact that such cases can seldom be traced to other cases of diphtheria, whether as having arisen from

them, or as giving origin to them. Thus in the 'Guy's Hospital Reports' for 1877 I have recorded a series of fifty cases of what would be regarded as diphtheria in the common acceptation of the term; and whereas eleven of fifteen cases in which the larynx was free could be brought into connection with other cases (by either ascent or descent) only eight out of thirty-five cases in which the air-passages were involved could be so brought; moreover, all the eight cases just referred to were cases in which the fauces were severely affected; of ten cases in which there were only very slight patches of false membrane upon the tonsils or palate, there was not one in which any evidence of infectiousness was obtained. I shall hereafter have to discuss whether these facts must be taken as showing that diphtheria attacking the air-passages is actually less contagious than usual, or whether they indicate that a large proportion of the cases in question really belong to a different disease, a simple non-specific inflammation, attended with the formation of false membranes.

In support of the opinion that diphtheria is highly infectious, must be mentioned the fact that its contagious principle sometimes adheres with great obstinacy to particular houses or apartments. Squire mentions that in a country house in Scotland a visitor was attacked while occupying a chamber in which a case had occurred eleven months before. The infection from a mild case may generate a severe one, or *vice versâ*. How soon a patient ceases to be infectious is not yet known. Bristowe says that the disease has sometimes seemed to be communicated by a child who had apparently been well for two or three weeks.

Presence with other specific fevers.—In speaking of measles and of scarlet fever I have mentioned that each of those complaints is now and then accompanied by an affection of the fauces resembling that of diphtheria. A fact related to Dr George Johnson by Dr Dewes, of Coventry ('Lancet,' 1875), tends to show that both the specific contagia are present in some cases of this kind. Two brothers had been in succession attacked by scarlet fever, and had been separated from one another throughout the course of the disease. The elder had remained at his school to be nursed; the younger had been sent to a cottage in the country. The former passed through a mild form of the disease; the latter had it severely, and also had his fauces covered with diphtheritic exudation. After a time, the boy at the cottage sickened with scarlet fever; and he, too, had a diphtheritic throat. When both brothers had been convalescent for a fortnight, they were allowed to be together, and even to lie in the same bed. Presently the elder one fell ill of diphtheria with laryngeal complications, and died on the third day. A point to which Senator draws attention is that when scarlet fever is accompanied with the formation of false membranes upon the fauces it scarcely ever happens that the morbid process extends to the air passages, although the throat affection may be apparently identical with that of diphtheria.

Predisposing causes.—Reverting to the question of the causes of diphtheria, one must admit that the disease often springs up in isolated houses, and under circumstances which accord ill with the theory that it must be due to a specific contagion. In this there is not, indeed, anything to be wondered at; for the same thing is likewise true of all the exanthemata. But Dr George Johnson has advocated the opinion that cases not directly traceable to infection are often due to contaminated water, or to the effluvium from foul sewers or cesspools. He relates the cases of four children, who were

all attacked on the same day; three days previously, a cesspool distant about twenty yards from the house had been emptied; and the wind was blowing towards the house at the time, and conveyed to it a very offensive smell. Soon afterwards two of the servants fell ill, and at length the mother. The only members of the household who escaped were the father (who was away from home all day) and one servant.

There is reason to believe that, even when diphtheria is epidemic, and when the specific contagion has perhaps already been introduced into the system, exposure to cold may act as an exciting cause in bringing out the manifestation of the disease upon the fauces. At least Dr Yeats ('Ed. Med. Jour.,' 1876) states that this was the case in an epidemic which occurred in Auchtergaven, Perthshire, between March and June, 1875. Several persons, he says, were attacked who had been working late in their gardens, or playing croquet on a damp lawn, or driving after sunset. Others, who had been confined to bed for a considerable time from other causes, were infected on their first appearance in the open air. It is obvious that these cases have an important bearing on the question of the existence of a membranous croup distinct from diphtheria, since the former affection is supposed to be due merely to exposure to cold.

Whether cold, altogether independently of epidemic influences, can ever set up a simple, non-specific inflammation of the fauces attended with the formation of false membranes, it is very difficult to say; I shall have to revert to that question when discussing the relations of croup to diphtheria. It is at any rate certain that such a morbid process may result from the action of more powerful irritants.

For example, I have related in the 'Guy's Hospital Reports' for 1877 the cases of two children, in whom the palate and tonsils presented appearances exactly like those of diphtheria; but in one of them the affection was caused by swallowing a piece of hot potato, in the other by a burning stick, which had been thrust into the little patient's mouth by another child. A similar condition has been met with in three or four cases of scald of the throat, in which boiling water or steam had been sucked out of a teapot or kettle. Lately I found a well-marked separable false membrane on the lingual surface of the epiglottis, as well as on the base of the tongue, in a woman who committed suicide with white precipitate, but who lived six or seven days after swallowing the poison. There was extensive "diphtheritic inflammation" of the œsophagus, the stomach, and the lower part of the intestines.

It is well known that, instead of attacking the throat, diphtheria sometimes shows itself on the mucous membrane of the genital organs, or of the eyes, or upon denuded parts of the cutaneous surface. The study of such forms of the disease ought to throw great light on its ætiology. Dr Braxton Hicks has recorded in the 'Guy's Hospital Reports' for 1871, an epidemic of diphtheria in the obstetric wards, in which two women were in turn affected with diphtheria of the labia, while two others had the ordinary affection of the fauces. Moreover, in Berlin and some of the other cities of North Germany, where conjunctival diphtheria has prevailed to a remarkable extent, I believe that in each epidemic there have been some cases in which the disease has spread either downwards through the lacrymal passages to the palate and fauces, or in the reverse direction upwards, or in which the throat and the eyes have been attacked simultaneously and independently. Moreover, the conjunctival affection, as I myself have

observed, sometimes occurs as a sequela of measles, just as is the case with diphtheria of the fauces. The existence of so close a relation between these different forms of the disease affords a strong argument for its specific character, and against the supposition that it arises spontaneously, as the result of defective hygienic conditions. But it must be added that both Jacobson (of Königsberg) and Hirschberg admit a sporadic variety of conjunctival diphtheria; stating, for instance, that gonorrhœal ophthalmia often assumes a diphtheritic character. And it may be that the affection of wounds and raw surfaces known as hospital gangrene is a sporadic representative of cutaneous diphtheria. Jacobson thinks that the conjunctival disease, even when it is of purely local origin, may spread by contagion. I should hesitate to admit this; although it would certainly be somewhat analogous to the case of contagious porrigo of the skin, which often seems to develop itself out of simple eczema.

Whether the poor are proportionately more liable to diphtheria than the rich is perhaps a question. Sir William Jenner remarks that only a small number of his cases occurred in his hospital practice; and it is notorious that the disease often carries off very healthy and robust-looking children. All writers say that it is far more apt to attack those who are under ten years of age than older persons; and there appears to be no doubt that this is the case, although I myself think that many observers have made the proportion of children appear unduly large by including membranous croup under diphtheria, since the former affection never occurs in adults.

There seems to be no proof that one season of the year rather than another is favourable to the spread of diphtheria. The very cold winter of 1860 did not hinder its epidemic diffusion in London; and it has often prevailed during the summer. Fothergill found it far most frequent in September to December inclusive. Oertel says that diphtheria is of much less frequent occurrence towards the tropics than in the temperate zone and the adjacent part of the frigid zone. Geological conditions of soil seem to have no part in its causation.

Protection.—Does the fact that a person has had diphtheria imply subsequent immunity from the disease? In Simon's 'Report for 1859 several cases are recorded of individuals who suffered twice, at intervals of two or three months or longer. And it is to be noted that the second attacks were always more severe than the first ones. Second attacks are not uncommon according to Dr Eustice Smith.

Incubation.—This period in diphtheria is said by Trousseau to be from two to seven days. Oertel puts it at from two to five days; but he quotes cases in which it was longer. One such instance is related by Jenner; that of a young lady who sickened eight days after having been sent into the country from a house where her brother and her sister had before been attacked. Senator even says that the interval may be as much as three or four weeks. Where there has been a direct transference of the poison from one person's fauces to another's, the period seems to be much shorter. Valleix had a pellicular deposit in the tonsil the day after he became infected with the disease, and died in forty-eight hours. Oertel supposes that the incubation is likely to be less prolonged during the prevalence of an epidemic, especially where the type is malignant.

Course.—The symptoms of diphtheria differ widely in different cases. Sometimes the disease begins with marked constitutional disturbance: ano-

rexia, headache, lassitude, and loss of energy ; nausea or vomiting, acceleration of pulse, and shivering, followed by fever. There may from the first be difficulty of swallowing, the sensation varying from a slight pricking up to a severe darting or shooting pain which may quite prevent the patient from taking food, but this pain is rarely so extreme as in acute tonsillitis. On looking at the fauces one finds that the uvula, the palate, and the posterior wall of the pharynx are more or less swollen and of a red or violet colour. After a few hours, or at latest two days, one or more whitish grey spots begin to appear on some of those parts ; they are at first small, and they may remain of the same size for several days. From an early period the lymphatic glands of the neck, especially those near the angles of the jaws, become swollen and painful.

In some instances the febrile disturbance is very slight, and in others when it is more considerable, it is of very short duration. The patient often seems to have entirely recovered within two or three days, by which time the pellicles are sometimes detached and cast off.

But in many cases the disease takes a less favourable course. The fever continues, the temperature remaining at 102° or 103°, or mounting higher ; or, if it should have fallen, it rises again on the fourth, fifth, or sixth day. The diphtheritic spots on the fauces rapidly run together, and extend in every direction ; so that the tonsils, the uvula, and all the visible parts of the throat may in a few hours be covered with a yellowish or greyish membrane. The cervical and submaxillary lymph-glands swell still more, and the tissues about them may be so infiltrated with inflammatory products that the hollow of the neck is entirely obliterated, the whole space from the chin to the sternum being occupied by a uniform brawny mass, the skin over which is shining and red, as in erysipelas. Presently the diphtheritic membranes begin to soften and decompose, acquiring a brownish colour, and separating here and there in shreds. The patient's breath then becomes horribly fœtid. An ichorous discharge may run from the corners of the mouth, which excoriates the surface and gives rise to ulcers which in their turn become covered with a greyish-white layer. At this time it is not uncommon for sore spots—behind the ears, for instance, or in the creases of the skin, even of remote parts of the body—to become distinctly diphtheritic. If a blister should unfortunately have been applied, the raw surface may be coated with a membrane ; and leech-bites are apt to pass into unhealthy spreading ulcers.

From the first commencement of these more serious local changes, the constitutional symptoms begin to present features of depression which characterise the whole further progress of the case. The pulse may still be quick, its beats rising to 120 or more in the minute, but it becomes daily smaller and weaker. The countenance acquires a waxy pallor, which perhaps contrasts strongly with the ruddy aspect of the patient a few days before ; the muscular power is greatly enfeebled. Purpuric spots often appear upon the skin. Delirium sometimes sets in, as in a case recorded by Sir William Jenner ; the tongue may become dry and brown, sordes may collect on the teeth and lips, and in fact a "typhoid" state may be developed. Such cases are generally fatal, and on *post-mortem* examination the serous membranes, and even the endocardium and the substance of the lungs are commonly found to be ecchymosed, as in septicæmia. Pneumonia or œdema of the lungs is, in many instances, the immediate cause of death.

Most frequently the mind remains perfectly clear. At length the tem-

perature falls below normal, to 97° or 96° F., and the pulse may become irregular and intermittent, its beats scarcely amounting to 50 or even 40 in the minute. Death may either occur very gradually by asthenia, or more often it takes place suddenly while the patient is in the act of sitting up in bed or making some other slight movement. Such cases generally terminate between the tenth and the fourteenth days.

Fatal syncope, however, is not limited to those cases of diphtheria in which the disease has been severe throughout its whole course; it sometimes happens when all the symptoms have been of the slightest. Jenner relates such an instance: a boy, aged ten, who was convalescent from a very mild attack, was attacked with vomiting. His medical attendant found, on visiting him as usual, that the pulse (which had been becoming less frequent for two days) was only 36 in the minute. There was nothing in the patient's appearance which would have suggested that he was in imminent danger of death, but notwithstanding the free use of stimulants the pulse continued to fall; by the next afternoon its beats were only 24, and soon afterwards they ceased altogether. I have seen two similar cases in children. One was that of a little girl in whom there were all along well-marked diphtheritic patches in the fauces, but who seemed to have so little the matter with her that she was allowed to play with other children in the garden in front of the house. Early one morning she was being brought downstairs from the nursery as usual before being dressed, when it was noticed that she looked very pale, and I was hastily sent for. I found her pulseless with her extremities perfectly cold, and in spite of all that could be done she died about eighteen hours afterwards, without having rallied in the least from her collapsed condition. I also remember a third case, which I believe to be of the same kind, but in which there was no direct proof that the disease was diphtheria. It was as follows: A man, aged fifty, a sailor, was admitted into Guy's Hospital under the late Dr Barlow on June 24th, 1866. His pulse at the wrist was imperceptible, and he was unable to swallow. When he took any liquid into his mouth he made a gurgling noise and spat it out again. He was perfectly conscious, but his speech was thick, and he dragged his legs after him and was unable to walk without support. He said that he had been perfectly well until the morning of the 19th (five days before he was brought to the hospital), when "he found himself unable to speak; his throat afterwards felt sore and seemed full of phlegm, and his breath was short." On the day after his admission he had a sort of fit, and threw himself out of bed. This was followed by a paralytic condition of all the limbs, and he seemed as if he would die at once; but he rallied a little and lived until 1.40 a.m. of the 26th. A *post-mortem* examination was made; no disease could be found in the body, and the only possible conclusion seemed to be that the case was one of diphtheria: but neither during life nor after death were any definite morbid appearances discoverable in the fauces.

Even when characteristic diphtheritic patches exist on the mucous membrane of the fauces, it sometimes happens that the real nature of the disease may be overlooked from the patient failing to complain of difficulty of swallowing or pain in the throat. I have had children brought to me in the out-patient room at the hospital, suffering from febrile symptoms for which I could discover no cause until, as a matter of routine, I examined the fauces. And some years ago a chlorotic girl, who had been an inmate of one of the wards for a few days, died without anyone suspecting the real nature of her disease. The chief symptom was a weakness so extreme that she

could not sit up even when supported. A *post-mortem* examination showed that the fauces were covered with false membrane.

But perhaps the most obscure of all the forms of diphtheria is one which is limited to the *nasal mucous membrane*, or which, at least, may be unattended with the presence of any obvious pellicles upon the pharyngeal surface. The chief local symptom is then the escape of a thin sanguineous or muco-purulent fluid from the nostrils, the orifices of which become more or less reddened and excoriated. Epistaxis, however, is not infrequent, and it may be so profuse that one would have been satisfied to attribute to it the bloodless appearance of the patient if it had not been for the fact that anæmia is so constantly present in all severe cases of diphtheria. With a speculum we may sometimes perceive that the turbinated bones are covered with a membranous layer, or casts of them may be discharged from the nostrils. After the first day or two the secretion from the affected parts is commonly of a brownish colour and very offensive. The disease not infrequently extends along the nasal duct to the conjunctiva, which then becomes coated with a perfect diphtheritic membrane. Or it may pass through the Eustachian tube to the tympanum, causing a singing or buzzing noise in the ears and deafness; perforation may then take place, and matter be discharged through the external meatus.

Another direction in which diphtheria sometimes spreads, is from the pharynx down the *œsophagus*. In one fatal case I found a number of small ulcers in the stomach close to the cardiac orifice, some of which were coated with a distinct layer of false membrane; and instances have been recorded in which the whole œsophageal and gastric mucous membranes have taken part in the morbid process. This condition seems to have given rise to no special symptoms.

Laryngeal diphtheria.—Of all the secondary forms of the disease which result by extension from the fauces to adjacent parts, the most important by far is that which affects the *air passages*. The continuity of the false membranes is often traceable directly over the epiglottis and the arytæno-epiglottidean folds; but sometimes this is not the case, and certain observers have in consequence been led to regard the spread of the disease to the larynx and trachea as the result of what they have termed "auto-infection," an inoculation of the mucous membrane by particles of secretion drawn downwards with the air inspired through the mouth. Upon the epiglottis and the vocal cords the false membrane is firmly adherent; it may pass straight over the space between the true and the false cords. Below the glottis it is very loosely attached to the mucous surface. It becomes thinner as it descends, and in the trachea, at a variable distance down, it commonly ceases, and becomes continuous with a muco-purulent layer which lines the rest of the air passages. But in some cases, even the bronchial tubes within the lungs present a delicate tubular diphtheritic lining. Oertel quotes Bartels as having pointed out that this is always confined to those tubes which run upwards or downwards in the back parts of the lungs, and never occurs in those which course forwards towards their anterior edges. These writers also maintain that the existence of old pleuritic adhesions over any portion of a lung favours the penetration of the fibrinous exudation into the corresponding bronchial tubes.

It is seldom practicable to employ the laryngoscope to determine the presence of a false membrane in the air passages in a case of diphtheria, and only a very practised observer would be able to obtain a satisfactory view

of the interior of the larynx, at least in a child. The diagnosis of laryngeal diphtheria rests mainly upon the fact that the entrance of air into the lungs is impeded. This is shown not only by the rapidity and gasping character of the respiration, but also by the way in which at each breath the soft parts above the clavicles and sternum are sucked in, as well as the lower intercostal spaces, and in young children even the sternum and ribs, which at an early age are soft and yielding. Every time that the patient inspires a loud crowing or croupy noise may be audible, and the cough is often hard and brassy. In other words, the symptoms are precisely the same as those of Croup, and as I have already stated more than once, some observers believe that croup, when attended with the development, of membrane, is always an effect of the diphtheritic poison. The dyspnœa is apt to become greatly aggravated from time to time, a circumstance that must be due either to the supervention of spasm in the muscular walls of the affected parts, or to the accidental impaction of portions of membranous or other secretion in the narrowed chink of the glottis.

When diphtheria affects the larynx, extension of the disease to that part commonly occurs within from three to six days after the commencement of the disease. Jenner, in 1861, had never known it delayed beyond the end of the first week ; but Oertel says that it is not infrequent on the eighth or tenth day, and may be as late as the thirteenth. In Jenner's cases death always occurred in five days from the setting in of laryngeal symptoms ; and he says that out of twenty-six fatal cases of Bretonneau's there were only five in which life was prolonged after the third day, and that there was but one in which it was prolonged after the sixth day, except as the result of operative interferences.

Were it not for the circumstance that the presence of false membranes in the air passages is in itself dangerous to life, from mechanical interference with respiration, it is probable that many cases in which the disease assumes this form would be of a mild type. In other words, one need not wonder that the laryngeal variety of diphtheria, even when it terminates fatally, is often unattended with those symptoms of bodily prostration, anæmia, and depression of the heart's action, which play so prominent a part in many uncomplicated cases of the disease.

It is said that subpleural emphysema is of frequent occurrence from distension and rupture of the pulmonary alveoli, and that the escaped air may diffuse itself into the mediastinum and even into the subcutaneous textures of the neck and of the body generally. Bartels, in one case, heard a loud sound on auscultation, which he supposed to be due to pericarditis, but which proved to depend upon the presence of a quantity of air in the areolar tissue outside the serous sacs.

In certain cases, diphtheria seems to commence in the air passages, there being no primary affection of the fauces. In the epidemic form of the disease, however, this is of rare occurrence. Bretonneau states that he met with but two instances of it ; of one of these he gives details (Case 45 in his fourth 'Memoir'). But I think that there is no more than a presumption against its being a case of simple membranous croup—unless, indeed, we absolutely deny the existence of that disease. The patient was an infant, a year old, in charge of a nurse at Tours, where no case of diphtheria had been seen for months. The nurse was a native of a hamlet some miles distant, where the disease was prevailing, and a nephew of hers had died of it a few days before the infant fell ill. Bretonneau puts

the relative frequency of a primary laryngeal diphtheria at one in thirty cases; Guersant at one in twenty cases. In the epidemic which occurred at Auchtergaven, in Perthshire (cf. p. 266), Dr Yeats observed among one hundred and eighty-three cases, fifteen in which laryngeal symptoms were present from the commencement, but in which there was no visible affection of the fauces when they were first brought under notice; and in six of these the pharynx remained free throughout the whole progress of the disease.

When we come to discuss the question of croup, we shall find that it is important to know what is the proportion of cases of epidemic diphtheria in which the morbid process spreads to the larynx; but with regard to this I have been able to find no satisfactory information. In the reports made in 1859 by Greenhow and Sanderson to the Privy Council, in reference to the epidemic which had been prevailing for three or four years in various counties of England, it is generally spoken of as exceptional. Among a large number of cases occurring at Spalding in Lincolnshire and recorded by Dr Capron, there are only two in which it is mentioned. Out of twelve fatal cases that were seen by Mr Schofield, of Highgate, nine terminated by "asthenia," three only by "croup." At Birmingham it is reported that croupy symptoms were comparatively rare. Mr West had never seen any marked affection of the larynx, and Dr Heslop did not think that such complications occurred in more than 5 per cent. of the cases. Only at Stalham and at Smallburgh is it said that the majority of patients died with croupy symptoms or that such symptoms were very common. So, again, at Crowle, Sanderson reported that he had "no means of determining in how many cases the symptoms of laryngeal complication existed; it is certain, however, that they bore a small proportion to the fatal cases. At Launceston, on the other hand, out of twenty deaths, it appears that eighteen were preceded by the symptoms of croup." Now, it is true that in a schedule of questions, previously sent round to practitioners in the districts where diphtheria prevailed, it had been asked whether or not "the diphtheritic inflammation . . . *sometimes* descends along the mucous membrane of the air passages;" so that the medical men in question must have seen that the disease was regarded as distinct from the well-known membranous croup, and may to some extent have been influenced in excluding from their returns cases in which the main symptoms were laryngeal. But I think that no one can read their reports without coming to the conclusion than an extension of the disease to the larynx and trachea was comparatively infrequent.

On the other hand, the assertions of French physicians as to the extreme frequency of such extension are deprived of most of their value by the very fact that these physicians recognise no membranous croup apart from diphtheria. Bretonneau's 'Memoirs' contain forty-five cases, related in detail, and in the immense majority of them the air passages were involved. But one must remember that the main object of this writer was to "establish the identity of croup with malignant angina," under the common name of diphtheria; and in one place, after recording an ordinary instance of "pharyngeal diphtheritis," he says, "a larger number of special cases of this kind would present no interest."

It seems to me that Trousseau's statement that diphtheria extending to the air passages is the most common form of the disease requires allowance to be made for the same fundamental belief on his part. For he proceeds to say that this is the form which diphtheria takes when sporadic, and also that which

it exclusively assumes in severe epidemics. Now, the former assertion involves the very question at issue, namely, whether sporadic membranous croup is a manifestation of diphtheria. As I have already stated, I found in 1877, in putting together a series of cases of diphtheria and croup which had occurred at Guy's Hospital, that among fifty cases, such as would commonly be regarded as examples of the former disease, there were thirty-five in which the air passages were affected. But I shall hereafter endeavour to show that in many of them the disease must have been a non-specific inflammation.

Albuminuria.—The urine is not only scanty and high coloured, as in other acute diseases, but it very commonly contains a considerable quantity of albumen. This fact, which was first pointed out by Dr Wade, of Birmingham, in 1858, is of great value, as sometimes aiding in the diagnosis of cases that would otherwise be obscure. There may also be epithelial and hyaline casts; and, much more rarely, blood. Eberth is quoted as having found that in his experience albuminuria was present in two cases out of three; but the proportion seems to vary in different epidemics. In some patients it is observed within a day or two from the commencement of the disease; in others not until convalescence already seems to be established. It is sometimes very transitory, and may be detected only once or twice, even in cases in which the urine is repeatedly examined; but it more often lasts for a week or two. It is generally supposed to be devoid of prognostic significance; but Oertel, although he admits that the urine of some patients who die rapidly contains very little albumen, yet says that he has been able to make out a close relation between the quantity of this substance excreted in the twenty-four hours, and the general intensity of the disease. From one to three drachms was the amount usually passed in cases so severe as to threaten life, or to terminate fatally; and the albuminuria persisted, in those who recovered, for six or eight weeks after the subsidence of the diphtheritic affection of the throat.

When an opportunity is afforded of observing the state of the kidneys, they are said to be found large and congested, the epithelial cells being swollen, opaque, and granular, and filling the tubes. Minute extravasations of blood are also described as present in many cases; and, according to Oertel, masses of lymph corpuscles often surround the capsules of the Malpighian tufts. The observations which have been made at Guy's Hospital would, however, suggest the belief that the affection of the kidneys differs widely in degree from scarlatinal nephritis; that observed in cases of diphtheria being comparatively of a slight character. And this accords with the well-known fact that dropsy very rarely occurs after diphtheria. A few instances of general anasarca have, indeed, been recorded by different observers, one, for example, by Oertel in the 'Deutches Archiv' for 1871. But when this writer speaks (in 'Ziemssen's Handbuch') of fifty fatal cases, mostly attended with suppression of urine and dropsy, as having occurred in Kiel and the neighbouring villages, the doubt arises whether the epidemic was not really one of scarlet fever with diphtheritic complications.

It has been recently maintained by MM. Bouchut and Labadie-Lagrave that *endocarditis* is of frequent occurrence in diphtheria. I have repeatedly searched for such an affection in making autopsies in children who had died of the disease; but the valves have always appeared to me to be perfectly healthy. And Sanné (who has had good opportunities of testing the value of the statements in question) declares that the slight irregularities that are

commonly found under normal conditions on the upper margins of the mitral and tricuspid valves have been mistaken for vegetations.

Paralysis.—Convalescence from diphtheria is sometimes attended with the development of a series of sequelæ, which are perhaps the most remarkable that have hitherto been traced to any acute disease; I refer to *diphtheritic paralysis.* This commonly begins during the second or third week after the subsidence of the throat affection, but is said to be sometimes postponed until the lapse of a month or six weeks. First, the soft palate is affected; it hangs flaccid, the uvula cannot be drawn up; its sensibility is lost, so that neither pricking it nor applying the solid nitrate of silver causes any pain. One consequence is that the patient speaks indistinctly, or "through his nose;" another, that when he attempts to swallow any liquid, part of it passes upwards and escapes through his nostrils. The pharyngeal muscles are occasionally involved; deglutition is then difficult, and some of the food is apt to find its way into the air passages. Next, generally after an interval of a few days, one or more of the ocular muscles may be attacked; the patient then sees double and squints; or paralysis of accommodation sets in, affecting both eyes, so that he is not able to distinguish near objects properly, and cannot read small print with comfort. The limbs are commonly affected somewhat later still, but sometimes they are the parts in which a loss of power is first observed. Sensations of numbness or pain in the feet are complained of; and presently the legs grow weak and tremble, the gait is shuffling and uncertain, or the patient may be unable to stand without support. The arms are much more rarely involved, but sometimes the patient cannot dress himself or hold anything in his fingers. According to Oertel, the electric sensibility and contractility of the affected muscles are greatly impaired or entirely abolished, and their substance undergoes a very rapid wasting.* In some instances the bladder and rectum take part in the paralysis, and the genital organs, with complete loss of sexual power in adults. Again, the muscles of the larynx may be affected, in which case the vocal cords may be seen in the laryngoscopic mirror to lie motionless in a position midway between that of respiration and that of phonation,—the position which they always occupy in the dead body, but which is never seen under normal conditions during life. Wilks has insisted on this affection as a frequent cause of suffocative dyspnœa, when a tracheal tube is removed, after having been worn for a few days. The inspired current of air draws the paralysed cords inwards, until they meet and close the glottis. The muscles of the neck and of the trunk may be affected, so that the patient is unable to keep his head supported, or to raise his body from the recumbent position, or to turn over in bed. Lastly, the diaphragm and the intercostal muscles may be paralysed, in which case he is very likely to die of suffocation.

As to the cause of this sequel of diphtheria there is still much difference of opinion. In one extreme case Oertel says that he found extensive changes in the spinal cord and its membranes; hæmorrhages surrounding the roots of the nerves, proliferation of nuclei in the grey matter, and fibrinous exudation in the central canal. In another instance Buhl observed hæmorrhages in the membranes and substance of the brain, and a red, swollen, softened state of the roots of the spinal nerves. See also Dr Percy Kidd's

* Duchenne, however, denied the loss of electric contractility, and Buzzard agrees with him. The degeneration-reaction is certainly not constant. Atrophy is usually moderate, in some cases absent, but the muscles have a characteristic flabby feel. There is more or less anæsthesia, but no pain. The knee-jerk is sometimes abolished.

paper in the 'Medico-Chirurgical Transactions' for 1884. The muscles have also been found atrophied and in a state of fatty degeneration. But it is difficult to believe that similar conditions are present in those cases which terminate quickly in recovery. And the fact that the palate is so commonly first affected is obviously favourable to the theory of a "*neuritis migrans*," a morbid process starting in the nerves of the part originally attacked, and spreading along the fibres until it reaches the centres.* Such a view is quite consistent with the observation of Trousseau that paralysis may follow cutaneous diphtheria; for, in the case related by him, the limbs seem to have been affected as early as the fauces. Moreover, Senator declares that an abscess of the tonsil may give rise to paralysis; and in speaking of mumps I shall relate a case of that disease, attended with a remarkable depression of the vital functions, very like that which occurs after diphtheria. On the other hand, some writers maintain that the power of producing such nervous symptoms belongs to diphtheria only as a member of the large group of acute diseases, and that paralysis not unfrequently arises after typhoid fever, typhus, or smallpox. But I think that this must be of infinitely rare occurrence. The only case in point that has ever come under my observation is that of a boy, aged five, who was admitted into Guy's Hospital under Dr Wilks in the autumn of 1877 for a paralytic affection, which had commenced four months before, after a very mild attack of measles. He was unable to speak, and had difficulty in protruding his tongue and in swallowing. He could not stand, and his hands and forearms were rigidly flexed.

Prognosis.—The result of diphtheria is always doubtful; no case, however mild, is to be regarded as free from peril. The average mortality varies greatly in different epidemics, but according to Oertel it generally ranges between 30 and 40 per cent. The larger the proportion of young children among those who are attacked, the more fatal the disease; for in adults it comparatively seldom assumes the laryngeal form, in which from 90 to 95 per cent. die. However, according to Trousseau, the nasal variety is almost equally dangerous. It was observed by the Spanish physicians in the seventeenth century that "at its first coming it was most severe, but by degrees became less violent—as is usual with other epidemical disorders" (Fothergill).

Treatment.—The main indications in treatment are to maintain the patient's strength, and to control the various tendencies that might lead to a fatal termination. From the very first he should be strictly confined to bed. If the skin be hot, and the pulse quiet and of fair power, stimulants may for a time be withheld. But in many cases the heart soon begins to flag; brandy or port wine should then be prescribed freely, and nourishing soups (especially turtle soup) should be given in small quantities at very short intervals, night and day. Jenner mentions the case of a child, three years old, who had from three to five ounces of brandy given to him in twenty-four hours with apparent advantage. At the same time tonic medicines are usually prescribed, of which the tincture of perchloride of iron is supposed to be the best. Dr Squire speaks of administering as much as half an ounce of this preparation in the course of the day even to very

* The tenderness in the course of the affected nerves, the loss of knee-jerk, and anatomical observations by Charcot, Vulpian, and Lépine in France, and by Leyden and Mendel in Germany, have rendered probable the hypothesis of diphtheritic paralysis depending on peripheral neuritis. See Dr Buzzard's 'Harveian Lectures' for 1885, p. 108.

young patients, and still larger quantities have been ordered for adults. For some cases quinine, or bark with ammonia, is preferred. Dr Hermann Weber has drawn attention to the fact that the practice, universal in England, of giving abundance of nourishment in diphtheria, is far from having obviated the liability to the occurrence of the sudden collapse, which we have seen to be one of the principal modes in which the disease proves fatal; but whatever may be said with regard to this complication, I do not think there can be any doubt that the general tendency of such treatment must be good.

As to the *local treatment* of the throat affection, there has been uncertainty of opinion. All observers are now agreed that membranes upon the tonsils and palate should never be forcibly removed. Bretonneau recommended that the diseased surface should be brushed over every day with strong hydrochloric acid, or a mixture of one part of the acid to two or three of honey. And in 1861 Jenner expressed the opinion that this powerfully corrosive agent, if once efficiently applied, would frequently stay the spread of the inflammation; but he advised that it should not be repeated. One must be alive to the fact that mucous membranes with which the acid is brought into contact show whitish patches for at least twenty-four hours afterwards; for I have myself seen Bretonneau's practice continued day after day when no disease of the fauces was left, except that which was produced by the application. The white marks produced by nitrate of silver,—either the solid caustic or solutions in distilled water up to the strength of gr. xx to ʒj,—do not last so long. Liquor Ferri Perchl. Fortior has also been much used, and apparently with local benefit.

But of late years the practice of applying these powerful agents to the throat in diphtheria has been almost abandoned in all parts of Europe. The fact has become recognised that the local affection (or such of it as is within reach) never constitutes the really dangerous part of the disease. At the present time disinfectants are much employed,—for the purpose of destroying the micrococci, by those who hold the parasitic theory of the disease,—to lessen putrefaction, and purify the surface of the affected parts, by those who reject that theory. Diluted chlorine-water is said to be the most useful of such agents; and this accords with the results obtained experimentally by Oertel, who added various disinfectants to liquids in which diphtheritic membranes had been repeatedly washed, and afterwards tested their powers of setting up a putrefactive process in "Pasteur's fluid." The other substances which he found most effective were alcohol, solution of permanganate of potash (gr. iss—gr. iiss ad ʒj), and solution of carbolic acid (gr. iiss ad ʒj). He recommends that one or other of them should be used as a gargle, once or twice at least in every hour. Most English physicians allow patients suffering from diphtheria to suck small pieces of ice, which often give great relief.

The local application on which Dr Squire lays great stress is the weaker solution of perchloride of iron; he adds a little glycerin to it, and paints the fauces over with a camel-hair brush two or three times a day. This acts of course very differently from the undiluted Liquor Ferri Fortior. He also recommends for a gargle lime-water, which, I believe, was first suggested by Küchenmeister on account of its remarkable power of dissolving diphtheritic membranes. If the patient is very young, the fauces may be syringed out with equal parts of lime-water and milk. The editor has used neurin (the alkaloid of lecithin) locally in diphtheria of the fauces, following some trials made at Vienna: it clears away the membrane without pain or further

injury and leaves a clear surface. Another preparation introduced from the physiological laboratory is papain, from the Brazilian papaw-fruit. This powerful digestive agent dissolves false membrane with great readiness. We have used it at Guy's Hospital, to that extent with success. On theoretical grounds Oertel makes a great point of setting up a suppurative action on the surface of the affected parts as early as possible; and he therefore advises that the vapour of boiling water should be inhaled for a quarter of an hour at a time, twice every hour, and would even reduce the patient's time for sleep to three or four hours, in order to keep up this treatment fully. He also speaks highly of solutions of common salt and of chlorate of potass for inhalation; but for this special apparatus is requisite. Siegler's spray apparatus is a convenient way of applying remedies to the throat, and is now much used in cases of diphtheria, variously medicated. With very young children syringing the fauces with an antiseptic or solvent solution is probably the most efficient and least injurious procedure. Experience shows that there is no ground for the natural fear expressed by the elder Heberden that syringing the fauces and nares would poison the body by introducing putrid matter into the stomach. The danger is not in digestion but in absorption of this matter unchanged. That venerable author concludes the chapter in his 'Commentaries' which deals with the malignant sore-throat (under which he probably included some cases of scarlatinal as well as of diphtheritic angina) as follows: "the gargle may be injected with a syringe into the throats of children, but this should by no means be done so often as to tease or fatigue them. Similar reasons would forbid us still more strongly to take great pains in rubbing off the sloughs from these ulcers or in scarifying them. . . . My only reason for suspecting that I ought to lay more stress upon applications made immediately to the throat than I have here done is, that several physicians of deservedly great authority have judged them to be of more importance than they have appeared to me."*

When the disease has its seat in the *nasal cavities*, these parts should be frequently cleansed with disinfectants (dilute solutions of the permanganate of potash or of carbolic acid, lime-water or brine), which may be injected with a syringe every two hours, or even oftener. If the patient is old enough Weber's douche should be used. This consists of a flexible tube, with a nozzle fitting tightly into one nostril, through which a stream of fluid is made to run by syphon action from a vessel at a higher level. The patient is directed to incline his head forwards, and to breathe quickly with his mouth widely opened. The soft palate is thus drawn up against the wall of the pharynx, so that the fluid, instead of escaping into the fauces, passes round and runs out of the opposite nostril into a basin placed under the chin.

For diphtheria affecting the *skin*, the local application of calomel is said by Trousseau to be useful; we now use iodoform. I may take this opportunity of insisting on the rule, that, on account of the liability to the subsequent development of false membranes at spots to which blisters are applied, they should never be ordered for a patient suffering from diphtheria.

When diphtheria attacks the *larynx*, the treatment must be exactly such as would be employed in the more advanced stage of croup. An emetic of ipecacuanha or of sulphate of copper should be given, and if a good result

* Compare the excellent remarks by Senator on the useless and mischievous attempt to apply local remedies to the fauces of young children in an efficient manner.—'German Clinical Lectures,' 2nd Series, p. 447.

is obtained it may be repeated after an interval of some hours. Oertel recommends that a camel-hair pencil attached to a suitably-curved handle should be passed down into the larynx, and moved about so as to entangle and bring away any bits of false membrane that may be loose. This practice, he says, proved successful in three out of eight cases in which he employed it, the patients being respectively five, six, and eleven years old. The membranous pieces did not always come out with the instrument, they were sometimes ejected afterwards by means of a short choking cough. He also advocates the inhalation of lime-water from a spray apparatus, but (as he remarks) very little can be expected from such a procedure unless the patient spends nearly half his time at it, and most physicians will be of opinion that to advise this is to lay too heavy a tax upon his strength.

Sooner or later the question of tracheotomy has to be taken into consideration; many circumstances render it a desperate resource in diphtheria. Even if the air passages can be freed from the obstruction which immediately threatens the patient's life, there is always the fear that the general symptoms may increase in intensity and prove fatal; and, moreover, the edges of the incision are very likely to become coated with a diphtheritic layer. I may here remark that I do not take the mere fact that the wound looks unhealthy, and becomes coated with an ash-coloured layer, to be proof that it has been exposed to the action of the specific poison of diphtheria.

The indications for tracheotomy are rapid respiration, with laryngeal stridor, audible on auscultation or without putting the ear to the chest, and with deficient breath-sounds at the base of the lungs; pallor combined with a purplish tinge of the lips; and sucking in with each inspiration of the soft parts at the root of neck and at the epigastrium, as well as of the lower ribs and ensiform cartilage when they are soft and yielding in a young child. It is better not to delay the operation after vomiting either has failed to relieve or has not been excited by the means used.

After tracheotomy it is best, I believe, to abstain from all treatment except frequent food and brandy, cleansing the tube and removing membrane and muco-pus from the trachea with a soft feather. Children under a year old generally die; the small calibre of the trachea, the yielding nature of the chest walls, and the difficulty of feeding them are the causes of this high mortality. When a child refuses to swallow, liquid food must be poured down one nostril while the other is closed, not through a catheter, but by a funnel inserted into the orifice. The narrowness of the meatus makes this very difficult in the case of infants. Nutrient enemata are rarely successful for more than a very short time with children.

Mr. Cheyne has lately recommended a bolder plan of employing tracheotomy as a means of applying antiseptic solutions directly to the mucous membrane of the larynx ('Brit. Med. Journ.,' March 5th, 1887).

Special dangers.—After death from diphtheria, the lungs are found in young children *collapsed* in several lobules; in all patients the tubes are filled with pus and mucus mixed with more or less membrane, but this is often scanty or absent below the trachea and primary bronchi, and never exhibits the same continuous fibrinous casts which are sometimes thrown off from the larynx and trachea. Ammonia and senega, sweetened with treacle or syrup of tolu, is the best medicine, and brandy the best form of stimulant, with which to meet this condition.

Another danger to be guarded against is *failure of the heart.* A boy of

eight or nine under my care went well through a severe attack of diphtheria, then developed paraplegia, and died suddenly from syncope. *Post mortem* we found acute dilatation of the heart, as after some cases of scarlet fever. Steel and small doses of digitalis are probably the best means of preventing this catastrophe in addition to food and alcohol. Feebleness of the pulse and the first sound of the heart resembling the second are indications of the danger.

Thirdly, patients may die from *septicæmia*, and this must be met by local application of antiseptics, and by such remedies as quinine and alcohol.

Hæmorrhage is very rarely fatal; if so it is from the sudden opening of a branch of the external or possibly of the trunk of the internal carotid artery.

Treatment of sequelæ.—Diphtheritic paralysis is the most important of these. It generally subsides spontaneously within three or four months, and sometimes much earlier; a case under Donders in which recovery did not take place until ten months had elapsed is mentioned as rare. Some years ago, however, a boy came to me as an out-patient who had been in the hospital four years previously for this affection; he was still unable to swallow perfectly, and fluids still sometimes returned through his nose if he tried to drink rapidly. This paralysis is very rarely fatal. In 1876 a little girl died unexpectedly a day or two after her admission into Guy's Hospital for partial paraplegia of obscure origin. I only saw the child once; the house physician drew my attention to the odd way in which she would roll about in walking across from one bed to another, so that she fell unless supported by the nurse. After her death we learnt that she had recently had diphtheria, a cause for her paralysis which had not occurred to my mind. I then regretted that she had not been kept strictly at rest and in bed.

Iron is indicated in cases of this kind, and often seems to be very useful. Oertel objects to the administration of nux vomica or strychnia, and he does not recommend that galvanism or faradisation should be employed, except at a late period. Sometimes the patient is left permanently with slightly impaired power of certain muscles.

CHOLERA*

History and name—Onset and early symptoms—Stage of collapse—Stage of reaction: the urine at this stage—So-called "cholera-typhoid"—Cholerine—Complications—Post-mortem conditions—Pathology—Ætiology—Theory of contagion—The Comma Bacillus—Prognosis—Treatment.

In the winter of 1817–18 there appeared in the camp of the Marquis of Hastings, then engaged in the Mahratta war on the banks of the Sind, a very fatal malady attended with vomiting and purging. It is now believed to have prevailed in India from time to time during the previous century, and indeed as far back as history goes; but it was then taken for a new disease and created the utmost terror. During the next few years it spread over a large part of Asia, through many of the countries of Europe, and even to America. It had, of course, to be named, but the designation which was chosen for it, Cholera, was extremely inappropriate, having been used since the days of Hippocrates for diarrhœa attended with a flux of bile (χολή), whereas the Indian disease was marked by an absence of bile in the matters vomited or discharged from the bowels. For a time, therefore, there was much confusion, and the epithets "Asiatic," "epidemic," "malignant," were commonly applied to the new malady by way of distinction. But of late it has become more and more usual to speak of the former, which is certainly a specific disease, as the only *cholera*, and to classify as diarrhœa or gastro-intestinal catarrh cases which are now and then seen, and which appear to be merely exaggerated forms of these affections, although their symptoms are more or less "choleraic" in character.

Course.—After exposure to the exciting cause of the disease, there is a period of *incubation*, which is believed to be generally two or three days, but sometimes not more than twelve or twenty-four hours. Dr Goodeve, in 'Reynolds' System of Medicine,' cites an instance, recorded by Dr Barry, in which a detachment of sepoys, on their march from one place free from cholera to another, passed through a village where it was raging; one of the sepoys was attacked after forty hours, and fresh cases appeared subsequently.

The *onset* of cholera may be either gradual or sudden. When the disease sets in gradually the earliest symptom is generally diarrhœa, which is often called "premonitory," and which may be attended with griping pain and with a sense of exhaustion. In some cases malaise, depression of spirits, headache, vertigo, noises in the ears, oppression at the epigastrium, are present at this period. At the London Hospital in 1866 a nurse was doing her work as usual when she was attacked with "singing in her ears," and nausea, so that she lay down hoping that her symptoms would pass off, but they were quickly succeeded by those of cholera. Another patient there complained not only of noise in the head, but of being pained by the sound

* *Synonyms.*—Epidemic, Asiatic, malignant or blue cholera; Cholera pestifera.

of his own voice ; and another could not rest, and kept shifting his head from the top to the bottom of his bed every few minutes, trying to escape from the ever-pursuing noise. The countenance of a patient during the premonitory stage is often pallid, anxious, and sorrowful. Annesley cites a case in which the approach of cholera was suspected mainly from the aspect of the patient nine hours before the characteristic symptoms appeared. The duration of the premonitory stage varies from a few hours to two or three days. In many instances it is altogether absent, and then the disease develops itself with the most startling suddenness. It very often—according to Lebert, who writes on the disease in 'Ziemssen's Handbuch' in more than half the cases—begins in the early morning, perhaps waking the patient up from sleep.

It then sets in with violent purging, the contents of the bowels are rapidly swept out in a fluid form, and the discharges soon become almost colourless, like whey, or like water in which rice has been boiled, so that they are commonly spoken of as "rice-water evacuations." On standing this fluid deposits a loose whitish-grey material, which consists of mucous flocculi, containing numerous leucocytes, and of immense numbers of granules, including many bacteria. At one time it was thought that columnar epithelial cells were present in abundance, but this is now known not to be the case in rice-water fluid discharges during life, although that which is found in the intestine after death is full of them. The specific gravity of the liquid is from 1006 to 1013 ; it has a neutral or slightly alkaline reaction, and chiefly contains chloride of sodium, with a very small quantity of albumen. So profuse is the flow that Dr Goodeve speaks of the patient as almost filling the pan of a nightstool in two or three hours, and sometimes voiding several pints, or even quarts. He remarks that when all that is passed is collected in the same vessel, the bile contained in what was first passed may give it a yellowish colour. Sometimes the tint is pinkish from the admixture of blood. There is often no pain whatever in the bowels, but some patients complain of more or less griping. After an interval, or occasionally at the very commencement of the attack, vomiting sets in ; the fluid rejected from the stomach (unless mixed with the food) is pale and watery, being in fact identical with the rice-water liquid ; nay, it may even have a still lower specific gravity of 1002 to 1005, due probably to a large admixture of water ingested during the attack. It is often poured out of the mouth with great force and quite suddenly. Another early symptom is severe cramping pain in the muscles of the feet, in the calves of the legs, and sometimes in the thighs, hands, chest, or abdomen. This usually comes on at intervals and lasts for a few minutes at a time. It is of a most agonising character, causing the patient to shriek out and to start up from the bed on which he is lying. Lebert says that in a third of the cases observed in the epidemic at Zürich in 1855 cramps were absent.

The stage of collapse.—These symptoms are followed, more or less rapidly, by the development of a very remarkable condition, which is known as "cholera collapse." By Goodeve and others it is described under the name of the "algide stage" of the disease. It commonly appears within six or seven hours of the commencement of the purging, and often earlier still. Indeed, in some exceptional cases the patient actually dies collapsed before there has been any evacuation, and the rice-water fluid is found accumulated in the bowels after death. The essential sign of this state is a failure of the circulation, beginning at the periphery, but afterwards affecting parts

less distant from the heart. The pulse at the wrist becomes more and more feeble and thread-like, until it is altogether imperceptible; even the brachial artery may no longer be felt beating; on auscultation, according to Lebert, the second sound of the heart may be inaudible, while the first sound is still heard. The surface of the body becomes cold, livid, and shrunken. The hands feel like ice, and look shrivelled, as though they had been long soaked in water. The features have a leaden hue, the eyeballs are deeply buried in their sockets, the nose is pointed, the tongue feels cold, even the breath may be entirely devoid of warmth. The skin is often covered with a profuse sweat, which must still further deprive it of heat.

According to Goodeve a thermometer placed in the mouth indicates from 79° to 88°. In the axilla, however, the temperature is seldom below 93° or 94°, as was shown by careful observations made at the London Hospital in 1866 by Mr F. M. Mackenzie. And in the rectum, or in the vagina, the temperature rises through the period of collapse, reaching 100°, or 102°, or even 104·4°. Mr Simon, in discussing these facts in his 'Ninth Report,' infers that the choleraic affection of the bowels is a "heat-making" process. But I see no reason for doubting that the heat is generated in the muscles and in the glandular organs, just as it is under ordinary circumstances. Mr Mackenzie observed that a severe attack of cramp raised the axillary temperature as much as 2° F. The only question seems to be whether the elevated temperature in the interior of the body is due to a mere accumulation of heat from deficient loss, or whether there is a positive shifting upwards of the point to which heat regulation is set, as in true pyrexia (see p. 43). It is a strong argument against the latter view that during reaction, as we shall presently see, when the peripheral circulation becomes restored, the internal temperature falls and is actually below normal. And I think that cholera collapse must be regarded as essentially different from the initial rigors of acute specific or inflammatory diseases. One distinction, on which Hutchinson has insisted ('Lond. Hosp. Rep.,' vol. iii), and which is of much interest, is that in collapse the pupils are of natural size, whereas in rigor they are widely dilated; but I am not sure that his conclusion is necessarily justified—that there is no arterial or vaso-motor spasm in the one condition, as there doubtless is in the other.

The breathing during collapse is increased in frequency, being at the rate of 24, 30, or even 40 in the minute; and there is often a sensation of dyspnœa. The heart's action, however, is but little accelerated, remaining at about 100. There is great muscular weakness. The voice becomes feeble and whispering, or may be so completely extinguished that when an attempt is made to speak nothing but a movement of the lips follows. Lebert thinks that this is due to dryness of the vocal cords, as well as to relaxation of their muscles. As a rule, the patient soon becomes unable to rise from the recumbent position; but sometimes he can sit up, or even walk across the room, very shortly before his death. There is usually great restlessness, the limbs being abruptly tossed and jerked about.

The mental state is generally, from the first, one of indifference rather than of anxiety; and in some cases it is only when the cramps cause the sufferer to cry out that he seems to be conscious of his condition. But he can easily be roused to understand what is said to him, and may answer quite rationally. Coma sets in only towards the very last, if at all.

It is to be noted that when collapse has developed itself in a marked form, the purging ceases altogether, or becomes greatly diminished. Probably

what is now passed had been poured out into the bowel at an earlier period; Goodeve describes it as often containing gelatinous mucus-like masses. The stomach, however, still remains very irritable. There is the most intense thirst; the patient craves for water, and drinks it with eagerness, only to vomit immediately afterwards. Another symptom which may cause much distress is a sense of burning heat at the epigastrium, often accompanied by great tenderness, and sometimes by hiccup. The abdomen is commonly retracted.

Lastly, one of the most marked phenomena of collapse is suppression of urine. This seems often to be present from the earliest period of the disease; but Lebert says that patients may for a time micturate when the bowels act. There is no doubt that, as was first pointed out by Griesinger, the arrest of the renal secretion in cholera is a result of the defective flow of blood through the kidneys. Hermann and Cohnheim have each shown that precisely the same thing occurs in animals when the renal arteries are compressed or ligatured.

Fatal event.—Cholera collapse often leads directly to a fatal termination, which usually takes place between twelve and twenty-four hours after the commencement of the attack, but sometimes earlier, and sometimes during the second day. Lebert mentions that before death the eyes may become dry and the corneæ slightly opaque.

The internal temperature has often been found to rise after life has become extinct. Another phenomenon sometimes observed in the dead body is the occurrence of spasmodic twitchings and quiverings of the muscles within the first half hour after death. The limbs may actually move; in a case at the London Hospital in 1866, the elbow became raised about three inches above the level of the chest, across which it was lying.

Reaction.—Not infrequently, even when collapse has been present in an extreme form it is nevertheless recovered from. In that case *reaction* is said to occur. This usually begins to develop itself at the end of twenty-four or forty-eight hours. Goodeve remarks that the subsidence of restlessness and jactitation often constitutes a sign of very favourable import. The patient dozes quietly with easy respiration; then a flickering pulse at the wrist is detected, which gradually becomes more distinct, the superficial veins on the back of the hands begin to fill, the surface is felt to be less cold; the features look less sunken and acquire a better colour. As a rule, the improvement occurs slowly and step by step. But the older Indian physicians spoke of cases in which it was very rapid, as for example, that of a man "standing at his door on Wednesday, who on Monday was in perfect collapse."

According to Mr F. M. Mackenzie the temperature during reaction, when the pulse has fairly recovered, is usually about 97° in the rectum or the vagina, and 96° in the axilla. But in some cases at the London Hospital in 1866 it was observed that certain parts of the surface conveyed to the hand a sensation of burning pungent heat, the degree of which, however, was not determined by the thermometer. Another point noticed at the same time was that the pulse often fell to 54, or even to 45, in patients who were doing perfectly well. Lebert, on the other hand, speaks of the pulse as remaining at about 100. It often becomes full and bounding, and may be dicrotic. The patient's aspect is peculiar. The cheeks usually present sharply defined patches of dusky redness; the conjunctivæ are deeply injected, the eyes half closed with the corneæ turned upwards, the expression heavy and vacant.

In some cases slight pyrexia occurs at this stage, and it is attended with the development of a bright crimson or scarlet *rash*, which is commonly spoken of as a roseola, though it may rather have the character of urticaria. Sometimes it nearly resembles the eruption of scarlatina. Mr Mackenzie found in 1866 that it was always accompanied by a rise of temperature, both external and internal. In one instance the former was 101·6°; the latter 102·4°. The backs of the hands and the forearms are its favourite seats, but it may also cover the trunk, and even be seen on the face. In the museum of Guy's Hospital we have excellent models of it made during the epidemic of 1854. It seldom comes out until a week or ten days after the commencement of the attack, and in one case recorded by Wilks it did not appear until the seventeenth day. It lasts two or three days, and may be followed by desquamation. It is more often observed in young patients than in old, and is particularly well marked in children. As a rule the cases in which it occurs end in recovery.

The period of reaction, however, is by no means free from danger. Sometimes a relapse occurs; purging, vomiting, and exhaustion set in again, and end fatally. In other cases the reaction is said to be *imperfect*. The pulse after improving up to a certain point, remains weak, and the surface of the body continues to be colder than natural. The bowels are still relaxed and the evacuations are watery, although more or less coloured with bile. There is no appetite and the patient is depressed and drowsy. Sometimes the most prominent symptom is irritability of stomach attended with thirst and burning sensations along the œsophagus and at the epigastrium. Goodeve also mentions sleeplessness, lasting for two or three days or more, and causing great discomfort to the patient.

Urine.—But in the reaction of cholera the most important condition is that of the kidneys. We have seen that during collapse there is generally complete suppression of urine; if any is secreted it is apt to be, even at that period, albuminous. When reaction sets in, the suppression often continues for several hours, or even for two, three, or four days. According to Buhl, if it lasts up to the sixth day the case is hopeless. Sometimes, however, when the renal function has really been re-established, the patient, nevertheless, fails to void urine, which is retained in the bladder. The hypogastric region must therefore always be carefully examined, and, if necessary, a catheter must be passed. The instrument should be new and lubricated with carbolic oil, for cystitis and pyelitis have often been observed in cases fatal at an advanced stage. In a female patient who died in Guy's Hospital during the epidemic of 1866 there was suppurative nephritis, which appeared to have arisen by extension from the vesical mucous membrane.

The urine which is first secreted during the reaction stage of cholera is described by Goodeve as high coloured, acid, and possessed of a strong animal smell. As to its specific gravity there are some discrepancies in the recorded observations. At the London Hospital in 1866 it was found to range from 1005 to 1017, the average being 1006. Wyss, however ('Arch. d. Heilk.,' 1868), found that the density of the first urine passed was from 1012 to 1033, the average being 1020. It was only at a later period, when the quantity was more abundant, that it fell to 1010, or even lower. The amount of urea and of salts is much diminished, but albumen is generally present, and sometimes blood, and there are usually hyaline casts in immense numbers. Lebert says that the occurrence of albuminuria at this

stage is constant, but his statement does not accord with the experience of English observers. At the London Hospital in 1866 albumen was found "in about half the cases examined." As a rule, the urine continues to be coagulable during from two to seven days. Until recently the renal affection attended with albuminuria in cholera was commonly regarded as parenchymatous nephritis, and as comparable with the acute renal affection that follows scarlet fever or diphtheria. But Bartels, in 'Ziemssen's Handbuch,' pointed out that it may rather be regarded as the result of the ischæmia which exists during the stage of collapse, and this view has since been upheld by Cohnheim.

In cases fatal before reaction has set in, the kidneys are found small, tough, and of a dark brown or slightly livid colour. At a later stage they are much enlarged—Dr Moxon speaks of them as weighing up to fifteen or sixteen ounces—soft, greyish white or yellowish. The epithelium very early becomes cloudy, opaque, or fatty; it has lately been suggested that this change in it is analogous to the "coagulation-necrosis" which Litten has found to occur in the renal epithelium of rabbits after arrest of the arterial blood-current through the kidneys. The Malpighian tufts and their capsules are said to present no morbid appearances in cases of cholera. Sometimes infarcts are found in the kidneys, being doubtless results of the ischæmia of the stage of collapse. The renal affection of cholera appears never to form the starting-point of chronic Bright's disease. This fact accords with the modern opinion that it is theoretically to be distinguished from the various forms of nephritis; but it is to be observed that dropsy and other renal sym ptoms do not accompany the renal affection of diphtheria, the inflammatory nature of which is indisputable.

Cholera-typhoid.—The reaction stage of cholera often presents a grave complication, which is known as "cholera-typhoid." This sets in about five or six days after the commencement of the attack. It is attended with great prostration, headache, giddiness, and stupor. The face is at first flushed, but afterwards becomes pale. The temperature does not rise more than 2° or 3°. The tongue becomes dry. The pulse, which is sometimes at first very slow, becomes weak and small. There is often a remarkable rigidity of the muscles, so that the patient strongly resists any attempt to open his mouth, to separate his eyelids, or to straighten his elbow. Convulsions are seldom observed. Death by coma usually occurs in about forty-eight hours, but sometimes the typhoid state continues for several days, and sometimes ends in recovery.

Following Frerichs, most writers are of opinion that this complication of cholera is due to *uræmia.* Urea has, in fact, been found in the blood, in cases of cholera-typhoid by several observers. Buhl estimated the quantity of it in one instance at ·2 per cent. In some instances, too, there has been observed upon the face and neck an efflorescence of urea in a crystalline form, excreted by the sweat-glands. But Goodeve has pointed out that in some cases a consecutive fever, attended with typhoid symptoms, occurs independently of uræmia, or at least while urine free from albumen is being secreted. Both Bartels and E. Wagner drew attention to the same fact, and attributed the pyrexia in many cases to local inflammations of the intestines or other internal organs.

Slighter forms.—I have still to mention certain minor forms of cholera, cases of which are certainly not uncommon, but of which the frequency in relation to that of the typical disease cannot be positively stated, because

they are often allowed to run their course without being brought under medical observation. One of them is commonly called "choleraic diarrhœa." It is identical with the "premonitory diarrhœa" already described, except that it subsides after a time and does not pass on into cholera. It often begins suddenly, after a chill, or after the patient has eaten unwholesome food. There are three or four or more evacuations of yellow fæcal matter daily, with some pain, and perhaps with slight cramps. A more severe form is sometimes termed by foreign physicians "cholerine;" it is attended with vomiting, with cramps, with a transitory disappearance of bile from the stools, and even with some degree of coldness of the limbs. Lebert says that he has seen it followed by pronounced typhoid symptoms.

Complications.—The convalescence from cholera may be accompanied by certain complications, which require brief mention. One of them is an affection of the lower segment of the cornea, generally of each eye. This doubtless results from the part having been exposed and irritated during the stage of collapse. It now, four or five days after reaction, becomes hazy, or covered with a layer of opaque lymph; and ulceration presently occurs in it. Sometimes perforation takes place, but generally—if the patient should recover, which seldom happens—the eye is preserved with little or no disfigurement. In other cases, during the second or the third week the parotid glands swell or suppurate. This interferes with swallowing food, often with a fatal result. Sometimes bronchitis, pneumonia, or pleurisy sets in; or diphtheritic inflammation of the fauces, or the bladder, or of the sexual organs, or gangrene of the scrotum or penis, or even of the nose. Bedsores, too, are often a source of danger at this period of the disease.

Morbid anatomy.—This throws but little light on the pathology of cholera. The bowels commonly contain more or less of the rice-water fluid; this microscopically is found to contain an immense quantity of columnar epithelium, not only loose cells, but coherent masses of considerable size, forming complete casts of the villi. If there is no liquid, the cast-off epithelium forms a soft creamy pulp. At one time the opinion was held that shedding of the intestinal epithelium was the fundamental lesion of cholera; but it is now known to be of *post-mortem* occurrence. For, not only are the cells absent from the evacuations passed during life, but (as Cohnheim observes) denudation of the mucous membrane, if it took place before death, could not but be followed by severe inflammatory changes. All that is found is a slightly injected, swollen, and œdematous condition of the intestinal coats, with perhaps some ecchymosis of the valvulæ conniventes. The lymph-follicles in the mucous membrane are also in many cases enlarged, and Lebert says that there may be slight ulceration of Peyer's glands. In a case that occurred in Guy's Hospital in 1854 the lining of the rectum showed a patch which was œdematous and sloughing, with suppuration in the submucous tissue. The peritoneal coat may be reddened and sticky, and in 1866 Dr Moxon found in one case, in which the collapse had been unusually protracted, that a tenacious viscid material like spider's web lay between the coils of intestine, and could be drawn out into long filaments by separating one coil from another. Other points on which he insists are the want of fæcal odour and of the blackening produced by sulphuretted hydrogen, and the absence of gas in the intestine, so that the coils lie in a flabby compact mass in front of the spine. The mesenteric glands are sometimes slightly swollen.

The blood which is found in the heart and in the great vessels is remarkably viscid and tarry; and, whenever venæsection has been practised during life, the same peculiarities have been seen. This state of the blood, however, is not peculiar to cholera; for it was well marked in a patient who died in Guy's Hospital in 1871 of acute ulceration of the intestine. It is no doubt a consequence of the drain of fluid from the body. Another result which has been noted by more than one observer is the rapid disappearance of dropsical exudation in the course of a few hours when a patient suffering under dropsy happens to be attacked with cholera. In all probability the change in the blood is the cause of the formation during life of ecchymoses, which at the autopsy are often found scattered over the outer surface of the heart, upon the pulmonary pleura, in the pia mater, and elsewhere. It also renders the tissues peculiarly dry. Cohnheim remarks that the serous cavities in the dead body of a cholera patient have a soapy feel which is not likely to be forgotten by anyone who has ever placed his hand in them; and the spleen, the lungs, the liver, and the other organs are tough and leathery. The peculiarly shrunken appearance of the lungs is attributed by Dr Moxon, no doubt correctly, to the dry empty state of the bronchi, which allows the elasticity of the organs to drive the air out of them more completely than usual after the chest is opened. On the other hand, when death has occurred during reaction, the smaller tubes are often found full of pus, and parts of the lungs may be œdematous, or even in a state of broncho-pneumonia.

Pathology.—Hitherto there has been the greatest uncertainty as to the origin of the various symptoms of cholera, and as to their relations to one another. The characters of the rice-water liquid are so unlike those of any inflammatory exudation which is known to occur, in whatever part of the body, that we cannot possibly suppose it to be of such a nature. An experiment, however, originally made by Moreau has perhaps suggested the clue to its real significance. Physiologists had previously learnt from Thiry how to isolate a portion of the intestine, so as to obtain from it an unmixed succus entericus in small quantity. Now Moreau has found that when an isolated loop of intestine has all the nerves in its mesentery cut through, it becomes filled, within a few hours at latest, with an abundant secretion of a thin alkaline yellowish fluid, which is of very low specific gravity, which contains numerous mucous flocculi, but in which there is only a very small quantity of albumen, while the chief salts in it are those of soda.* There seems, in fact, to be a paralytic over-secretion of the succus entericus; and, according to Masloff, it possesses the property of converting starch into sugar. Now Kühne has shown that the rice-water liquid of cholera likewise contains a ferment having this property, besides corresponding closely with the other liquid in all other respects. The inference, therefore, seems justifiable, as Cohnheim points out, that the cholera liquid itself is nothing else than the secretion of the intestinal glands, enormously increased in amount. Whether its formation should be regarded as a sign of paralysis of intestinal nerves is left by Cohnheim an open question. In proof that the muscular coats of the bowel are not always paralysed in cholera he cites

* Moreau's results have since been confirmed and extended. See the Reports of a Committee appointed by the British Association, published in the 'Transactions' for 1874, 1875, and 1876; also papers by Dr M. Hay in the 'Journal of Anatomy and Physiology' (vol. xvi, p. 243 to vol. xvii, p. 441), and by Dr Brunton and the Editor of the present work, in the 'Practitioner' for November, 1884, *et seq.*

the fact that invaginations are often found, of the kind which are known to be of *post-mortem* origin. But he insists that the so-called "cholera sicca," in which rice-water liquid is found in the intestines after death, though none had been voided during life, owes its peculiarities to an early exhaustion of the excitability of the intestinal walls; and he refers to the same cause the frequent cessation of vomiting and purging as collapse becomes developed.

It cannot be doubted that the acceptance of this view with regard to the nature of the rice-water liquid will tend powerfully to support the doctrine, already held by many observers, that the primary action of the cholera virus is upon the solar plexus, rather than upon the bowel itself. Cohnheim, indeed, agrees with those who think that the phenomena of collapse are adequately explained by ascribing them to the drain of fluid from the body, and to the viscid inspissated condition of the blood which we have seen to result from it. This opinion was opposed several years ago by Dr George Johnson, one of whose arguments was that no relation could be traced between the degree of severity of the collapse in a given case, and the amount of fluid discharged from the stomach and from the intestine. But I think that a much stronger argument has since been advanced by Mr Sedgwick, namely, that cholera collapse closely resembles that which follows cases of perforation of the stomach, of intestinal obstruction, and of other abdominal diseases in which no purging occurs. At Guy's Hospital, during one of the London epidemics, a man actually died of internal strangulation of the bowel, who was supposed during life to be suffering from the so-called "cholera sicca." I imagine, however, that this mistake would have been avoided if the duration of the symptoms had been taken into account. It must therefore be supposed that collapse is the result of an action on the part of the great ganglia of the abdominal sympathetic. This action, however, is by no means identical with that which causes syncope. Cholera patients have no sensation of faintness; and even when they have no pulse at the wrist, they may be able to sit up, or to walk about, without sudden failure of the heart's action. On the other hand, the collapse of cholera is essentially different from rigor. May it not be that, whereas in syncope an influence from the solar plexus is thrown solely upon the heart, and in rigor solely upon the arterial system, in collapse it is exerted evenly upon them both, there being spasm of the peripheral arteries, while the force of the heart is lowered just to the point at which it can effectually propel the blood, diminished as it is in volume, through the deeper arteries? The suppression of urine which occurs in collapse is a part of the general state, since it is observed likewise in cases of acute intestinal obstruction, of perforating ulcer of the stomach, and of arsenical poisoning. Thus I think that there can be no doubt that Dr Johnson was in error when he endeavoured to trace all the phenomena of cholera collapse to obstruction of the flow of blood through the pulmonary capillaries. And although the high authority of Parkes can be cited in proof of the fact that the lungs weigh much less than usual in cases of cholera, this may be sufficiently accounted for by the diminished volume of the blood generally, since the lungs contain so little solid material that their weight, so long as they retain their spongy character, must be mainly that of the fluid in their vessels or in their interstices. During the epidemic of 1866 I, like my colleague Dr Moxon, failed altogether to observe that the branches of the pulmonary artery and the right side of the heart were distended or gorged with blood when death had

occurred even at an early stage. It is true that cholera is attended with a lividity which is wanting when collapse is due to other causes. But the altered constitution of the blood may be fairly supposed to interfere with its due aëration, and the amount of carbonic acid exhaled from the lungs has been shown to be really much reduced.

Absorption is retarded, although not entirely annulled. Magendie is said to have found that when camphor was injected into the rectum, five minutes elapsed before its odour could be detected in the breath, instead of only one minute, as in health; and Lebert states that atropine fails to dilate the pupils when administered by the mouth, though not when injected into the blood. It is, however, a very curious circumstance that in women who are suckling children the secretion of milk continues during collapse, even to an extent which is troublesome. The menstrual flow also goes on unchecked; it may actually set in, before reaction is established, if the proper time has come round for its appearance. A sanguineous muco-purulent discharge from the vagina also frequently appears, independently of the catamenia. And it must, I think, be admitted that the mental and the bodily vigour are often maintained throughout the whole of an attack of cholera in such a way as to show that the circulation still remains very active in the brain and also in the muscles. When, the disease seizes upon a pregnant woman, the fœtus seems always to die; if the period of gestation is early, abortion takes place; if late, the mother often dies undelivered. In this case it appears to be useless to perform the operation of Cæsarean section, even immediately after her death.

The facts which have been already stated as to the course of the external and the internal temperature respectively during the reactive stage, seem to show, as I have already remarked, that there is no true fever, such as might be supposed to have for its aim the destruction of specific organisms in the blood. The reaction seems to be what its name implies, and analogous to the hot swollen condition observed in a part when its vessels are allowed to fill with blood, after having been kept empty; as, for example, in experiments on the ears of rabbits.

Ætiology.—With regard to the causes of cholera much has been learnt in the course of the last half century, although many points still remain obscure.

In the first place we may take it as established that the diffusion of the disease over other parts of the world from India is the result of human intercourse.*

When in 1823 *the first epidemic* entered Russia by Astrachan, and afterwards spread in a north-westerly direction, its resemblance in this respect to Influenza led many physicians to think that, like that disease, it was caused by some mysterious atmospheric or telluric agent. But its progress was in reality far too slow and halting to render such a view probable. Prussia was reached by cholera in 1831, in October of that year it passed from Hamburg to Sunderland, and entered London in January, 1832. Shortly afterwards it invaded France *viâ* Calais, and it also spread across the Atlantic to Canada and the United States. In 1833 it appeared in Portugal, passed in an easterly direction through South Europe, and became for the time extinct in 1837. In fact one can hardly doubt that its

* See the full and interesting accounts of the spread of the several historical epidemics of India by Mr Macnamara in his 'Treatise on Asiatic Cholera,' 1870 and 1876. Also Dr Bellew's 'History of the Cholera in India from 1862 to 1881,' published in 1885.

diffusion from India to the West rather than to the East depended simply upon the circumstance that communication and intercourse from town to town and from country to country are so much more free in the one direction than in the other. The *second* epidemic, which had begun in India in 1840, extended to Europe in a manner very like the first, reaching London direct from Hamburg in 1848, and continuing to prevail in England during the following summer. The *third* affected this country in 1853–54. The *fourth*, which occurred among us in 1866, differed from the others in having entered Europe from Alexandria; it had been carried from India to Arabia and thence to Egypt, partly by coast-trading vessels, but in great part also by Mohammedan pilgrims to and from Mecca. A detailed account of this epidemic, by Mr Netten Radcliffe, appeared in a supplement to Mr Simon's 'Report to the Privy Council' for 1874. The *fifth* and last extended over the South of France, Italy, and Spain during 1884–5–6.

One of the most remarkable circumstances with regard to cholera is that although it has spread to almost every part of the world, and has sometimes prevailed under widely different thermometric and other conditions, it seems to be capable of establishing itself permanently in no country except India, and there only in a particular region. This of itself would suggest the probability that, in what may be termed the home of the disease, its mode of entrance into the human body may not be generally the same as in districts in which it is merely an occasional visitor. So that we need not perhaps be surprised that Indian physicians have been unable to trace the spread of cholera in certain ways, of which the occurrence has been clearly established in Europe. Or perhaps their inability to confirm our observations is an example of the fact that far greater obscurity often hangs over the ætiology of a disease where it commonly prevails than where it is rarely seen.

However this may be, there can be no doubt whatever about the value of the discoveries with regard to the mode of diffusion of cholera in Europe; they definitely place the disease by the side of enteric fever, as *miasmatic-contagious* in nature. Like enteric fever, it but seldom passes directly from a sick person to those who nurse or visit him. A few instances have indeed been recorded, which look as though direct infection did sometimes occur. Thus, in a paper in the 'Edinburgh Medical Journal' for 1838, the late Sir James Simpson related how certain mendicants brought cholera with them to Bathgate on May 27th, 1832, and how one of the nurses who attended upon them was attacked and died on the 30th. He also cites the case of a woman who, having caught the disease at some ironworks where it raged, and being afraid of being sent to the hospital, went on May 12th to a cottage where her mother lived, four or five miles off: on the 14th the mother fell ill, and died in a few hours. Dr John Snow relates that a man who had been working at Chelsea died at Streatham of a bowel complaint which lasted only a day or two: at that time no other deaths from cholera had been registered within two or three miles of Streatham, but the man's mother, who attended him, was taken ill on the very next day and quickly succumbed to the disease. But marked instances of this kind are very rare, and one may say that, as a rule, persons who have come very closely into contact with patients are scarcely, if at all, more likely than others to fall victims to cholera.

The explanation is believed to be that the contagion of cholera escapes from the body in the rice-water evacuations, but that it is not then in an active state, and becomes so only after an interval of from two to four or

five days ; in other words, the evacuations are supposed to be infective only at a certain stage in their decomposition, and not when they are fresh. The evidence in support of this view consists mainly of the results of experiments made by Thiersch in 1854, and repeated by Burdon Sanderson in 1866. These observers took liquids passed by patients during life, or taken from the intestines after death, and left them exposed to the air in glass vessels. Every day pieces of filter-paper were carefully soaked, one with each liquid, and dried. The paper was then given to mice, a square inch to every animal. The consequence was that a considerable number of the mice died with an extremely low temperature and with disturbance of the functions of the intestinal canal, their excrement being soft and altered in colour. Of the mice employed by Sanderson, 11 per cent. suffered when the rice-water liquid had been one day old, 36 per cent. when it had been two days old, 100 per cent. when three days old, 71 per cent. when four days old, and 40 per cent. when five days old; by the sixth day it became innocuous again. The morbid appearances found in the mice after death appeared to be consistent with the view that they were affected with cholera, and Mr Simon afterwards expressed the opinion that the value of the experiments in explanation of the facts of human infection was conclusive. No equally satisfactory results have hitherto been obtained with other kinds of animals ; but some instances have been recorded of dogs being attacked by a fatal disease resembling cholera after having devoured matters vomited by cholera patients, and in 1853 Dr Lindsay set up a similar disease in dogs by confining them in a small room, the floor of which was strewed with the excreta of such patients.

It may be thought that if a living organism, possessing specific properties, is present in however immature a form in rice-water discharges, it ought to be easily demonstrated with the aid of the microscope. But the alvine evacuations, even in health, contain various organisms in large numbers, and it is in reality by no means surprising that the search for special cholera microzymes has not yet met with success (1883). Dr William Budd, indeed, published figures of what he regarded as a cholera fungus as far back as 1849 ; and many observers have since made statements more or less similar. In the 'Journal of Microscopical Science' for 1881 Dr Cunningham states that "monads," belonging either to *Cercomonas* or to *Trichomonas*, are found in excreta of patients suffering from cholera in unusual numbers and in a peculiarly active state, but he adds that they occur to some extent under other conditions of disease, and even in health. He also finds amœbæ to be very abundant in certain cases of cholera ; bacteria, he says, make up a very large part even of the normal fæces, at least in India.

The comma bacillus.—When the epidemic of cholera which prevailed in Southern Europe in 1884–85 first made its appearance in Egypt, the German Government sent thither Dr Robert Koch, who had lately discovered the bacillus of tubercle as above narrated (p. 86). As the result of his examination of the disease at Alexandria, he believed that he had ascertained its contagium vivum to be a minute rod-shaped organism, which he called a bacillus. He was afterwards sent to Bombay, where cholera was prevalent, and there announced that the microphyte characteristic of cholera was of a curved shape, and named it from this character "the comma bacillus of cholera." Subsequent researches have only confirmed Prof. Koch in this belief, which is shared by other competent observers.

It was, however, opposed to the opinions of the majority of Indian

surgeons, and met with much adverse criticism. The late Dr Timothy Lewis, the discoverer of the *Filaria sanguinis*, stated that the so-called bacillus is frequently to be met with in the mouth of healthy persons, and it was found also in cases of diarrhœa by Prior and Finkler in Germany. The English Government sent Dr Klein and Dr Heneage Gibbs to India to investigate the subject, and their report was adverse to Dr Koch's theory. In 1885 the Medical Research Association and the Royal Society united to send Prof. Roy, of Cambridge, to investigate cholera then prevalent in Spain, and in the following year the former Association deputed Dr Sherrington (who had accompanied Dr Roy in Spain) to clear up doubtful points by researches in Italy, where cholera was still partially epidemic in the summer of 1886. Other observations had also been made by French physicians at Toulon.

As the result of these difficult and prolonged investigations it must, I think, be admitted that Koch's statements cannot be accepted in their full extent. This was the verdict given by Burdon Sanderson and also by a Commission appointed by the Crown to examine and report on the evidence.*

Referring to the criteria for determining the causal relation of a microphyte to a contagious disease stated in a note on pp. 22, 23, we ask the following questions: (1) *Is the disease itself definite and capable of accurate diagnosis?* Notwithstanding the marked and striking character of the symptoms and course of cholera given in the preceding pages, there is no doubt that slight cases which occur during an epidemic might at other times be put down as only severe diarrhœa; that the diagnosis between so-called cholerine or English sporadic cholera (both of them bad names), and true Asiatic malignant cholera, is often difficult or perhaps impossible; and that isolated cases occur at seaports which, in the judgment of cautious and experienced men, are indistinguishable from genuine cholera and yet do not spread the disease. Even in fatal cases the *post-mortem* appearances are not always uniform or decisive. Hence Dr Sherrington found that a large number of the supposed cases of cholera in Italy were really diarrhœa, or enteric fever, and Dr Roy had observed the same in Spain. Even granting, what is no doubt true, that if sufficient care be taken in selecting well-marked cases no appreciable risk of mistaken diagnosis is present in the case of men, the difficulty becomes very much greater when we seek to determine whether the clinical features or the morbid anatomy observed in mice and other animals entitle us to regard a disease induced in them to be the true cholera of man.

(2) *Is the microphyte clearly distinguishable from others?* This we may probably allow as the result of many patient and independent investigations. The organism is not properly called a bacillus, it is a vibrio, or rather a fragment of a vibrio, and it is indistinguishable in size, shape, or reaction to staining agents from Lewis's microphyte. But when cultivated its colonies show distinctive characters, so that mistakes may be avoided by adequate experience and pains.

(3) *Does the microphyte only occur in cases of cholera?* The answer to this question is involved in the preceding. Very similar or morphologically identical comma bacteria occur in the alimentary canal both in health and disease, but the genuine organism, tested by its growth and development as well as its form and size, has not yet been proved to exist except in the intestines or the dejecta of cholera patients.

(4) *Is its occurrence in cases of the disease constant?* This is not yet

* Their Report is given in full in the 'Quart Journ. of Micr. Sci.,' vol. xxvi, p. 303.

proved, but in the great majority of cases there are plenty of "comma bacilli" to be found in the intestinal contents, and occasionally their abundance approaches what Koch called a "pure cultivation" in the small intestines. But they do not occur as the spirillum of Relapsing Fever, and (as we shall afterwards see) the bacillus of Anthrax, in the blood or tissues. The commas are found in the dejecta and in the rice-water contents of the bowels, and also in the detached epithelium found after death loose in the ileum, but they have never been discovered deeper than the epithelium, or at furthest the *mucosa* (corium), of the intestinal walls.

(5) *When separated by cultivation from other organisms, does a new brood of the microphyte reproduce the original disease when introduced into the circulation of an animal?* This, the last and crucial proof of the causal relation of the plants to the disease, is wanting in the case of cholera. Koch's inoculations are far from convincing, and the attempts of others to reproduce cholera by introduction of the commas into the stomach, or the intestine, or the blood of various animals have not succeeded, even when the acid digestion of the stomach has been evaded, or where the influence of the bile has been excluded by ligature of the bile-duct. Whether man is the only animal capable of contracting cholera, whether the right animal has not been found (for the earlier experiments of Thiersch and Sanderson to produce cholera by feeding mice on rice-water dejections are inconclusive), or whether the contagium of cholera is another microphyte than the comma, these are questions not yet decided, but at present the evidence that the true contagium has been discovered is defective in more than one crucial point.

Klein observed some very minute rod-shaped bacteria to be more constant in the intestines than the comma vibrios, and thinks they may be more closely related to the disease. Emmerich also found straight bacilli in the blood of cholera cases.

It may be that vibriones or bacteria in the intestines cause diarrhœa with catarrh of the mucosa, and that in this nidus the true contagium of cholera finds its suitable conditions for development.*

For the present we must conclude that the organism producing cholera has not been discovered. Contagious and specific it is, as the facts to be presently given appear to prove; but we cannot identify its contagium vivum (if living it be) with any known organism. In this respect it is in the same condition with Typhus, Variola and Measles.

Modes of transference of the contagium.—Passing on now to consider how the contagious principle of cholera gains access to the human body, we find the best ascertained fact to be its frequent entrance by means of drinking-water. The late Dr John Snow deserves special commemoration, not only as having been the first to uphold this view with regard to the ætiology of the disease, but also as having devoted infinite labour and pains to establish it. He collected instances occurring as far back as 1849, in which local outbreaks were traced more or less conclusively to the contamination of surface-wells by sewage; one such occurred in Thomas Street, Horsleydown, another in Albion Terrace, Wandsworth, a third at Salford, a fourth at Ilford. A

* Professor Roy described, in addition to the comma-vibrios and numerous bacilli, bacteria and micrococci found in the rice-water intestinal contents, a new branching organism, not like one of the schizomycetes, but more like a fungus with delicate mycelium-like filaments and nodal thickenings ('Proc. Royal Soc.,' 1886, vol. xli, p. 173). It seems altogether improbable that this can be a pathogenic organism, and certain that it is not one of the Chytridiaceæ. Dr Klein believes that it is a fungus invading the preparations after they were mounted. See the correspondence in 'Nature,' Jan. and Feb., 1886.

dramatic incident was the following :—A gentleman who lived at Bath, was the owner of some houses at Locksbrook, near that town. Cholera, which did not prevail at Bath, appeared at Locksbrook and became very fatal. The people complained of the water of the well attached to their houses, drainage from the cesspools having entered it. The owner went to the place, said he could smell nothing wrong with the water, was asked to taste it, and drank a glass of it. This was on a Wednesday; he returned home, was taken ill with cholera, and died on the Saturday.

But it was not until 1854 that the evidence of the communication of cholera by drinking-water became irrefragable. Then occurred the celebrated ontbreak in and around Broad Street, Golden Square, which is said to have destroyed in ten days more than five hundred persons living within a radius of 250 yards. This was traced by Dr Snow to the water of a surface-well, with a pump situated at the corner between Cambridge Street and Broad Street. At least sixty-eight of the first eighty-three deaths occurred in persons who were actually known to have drunk the water in question; and it may be supposed that many took it without being aware that they did so, mixed with spirit in public-houses, or with effervescing powder under the name of sherbet. One very striking case was that of a lady at Hampstead, who was in the habit of sending every day for the Broad Street water; she was attacked by cholera and died. Her niece, too, being on a visit to her, drank some of the water, returned afterwards to her own house at Islington, and died there. On the other hand, scarcely any of the inmates of the workhouse in Poland Street, where there was a separate well, were affected; and the disease also spared the men employed at a brewery in Broad Street, close to the pump.

Observations on a still larger scale were made, in 1854, in the south of London, over a district containing a population of at least 300,000 persons, and supplied with water partly by the Lambeth Water Company, conveyed from the Thames at Ditton, partly by the Southwark and Vauxhall Company, whose intake was from the same river at Battersea. What rendered these observations peculiarly conclusive was the fact that over a large area the mains of these two companies ran side by side, each feeding some houses and not others, according to the arbitrary preference of owners or occupiers at a former time, when there had been an active competition for custom. Dr Snow went, in detail, throughout the area in question, from street to street and from house to house, and he found that, during the last four weeks of the epidemic, out of three hundred and thirty-four fatal cases of cholera, there were two hundred and eighty-six in houses supplied by the Southwark and Vauxhall Company, twenty-two among persons who obtained water directly by dipping a pail into the Thames, but only fourteen in houses receiving a supply from the Lambeth Company. It is to be noted, however, that the number of houses supplied by the Southwark and Vauxhall Company was greater than that of the houses supplied by the Lambeth Company, in the proportion of forty to twenty-six. As the epidemic continued, the difference in the cholera death-rate between the customers of the two companies became less, although it still remained very striking. Bethlehem Hospital, the Queen's Bench Prison, and other institutions on the south side of the river which had deep wells, scarcely suffered at all from the disease.

In 1865 and in 1866 further evidence was collected bearing in the same direction. In 1866 the parts of the metropolis mainly affected were the eastern districts, and Mr Netten Radcliffe, who investigated the matter for

the Privy Council, found that there was a great preponderance of cases among persons whose water, supplied by the East London Water Company, had passed through the reservoirs at Old Ford, whereas comparatively few cases occurred among those who received the water of the same Company pumped directly from the filtering beds at Lea Bridge into the mains. The difference was far from being as conspicuous as in South London during 1854, but one must bear in mind that among the poorer classes very many eat and drink and pass their days at a distance from the houses in which they are said to reside. Moreover, at an early period of the epidemic a notice was issued, warning people not to drink any water which should not previously have been boiled, and it is worthy of notice that from the very week in which this appeared the epidemic began to decline. A point of particular interest is that, assuming the reservoirs at Old Ford to have been in some way concerned in causing the disease, one can plausibly account for the entrance of the cholera poison into their water; for, although this water should have been all filtered at Lea Bridge, part of it was sometimes drawn directly from two other reservoirs which differed from the rest in being uncovered, and which communicated freely with the River Lea by soakage. But this part of the river was, in fact, a canal with locks, and received a large quantity of sewage, so that it was little better than a cesspool. Now, shortly before the epidemic in East London began, a man and his wife, living in Priory Street, Bromley, near the banks of the Lea, had died of cholera, and their evacuations had entered the river about 600 yards below one of the open reservoirs.

In the previous year, 1865, there had occurred in an Essex village, Theydon Bois, a most remarkable local outbreak of cholera, which illustrates in a striking manner how insidious its propagation may be. Between July and November the disease seems to have been several times introduced into Southampton by persons who arrived from the Mediterranean ports where it was then prevailing. Whether it was carried from this town during August or September to Weymouth or Portland or Dorchester, or whether it reached either of these places in some other way, was never ascertained. But so much is certain; that a gentleman and his wife, visiting these places (but not Southampton) in September, contracted diarrhœa at one or other of them, and this, in the case of the lady, developed into fatal cholera after her return to her home at Theydon Bois on September 25th. On the 30th, while she was still collapsed, one of her daughters was attacked and died in a few hours. the same night a serving-lad in the house was seized and barely escaped with his life. Altogether, in a fortnight, eleven persons within a narrow circle, father, mother, grandmother, two daughters, son, doctor, foot-boy, maid, labourer, and countrywoman, fell ill, and only three of them recovered. Now, the drinking-water of the house all came from a well beneath the scullery, and into that well there was habitual soakage from the water-closet.

Such cases as those of Theydon Bois are of especial importance, because they are inconsistent with a view which at one time found many supporters: that drinking impure water, instead of being the means of introducing the specific contagion of cholera into the body, acts merely as a predisposing cause of the disease, by rendering a person unduly susceptible to the action of the true exciting cause. Dr Snow long ago pointed out that some of those who in 1854 died of cholera in houses supplied by the Southwark and Vauxhall Water Company were young men and maidservants who had come from the country only a few days before.

Soil-water theory.—I must now briefly mention a view with regard to the mode of diffusion of cholera which has been put forward by certain German observers in rivalry with the "drinking-water theory." This is the so-called "soil-water" or "ground-water theory" of Pettenkofer. The observations on which it is based are such as cannot be described in detail within the limits of this work, but I believe that his fundamental proposition may be said to be that cholera never prevails epidemically where the soil is impermeable to water, or where the level of the soil-water is not liable to fluctuations. He brings forward several instances in which districts seated upon hard rock escaped, while adjacent towns built upon alluvium suffered. Some apparent exceptions to the rule he accounts for, more or less satisfactorily. Thus Gibraltar, as is well known, shows no immunity, but, in visiting it, he found that the town really lies on a slope of red earth, containing more than 200 surface wells. So, again, Malta, in which the disease has prevailed severely, is built on a solid rock; but this, a sandstone, is so soft and so permeable by water that the Government Comptroller told Professor Pettenkofer that it was like a sponge, saturated with all kinds of filth. On the other hand, Lyons has, during each of the European epidemics, remained free or nearly so from cholera, when both Paris and Marseilles were ravaged by it; in 1865 twenty thousand persons are said to have flocked thither from Marseilles for safety. But the greater part of Lyons lies on a river-alluvium, so that on Pettenkofer's theory one would have expected its inhabitants to be severely attacked. The explanation which he gives is that in this town, unlike most other towns, the level of the soil-water is diminished by that of the rivers which flow through it, the banks of the Rhone and the Saone being in fact so porous that their streams may almost be said to run in part subterraneously beneath Lyons. In other words, the supposition is that Lyons escapes cholera either because there is too much surface-water, or because its height is subject to scarcely any variation. But ingenious as these explanations are, one cannot help wondering whether it might not be possible, by similar reasoning, to explain away the very instances on which Pettenkofer himself relies.

In this country the ground-water theory of cholera has met with no acceptance, and I believe that observers have failed to find evidence of its applicability to those parts of India where the disease is endemic, and where such a doctrine appears *primâ facie* to have much to recommend it. But it is curious that in the East London epidemic of 1866 one fact was elicited which is strongly in favour of Pettenkofer's views. In a school at Limehouse there were four hundred pauper children, not one of whom was attacked with cholera or with diarrhœa. Now, the house had its sole water-supply from the Old Ford reservoirs, by which, as has been already stated, the disease was being spread, and the children at all times made free use of the water. A special investigation of the soil beneath this school brought to light the fact that it stood upon a thick layer of fine brick-earth and not of gravel, as appeared to be the case with the streets immediately adjacent.

According to Pettenkofer, the condition of soil under which cholera is most apt to prevail is that which occurs when the level of the ground-water has begun to fall after being high. This might perhaps cause noxious matters to be washed into wells which would at other times be free from them. But a still more likely cause for the development of an epidemic under such circumstances is that which has been alluded to at p. 32, namely

the desiccation of contagious microzymes in the upper layers of the soil, and their subsequent diffusion in the air.

Pettenkofer, however, considers that a complicated theory is needed to explain his facts. He supposes that when human intercourse conveys cholera from one place to another, that which is given off by a patient, or carried by an individual not himself susceptible, is, *not* the cholera-contagion, but what may be termed a "cholera-germ," which is capable of generating the contagion when it meets with a certain "material substratum." An analogy, which he suggests, will make his notion readily intelligible. It is, he says, conceivable that, if the yeast-fungus were absent from certain countries, the inhabitants might prepare beverages from grapes, or apples, or malt, and drink them without any liability to intoxication. But if someone were to come from a country where the yeast-fungus was found, and to bring its spores with him upon his clothes or in any other way, the hitherto harmless liquids might speedily produce an "epidemic of drunkenness." Yet the cause of the symptoms would not be the yeast-fungus, but the alcohol of the fermentation set up by it. So Pettenkofer imagines that the "cholera-germ," which he symbolizes as x, acting on the soil under certain conditions, which he terms y, generates a "cholera poison," which he designates z.

Nägeli, who is a staunch supporter of Pettenkofer's observations, has more recently suggested what he terms a "diblastic theory," according to which the union of x and y is supposed to take place within the human body. He imagines that the soil gives off certain microzymes, which must be present in the body of everyone who is to afford favourable conditions for the development of another set of microzymes derived from a pre-existing case of cholera. This is, in fact, to reduce the "soil"-element in the ætiology of cholera to the rank of a "predisposing" cause. Nägeli appeals to cultivation experiments as affording frequent instances in which the growth of one kind of fungus in a medium induces changes that enable it to afford pabulum to another kind of fungus. Or, as he points out, it is possible that the contagious microzymes of cholera which enter the body are commonly too few to be able to overcome the resistance of the living structures, unless the latter has first been weakened by the previous operation of soil-microzymes.

The advantage of this form of the hypothesis is that it admits of the direct contagiousness of cholera in certain cases, if the cholera-microzymes happen to be introduced in very large numbers. For it is undeniable that cholera sometimes seems to be propagated from individual to individual in succession in such a way as to render very improbable the hypothesis that any predisposing cause is essential. Take, for example, the following case recorded by Dr Simpson, of York, and cited by Dr Snow. An agricultural labourer was attacked at Monkton Moor on December 28th, 1832, at a time when the disease was not known to be prevailing within thirty miles. His wife and some other persons who visited him were seized on the following day. Presently it was found that his sister had died of cholera a fortnight previously at Leeds, and that her wearing apparel had been sent to Monkton in a box which had been opened by him the evening before he fell ill. During the illness his mother, who lived in a healthy village five miles off, came to attend him, washed his linen, and after two days set off to return home, but was attacked on the road and had to be conveyed to her cottage. Not only did she die, but her husband and her daughter likewise.

In 1866 Parkes attributed an outbreak of cholera in Southampton mainly

to an atmospheric effluvium disengaged from sewage, which was constantly being pumped up by a steam engine, and sent churning down an open channel for some eight or nine feet.

A few instances have been recorded in which the disease has apparently been conveyed by articles of food. Dr Snow mentions that several persons were attacked in the village of Carisbrook, in the Isle of Wight, after eating some stale cowheels which had belonged to a man at Newport who had died a day or two before. In future epidemics one may be sure that observers will be on the look-out to trace the spread of cholera to milk-supply.

Altitude and climate.—There are still to be mentioned certain conditions which seem undoubtedly to influence the diffusion of cholera, however indirect their operation may be. Dr Farr laid down a "law of altitude," at least for London, showing that the incidence of the disease upon the population varied inversely as the height above the River Thames. This, however, is but an expression of the fact that on an average persons living at a low level are affected by the causes of cholera in larger numbers than persons living at a high level. Sometimes the case is reversed; thus Lebert says that throughout the epidemic at Zürich in 1855 the upper parts of the town suffered more than the rest. The circumstance that in Europe cholera has never been known to prevail at an elevation of more than 600 or 800 mètres above the sea may be set down to the absence of the conditions required for its development; in other quarters of the world it has raged at altitudes of 2000 to 2500 mètres. In temperate climates the summer and the autumn are the seasons most favourable to it. It often dies out as winter approaches, to reappear in the following year. But in Russia, in 1830, it withstood extreme degrees of cold.* Cholera appears never to have invaded Australia, the Cape Colony, or the South Seas.

Diagnosis.—To recognise cholera in its well-marked forms is seldom difficult. Almost the only morbid state which can be mistaken for it is that produced by the poisonous administration of arsenic. Lebert relates how Louis actually committed this blunder in regard to the Duke of Choiseul, who killed himself with arsenic the day after his arrest for the murder of his duchess. At that time, in the summer of 1847, the disease was not prevailing in Paris. There is therefore good reason for bearing in mind the possibility that a crafty person might take advantage of an epidemic to give poison with but little risk of being suspected.

To distinguish from an ordinary diarrhœa the slighter effects of the virus of cholera is often quite impossible. Under the name *cholera nostras* or *cholera Europæa* writers describe an affection of which the symptoms are identical with those of the specific disease (called by them *cholera Asiatica*), but which differs from it in being scarcely ever fatal and in generally occurring sporadically. A typical instance occurred at Guy's Hospital in 1865, and may be found recorded by Wilks in the 'Medical Times and Gazette' for that year. A blacksmith's hammerer, aged thirty-three, was at work as usual at 7 a.m. on May 25th, when he was suddenly seized with profuse vomiting and purging. He was admitted collapsed, with cold breath; he had cramps in the flanks and in the legs; the evacuations and the matters vomited

* The remarkable distribution of cholera in its visitation of England 1848-49, its spread, ingravescence, and decline in the several towns it attacked, are described, and their bearing on its ætiology as then appreciated impartially discussed by the late Dr Baly and Dr (now Sir William) Gull in their 'Reports on Epidemic Cholera' to the Royal College of Physicians, 1854.

had the appearance of rice-water, consisting of an alkaline liquid, with flocculi floating in it. He recovered in about twenty-four hours. Dr Wilks is reported to have said that every year he saw one or two such cases, but seldom so early in the summer. It is to be observed, however, that at the very time when this man was attacked, an epidemic of cholera was approaching England. And utterly improbable as it may seem that an isolated case should spring up in London several weeks before the commencement of the local epidemics at Southampton and at Theydon Bois in the autumn of the same year, I do not think that the possibility of such an occurrence can be denied. At any rate, so far as I am aware, no case of so-called "English cholera" (attended with collapse, rice-water stools, and cramps) has been admitted into Guy's Hospital since 1870; and it is certain that when the first epidemic arose in 1831, Sir Thomas Watson and the elder Dr Babington and many other physicians of experience declared that they had never "met with the same complaint before." I am therefore disposed to doubt whether it is necessary to call any disease cholera except that which I am now describing; and I am sure that on every ground it is very undesirable to designate attacks of diarrhœa, in infants or even in adults, "choleraic," whenever they happen to be attended with symptoms approaching those of collapse.

The *prognosis* is less favourable in children and in old people suffering from cholera than in adults or in middle-aged persons. The mortality, which over a whole population generally averages about 50 per cent. or a little higher, rises among the former classes to 70 or 80 per cent., while among the latter it may fall to about 40 per cent. The disease is more than usually fatal when it affects drunkards or those who are already sick or weakly. In an individual case the degree of collapse is of much greater prognostic importance than the amount of purging. As in other epidemic diseases, the earlier cases are far more often fatal than those which occur when the pestilence is subsiding.

The *protection* afforded by an attack of cholera against a subsequent one appears to be of the slightest. Cases are not infrequent of persons who have suffered in two epidemics, and some have died from the second attack. Moreover, in those parts of the East Indies where cholera is endemic, one illness not only affords no immunity from a second, but seems scarcely to mitigate its severity. All that can be said is that cholera does not predispose to fresh infection, as erysipelas undoubtedly does.

Treatment.—The *preventive measures* which should be adopted when a locality is threatened or actually attacked with an epidemic of cholera were carefully elaborated by Mr Simon and his colleagues in 1866, and will be found detailed in his ninth 'Report.' They are generally such as might be anticipated from what has been already stated with regard to the ætiology of the disease. Mr Simon also attached great importance to the avoidance of all such food as is apt to set up diarrhœa,—half-fermented beer or wine, meat or game no longer fresh or not completely cooked, stale fish or shell-fish, vegetables or fruit long gathered or badly kept, and the like. He deemed it unwise to take purgative medicines, except of the mildest kind. And in accordance with almost every other observer, he insisted on the necessity at such seasons of checking looseness of the bowels, however painless and trivial. Dr Bristowe, I believe, stands alone in maintaining that the diarrhœa which so commonly prevails when cholera is epidemic is neither more nor less likely to pass on into that disease whether it is left alone or encouraged by purgatives; nor will he allow that, if it is really premonitory of cholera, it can be

arrested by any medicine whatever. Most writers recommend that it should be treated with opium in considerable doses, and with astringents such as acetate of lead, nitrate of silver, catechu, or chalk. I think, however, that it is doubtful whether the physiological action of these drugs upon the hepatic and the other secretions may not be injurious. In India Dr Goodeve says that it is usual to give from one to five grains of calomel with the first two-grain doses of opium; in my limited experience I have found a simple alkali with a little ammonia and peppermint effectual in checking much of the diarrhœa occurring during a cholera outbreak; but whether this treatment would suffice for an attack really premonitory, I would not venture to say. It is of course admitted on all hands that some cases come under medical observation at a very early period, and yet run on into cholera in spite of everything that is done to control them.

Dr George Johnson's theory that the purging of cholera is an effort of nature to get rid of the *materies morbi*, and should therefore not be checked but even encouraged by the early administration of unirritating laxatives like castor-oil, does not appear to be accepted by any other physician.

When *collapse* has actually developed itself, the administration of opium or astringents is believed to be useless, or even worse than useless. The state of the circulation is now such that absorption is nearly arrested; and drugs introduced into the stomach in successive doses may, if not vomited, accumulate there so as to produce ill effects at a later stage, should reaction happily occur. For the same reason, alcohol must be used with great caution, if at all. Dr Goodeve says that its admissibility depends upon its effect on the pulse. If a dose of weak brandy-and-water causes the pulse to revive ever so little, there is no harm in continuing to give the stimulant in small quantity. In any case ether or ammonia may be used, properly diluted, unless sickness is produced. But Dr Goodeve finishes his remarks upon this point by saying that many cases in which there is extreme collapse come round by themselves, as well as, if not better than, with stimulants. Ice-cold water may be freely allowed to patients with cholera, not indeed in such large quantities as their morbid thirst might lead them to swallow, but by tablespoonfuls or wineglassfuls at a time. Lebert would also give effervescing draughts made with carbonate of soda and with lemon juice; and at the London Hospital in 1866 a "saline lemonade" was employed with apparent advantage.

There seems to be no objection to administering a dose of morphia subcutaneously if the cramps are very severe. Or an occasional whiff of chloroform may be given by inhalation. It may also give relief to rub pure chloroform or turpentine into the painful parts. The burning sensation at the epigastrium may be alleviated by the local application of cold, or of a mustard plaster.

The limbs should be kept wrapped in warm flannels, and hot bottles should be placed in the bed, but not in contact with the patient's skin. At the London Hospital, in 1866, baths at a temperature of from 98° to 104° were employed in about one hundred and thirty of the worst cases, and with most marked effects. For a few seconds after immersion there was commonly difficulty of breathing, and sometimes an unpleasant sensation of heat. But in less than a minute a favourable action generally became manifest; the pulse returned, or (if it had been perceptible before), became fuller and quicker; the patient grew less distressed, ceased to moan or scream with pain, and sometimes fell into a quiet slumber. In many

cases recovery appeared to be the direct consequence of the bath; but in many more the symptoms returned unaltered.

The experience at the London Hospital seems also to have been not unfavourable to the practice of injecting a warm saline solution into a vein, when patients are in an apparently hopeless condition from cholera collapse; among fifteen cases so treated by Mr Little there were four recoveries. Most writers, on the other hand, speak of this procedure as incapable of producing any permanent good result, and therefore as deserving to be abandoned. The immediate effect of an injection is admitted on all hands to be often most wonderful; a person speechless, and almost dead, may regain consciousness, sit up, talk, and even joke; and the pulse may become distinct and full. But in most cases the improvement has been only temporary; the symptoms soon return and end fatally even though the operation should be repeated. Still, as Sir Thomas Watson observes, even such a transitory amendment may sometimes be of great importance, enabling, for example, a will to be executed. The fluid used by Mr Little contained sixty grains of chloride of sodium, six grains of chloride of potassium, three grains of phosphate of soda, twenty grains of carbonate of soda, and two drachms of pure alcohol, to twenty ounces of distilled water. Eight ounces were introduced at a time, the temperature being about 110°, and from twenty to thirty minutes were occupied in the operation. There were three cases in which the injection was repeated; one of them ended successfully.

When *reaction* begins, the management of a case of cholera continues to require great care and caution. A little beef-tea or chicken broth may now be given in small quantities at intervals, or some light farinaceous food, such as gruel or arrowroot. Should vomiting continue, it may often be checked by a dose of opium, or by a blister applied to the epigastrium. But sometimes it is necessary, for a time, to have recourse to nutrient enemata. To make up for the loss of salts from the blood, carbonate of soda and chloride of sodium should be given in moderate quantities. If the tongue becomes dry and brown, a powder containing two grains of hydrargyrum cum cretâ, half a grain of ipecacuanha, and two and a half grains of Dover's powder is said to be very useful, according to the experience at the London Hospital. When suppression of urine continues during reaction, the patient should be encouraged to drink freely; he may have a drachm of liquor ammoniæ acetatis, with five or ten drops of tincture of digitalis, every two or three hours; and mustard plasters or cupping glasses may be applied to the loins.

THE PLAGUE*

History—Incubation, onset, and symptoms—Diagnosis—Anatomy—Fatality—Treatment—Conditions of contagion and causes of the decline of the plague.

THROUGHOUT the Middle Ages, the English word Plague was applied indiscriminately to every fatal epidemic disease; and when the malady now to be described appeared in the fourteenth century, it was called the "Black Death." But for the last 200 years the only epidemics comparable with those of former times have been distinguished as epidemics of Fever. Consequently we can now speak of "the Plague" without ambiguity, although some writers still prefer the term "Oriental Plague," or "Levantine Plague."

Epidemics of the Plague were frequent during the Middle Ages, when it was probably almost constantly endemic in Persia, Syria, and Egypt. The Black Death of 1348–49 began in China, devastated the whole Continent, and put an end to the war between England and France. Its ravages in Florence were recorded by Bocaccio. It probably reduced the population of England to two thirds, or even less, of its number, and led to important economic results. There were several epidemics in the sixteenth century, and again at the accession of James I, and in the first year of Charles I; but the most destructive and happily the last in England was the Great Plague of London in 1665, so graphically described by Defoe.

There was a terrible epidemic in the south of France known as the Plague of Marseilles in 1720, when the scenes described by the great English novelist were renewed. The disease did not visit Western Europe again, but epidemics occurred during the eighteenth century in Poland, Sicily, Wallachia, and Russia (1771).

Its prevalence has for many years been gradually declining. In the early part of this century there were outbreaks of it in Malta, at Noja in South Italy, upon the Lower Danube, and in the Balkan Peninsula. Up to 1844 they were of frequent occurrence in Egypt, but that country has since been free from them. The disease also vanished from Syria, and hopes were entertained that it had become entirely extinct. But in 1858 it appeared again in Tripoli, on the south shore of the Mediterranean; and it is now known to have visited the highlands of Western Arabia in 1853. There is also reason to believe that it had never been absent for many years together from parts of India; in 1834 an epidemic which occurred in Rajpootana became known as the "Pali plague," and as recently as 1877 it prevailed at Kumaon, on the southern slopes of the Himalayas. Between 1863 and 1876 there were six or seven outbreaks of it in Western Asia; some in Persian Kurdistan, others upon the Lower Euphrates. In 1877 it showed itself at Resht, near the south-west corner of the Caspian Sea. Many observers believe that from this place it made its way to the Lower Volga, where it prevailed, in the district between Astrakhan and Tsaritzin, from October, 1878, to February or March, 1879, and excited great alarm throughout Russia, and in Europe generally.†

* *Synonyms.*—Pestis vel Pestilentia, λοιμός, Typhus bubonicus, Black death.

† The English Government sent Surgeon-Major Colville and Dr J. F. Payne to investigate the disease on the spot, but it had already disappeared on their arrival at Astrachan.

Course.—I need not give a detailed account of the general symptoms of the plague, which resemble those of any other severe febrile disease. Its *incubation* is believed to be from two to seven days. This is followed by great malaise and depression, so that the patient sometimes totters like a drunken man. Intense pyrexia then sets in, which may be accompanied by violent delirium, and may rapidly assume a typhoid form. Death not uncommonly occurs at a very early period, before there have been any symptoms characteristic of the malady; it may be preceded by the development of petechiæ and large vibices in and beneath the skin; there may also be hæmaturia, hæmoptysis, and vomit blackened by altered blood. But if life is prolonged to the second or the third day, there commonly appear one or more *buboes*, attended with severe pain and tenderness. The seat of this affection may either be the groin (in which case glands belonging to the vertical rather than to the horizontal chain are affected), the armpit, or the neck. According to Liebermeister (in 'Ziemssen's Handbuch') a plague-bubo is sometimes so small as to be detected only on careful examination, but it may attain the size of a hen's egg, or more. Its formation is commonly attended with the subsidence of the delirium and of the fever, the skin becoming covered with a profuse sweat, and the pulse falling to 90 or 100. It may ultimately subside without discharging; but usually it breaks, or is opened by the surgeon, and gives exit to a mixture of blood and pus, and this may be followed by a tedious process of suppuration, which greatly prolongs the patient's illness. Another affection which may be associated with the buboes, but which is less often seen, is of a carbuncular character. It appears most often upon one of the lower limbs, upon the buttocks, or upon the neck; sometimes there are not less than a dozen carbuncles in the same case. Bullæ and pustules may also appear upon the surface of the skin.

Diagnosis.—It will readily be understood that to distinguish the plague from other malignant fevers is not always easy, unless the character of a prevailing epidemic has already been determined. Dr Milroy, in 'Reynolds' System,' cites the remark of Heberden that "on first breaking out, the disease has never been known to be the plague;" and he says that in Constantinople or in Cairo, no physician ever ventures to give a name to an epidemic until a case occurs in which a bubo or a carbuncle is seen. Moreover, as he goes on to point out, similar local affections are sometimes, though very rarely, present in other forms of pernicious fever, whether malarial or contagious. But it is certain that the diagnosis would not have been left open so long as it often has been, were it not for that reluctance to admit unpleasant truths which has always contributed so much to the spread of every infectious disease. According to Dr Milroy, a special difficulty has often arisen, during the prevalence of the plague, from the occurrence of glandular pains and swellings, or of carbuncles, in persons who remain well enough to follow their occupations, and who speedily get well under any simple treatment. He himself expresses no opinion as to whether such cases should be regarded as examples of the plague, and submitted to an enforced segregation. On the other hand, Liebermeister speaks of instances, especially when an epidemic is declining, in which the general symptoms are mild, and which run a favourable course without any local lesion appearing, so that they may be termed abortive.

Morbid anatomy.—Post-mortem examinations throw little or no light upon the plague. The viscera are soft and blood stained, and the spleen is more or less enlarged. The serous membranes are ecchymosed. Some of

the internal lymph-glands are enlarged; perhaps a chain of swollen glands extending up from the pelvis along the front of the spine, or a mass lying in the mediastinum; their tissue is of a bright red colour, or full of points of suppuration; and blood may be diffused and extravasated into the surrounding structures.*

Prognosis.—The fatality of the plague exceeds that of all other diseases. It usually destroys from 70 to 90 per cent. of those whom it attacks; very seldom less than 60 per cent. Moreover, it often carries off half the population of a town, or of a district, in which it prevails, and it may completely root out whole families, so that no survivor remains. The "Riley Graves," near Eyam, in Derbyshire, still tell of the tragedy that befell two families in that place during the epidemic of 1666; one, that of the Talbots, consisting of seven persons, was utterly eradicated within twenty-five days; the other, that of the Hancocks, lost seven out of eight members, the only one left being the mother. It is, however, probable that no small part of the excessive mortality from this disease was due to the fact that instead of isolating each case at the earliest possible moment, the ancient practice was to close up every infected house, preventing both ingress and egress, and confining the sick and the healthy together. Those who were attacked were often left without any nursing, to take their chance of death or recovery.

Treatment.—There can be no doubt that it is of the first importance to maintain the patient's strength with plenty of food. He should also be given alcoholic stimulants, ammonia, ether, and the like. Liebermeister suggests that quinine in large doses, as an antipyretic, should have a good trial. He also recommends the use of the cold bath, and says that cold affusion has already been found useful. The buboes should be opened early.

The plague protects against itself; second attacks are comparatively rare, and when they occur are generally of diminished severity.

Contagion and prophylaxis.—That the plague is propagated by an infective principle derived from the bodies of the sick is probably now doubted by no one, although in former times there were "non-contagionists" with regard to this, as to every other disease. Liebermeister, indeed, advocates the opinion that its mode of diffusion is "indirect," and that it is *miasmatic-contagious*, rather than simply *contagious;* but I fail to discover any sufficient basis for this suggestion. The statement commonly made that it is inoculable might be supposed to settle the question; but according to Liebermeister the results of the experiments which have been made in this direction are in reality far from conclusive.

The virus seems very often to be conveyed by articles of clothing, wool, silk, hair, paper, books, &c. In 1665 the local epidemic at Eyam, in Derbyshire, so well known by the heroism of the vicar, Mr Mompesson, began with the case of a tailor who had just received a box of clothes from London (150 miles off), and who was watching them hanging before the fire when he was taken ill; at that very time the disease in the metropolis was at its worst. The bedding and the linen of those who have had the plague are particularly apt to be carriers of infection to other persons, sometimes after a considerable interval of time. When epidemics used to occur in Western Europe, the notion was that it reached Holland direct from Turkey in bales of cotton or silk, and that it was brought over to England from Holland in a similar way.

* The first account of an autopsy is given in a curious work by Dr Geo. Thomson called 'Loimotomia or the Pest anatomised,' published in 1666.

Many collateral circumstances, however, greatly influence the spread of the disease. It is said to have been especially apt to prevail where the soil is alluvial and marshy, as for example along parts of the Mediterranean coast, and in the basins of great rivers. Warm and damp weather are favourable to it; thus in European Turkey it occurred chiefly in the spring and in the early summer, whereas in Egypt the winter was the chief season for it. In Nubia and in other countries with a hot dry climate it has hitherto been unknown. Overcrowding, dirt, and poverty seem greatly to encourage its development. It affects especially the poor; and at Malta in 1813, it was noticed, according to Dr Milroy, to be far less common among the inhabitants of the upper storeys of the lofty and more airy houses of Valetta than among those who lived in the basements. The higher parts of a district sometimes escape when the lower are infected with the plague; thus it is said that the citadel of Cairo and the village of Alem Dag, near Constantinople, used to remain free, although communications with the cities below were not interrupted. Whatever weakens the health, including the dread of the disease, is believed to favour its invasion. Lastly, it seems less apt to occur in persons over fifty years old than in those who are younger.

It is greatly to be hoped that the improvement in the hygienic condition of the inhabitants of Western Europe, which has been going on during the last two centuries, has been the chief reason why the plague no longer appears among us, and that Hirsch is wrong in attributing this result mainly to the system of quarantine. For it is certain that at the present day, if the disease were again to travel towards our shores, there would be extreme difficulty in maintaining that system effectually. Liebermeister cites in detail the proceedings that were adopted to prevent the extension of the plague from Noja in Italy in 1815. The town was surrounded by two deep ditches, a triple military cordon was drawn round it, the soldiers had orders to shoot down anyone who attempted to pass, and no articles were allowed to be sent out except letters which had been first dipped in vinegar. The advantage to be derived from such measures is well illustrated by instances in which large bodies of persons isolated in the middle of an infected city have escaped. Thus Dr Aitken says that during an epidemic at Marseilles a large nunnery was "shut up," with the result that all the inmates remained free, although there was an infirmary on one side for those ill of the disease, and a burying-ground on the other side for those who died of it. This writer, however, speaks of quarantine as a barbarism and as an unwarrantable nuisance; and it is well known that many of the best English authorities hold similar opinions. Indeed, the chief reason why our Government from time to time enforces quarantine regulations in our possessions in the East and elsewhere, is that if we did not do so, other countries would at once refuse intercourse with our more exposed ports, on the mere chance of our becoming a source of danger to them.

SYPHILIS*

History of the disease—Nomenclature—Its place among specific exanthems—Origin and incubation—Primary lesion—Infecting and soft sores—Premonitory stage—Secondary lesions of skin, throat and mouth, eyes, &c.—Tertiary lesions of skin, tongue, palate, bones and testes—Diagnosis—Infection—Special questions in syphilitic pathology—Prognosis—Treatment.

CONGENITAL SYPHILIS—*Its transmission to the child—Syphilitic placenta—Local manifestations—Its further transmission—Its treatment—Later effects.*

CONSIDERING how modern is the distinction between measles and smallpox, —diseases unlike one another, and each remarkably definite in its character, —one can hardly be surprised that neither the medical writings of antiquity, nor even those of the Middle Ages, contain descriptions of syphilis as an affection *sui generis*. What first drew attention to this disease was its epidemic prevalence in Italy at the end of the fifteenth century, when Charles VIII of France invaded and occupied Naples. The general supposition then was that it was a new malady; but some years later the suggestion was made that it had been brought from the West Indies by the sailors of Columbus, who arrived in Europe about the time when it broke out. However, there are grounds for the belief that the disease had been observed in France, in Germany, and in Italy, as well as in Spain, before the expedition of Charles VIII (1494–95), and when Columbus had not yet reached Palos (March, 1493), or at least had only recently landed his men there on his return from his first voyage to the West Indies. And critical inquiries seem to show that in all probability syphilitic affections were by no means unknown during previous centuries, although they were confounded with other maladies, and particularly with leprosy.

The Italian epidemic of 1494 and the subsequent years was extraordinarily severe. Lancereaux says that almost a twentieth part of the population were attacked, and that although few died, fewer still were entirely cured. But the disease soon lost its pestilential character, and by the middle of the sixteenth century its type resembled that with which we are now familiar. A point worthy of notice is that its diffusion was at first ascribed to climatic influences, in ignorance of its real mode of origin and spread. Smaller epidemics of syphilis have since been observed, which have sometimes been entirely misunderstood, and described under a variety of names; and some affections, long regarded as peculiar to certain regions, have only recently been recognised as modifications of syphilis. Under the former head I may mention a disease which attacked one hundred and eighty persons at Brünn, in Moravia, in 1578, one which raged in Canada in 1780, and the "Scherlievo" of Fiume at the beginning of this century; under the latter the "Sibbens," which prevailed in the west of Scotland during the eighteenth century, and the "Radesyge" of Sweden and Norway.

* *Synonyms.*—Lues venerea—Morbus gallicus—The pocks or great pox.—*Fr.* La grosse vérole, *Germ.* Lustseuche.

At the present time the disease is found in almost every part of the world, but with different degrees of frequency, according as the conditions are favourable or otherwise for its usual mode of propagation. But among the inhabitants of Iceland it is said never to have established itself, although it has repeatedly been introduced by sailors. And, according to Dr Livingstone, in the natives of pure blood in the centre of Southern Africa it does not persist in any form, but gets well spontaneously. The severity of syphilis in different countries is very variable. In China and in Japan it seems to be generally mild, and Lancereaux cites French physicians, who had studied it in these countries, and have suggested that its virulence has gradually become attenuated by its wide diffusion through the closely packed population in successive generations. Upon the same authority it is stated to acquire a disproportionate degree of intensity when it passes from a Chinese to a European. Similar assertions have been made with regard to its transmission from one race to another elsewhere, in the case, for instance, of the British army in Portugal. But its worst forms always appear in seaport towns, where vice and intemperance prevail together; and it is also aggravated by unfavourable hygienic conditions of all kinds.

The term "Syphilis" seems to have been invented by Fracastorius, a learned physician of Verona, who in 1521 published a poem under that title in which he related how *Syphilus*, a shepherd, was stricken by Apollo with the new disease, which was even then not recognised as venereal. The French from a very early period contrasted *la petite* with *la grosse vérole*, just as in England "smallpox," which we now regard as a single word, formerly conveyed a similar distinction. Many other names, employed in various countries, indicated a belief that it had been introduced by communication with foreigners. Thus, while it was *mal de Naples* to the French, it was to the Italians *mal francese**; and, unhappily, the Sandwich Islanders originally knew it as the "English disease." At one time the most common designation for it was *lues venerea*, which dates back to Fernelius (1556). But it is to be observed that the "venereal disease" was held to include both gonorrhœa, proved to be distinct by Ricord in 1831, and the soft chancre, which nearly all pathologists now regard as an independent affection. Indeed, the conception of syphilis as a general malady, comparable with the exanthemata, had no existence until about forty years ago. It is true that John Hunter described a constitutional form of the venereal disease, but he expressly taught that the action of the poison on the blood was different from that which occurred in any kind of fever. Since the modern view with regard to it has gained general acceptance the name of Syphilis has superseded all others. That this view should be firmly held is, indeed, of the first importance. One may almost say that the physician ought, as far as possible, to thrust out of his sight the idea that the disease carries with it the stigma of impurity derived from illicit intercourse. There are many ways in which a person may fall a victim to syphilis without any breach of morality. And in exceptional instances one must be prepared to recognise its manifold varieties in patients of either sex, at any age, and in every position of life.

In this second edition I have accordingly included Syphilis among the

* It was called the French disease also in England.

"News have I, that my Nell is dead i' the spital
Of malady of France."

K. Henry V, v, 1.

The passage in 'Timon of Athens' (iv, 3) shows how familiar the symptoms of secondary syphilis had become by the beginning of the seventeenth century.

specific febrile diseases. It arises by contagion alone, and breeds true; inoculation is followed by a latent period of incubation; the onset is febrile, with an exanthem and a local lesion (angina); lastly, it has definite sequelæ, and protects against a second attack.

The contagium has not yet been certainly identified. Lustgarten has discovered that a bacillus, resembling those of tubercle, lupus and leprosy, occurs in the cells of the diseased tissue.*

The chief peculiarities of Syphilis are the slightness of the pyrexia, the slow evolution of its stages, the importance and frequency of its sequelæ, its hereditary transmission, and, we may add, its reaction to remedies. As Mr Hutchinson well argues, smallpox and even typhus are occasionally transmitted from mother to fœtus, but circumstances rarely concur to make this possible in acute specific diseases.

Course of the disease.—Incubation.—When a person is affected with the syphilitic virus there elapses, in uncomplicated cases, a considerable interval of time before any change is observed. The disease is like the acute contagious fevers in having a period of incubation. It is very remarkable that this fact was unknown until, between 1856 and 1862, certain experimenters inoculated syphilis upon healthy individuals. Prof. Bäumler, in 'Ziemssen's Handbuch,' brings together thirty-one observations of this kind, in the very large majority of which the incubation was from fifteen to twenty-five days. Once it was only ten days: four times between thirty-five and forty-four days. In 1865 Fournier recorded a series of cases in which the affection had developed itself naturally. The incubation was more often over than under three weeks: it not infrequently reached a month or six weeks, and once was prolonged to ten weeks. In one patient of Bäumler's, in whom the exact date of exposure to the poison was known, the incubation was twenty-five days: in another twenty-nine days. Frequently incubation is prolonged to six weeks or more. The idea that no such period occurred was based partly upon a natural tendency to ascribe the disease to the last impure intercourse preceding its appearance, partly upon the circumstance that another virus (that of the soft chancre), which produces an effect almost at once, is often transmitted in association with that of syphilis proper.

Primary stage.—The earliest symptom of syphilis is manifested at the seat of infection; it is spoken of as the *primary* lesion, while the more remote symptoms which appear elsewhere are termed *secondary*. It is often called a hard or Hunterian "chancre," but upon the skin its typical character is that of a flat red papule. This, which is at first very small, soon increases in size, and as it grows larger becomes indurated, so that to the touch it feels like a little piece of cartilage let into the part. After a week or ten days it may desquamate slightly; or a little moisture may ooze from its surface, and presently dry up into a thin scab; or it may continue to look shining and glazed; or, lastly, it may become excoriated, and slightly depressed in its centre. Upon a mucous membrane, the primary affection seems to begin as a very small itching vesicle with a reddened base, which soon breaks, forming an erosion, and afterwards a shallow ulcer; this, too, acquires an indurated floor as it enlarges.

The histology of these lesions has been investigated by several observers, but especially by Biesiadecki. A very abundant infiltration of nucleated

* 'Med. Jahrb. der k. k. Ges. der Aerzte,' 1885. See also 'Brit. Med. Journ.,' Oct. 17th of the same year, p. 757.

cells is found, not only between the connective-tissue bundles of the skin or mucous membrane, but also in the adventitia of the blood-vessels, which are narrowed by it. Biesiadecki states that there is an actual development of connective-tissue fibres towards the periphery of the indurated mass, and that this is the cause of its hardness, which by Auspitz had been attributed to the presence of amorphous exudation between the cells and the spaces in which they lie.

The course of the primary affection of syphilis varies in different cases. Sometimes it quickly passes away, leaving no trace of its presence, but when it is of large size it generally takes many months in subsiding, and upon the skin its site remains marked by a brown pigmented patch, with more or less superficial scarring in its centre. On the other hand, mucous membranes never show pigmentation, and all that is left by even the largest papule is a little vascularity and looseness of texture. Mr Hutchinson has drawn attention to the fact that in some individuals an induration, like cartilage, appears again and again during a period of several years exactly where a former primary syphilitic lesion was situated, without any fresh infection. Ultimately even the pigment disappears, and it may then be quite impossible to discover that the patient had ever suffered from primary syphilis.

Hard and soft sores.—Hitherto I have avoided speaking of the typical primary syphilitic lesion as a chancre, for I wish to bring prominently forward the view that if (as is often the case) it has the characters of a chancre, they are accidentally present rather than essential to it. At least this seems to me to be the simplest way of stating the results of investigations which have within the last few years been made with regard to the mutual relations of certain different kinds of venereal sores. Bäumler calls the affection which I have been describing, not a chancre, but an "ulcerating sclerosis."

In 1852 Bassereau took the pains to trace to their origin in the opposite sex a number of venereal cases; and this method of "confrontation" (as he termed it) showed that, whereas sores which were followed by secondary symptoms had been derived from persons who themselves suffered under similar effects, other sores which remained simply local, or which at most were attended with suppurating buboes, came from individuals in whom the disease had likewise failed to produce any constitutional effects. His views were soon afterwards adopted by Ricord, who indicated a number of distinctions between the two kinds of lesions in question, of which one became known as the "indurated," "Hunterian," or "infecting" chancre, and the other as the "soft" or "non-infecting" chancre. His account of the infecting chancre need not be quoted in this place, except that I must mention the important fact, which was now brought out for the first time: that the ordinary slight secretion of such a sore cannot be inoculated upon the bearer of it, nor upon any other individual who has already had syphilis. Bidenkap has, indeed, since found that there are occasional exceptions, it being sometimes "auto-inoculable" during an early stage of the disease, when there have as yet been no constitutional symptoms. But such observations evidently do not affect the principle on which the rule is explained, since they are strictly parallel with the well-known circumstance that vaccination can be successfully performed at the commencement of smallpox before the general eruption appears. Ricord's description of the non-infecting chancre was that it begins (without any incubation) in a pustule, which in two or three days breaks; that there is then formed a deep, punched-out ulcer, with irregular

and slightly undermined edges, a grey surface, and a soft or at least not an indurated base; that it secretes pus freely, and that this is inoculable again and again upon the same individual, and also upon all other persons, whether affected with syphilis or not. One consequence of the difference in the inoculability of the two affections is that whereas a patient seldom presents more than one indurated chancre, several soft chancres are often seen side by side. Those who have recently investigated the histology of the latter affection have found that it is not so different as might have been expected from that of the primary lesion of syphilis. There is a similar infiltration of cells into all the tissues, including the walls of the blood-vessels, but it is said that the channels of these vessels are dilated, instead of being narrowed. The cicatrices left by non-infecting chancres are often hollowed out and very irregular in form, so that they are much more conspicuous than those which finally result from typical Hunterian sores.

There does not appear to be any doubt as to the correctness of Bassereau's observations, nor as to the general validity of Ricord's distinctions between the two kinds of venereal sore. But subsequent experience has shown that the practical application of those distinctions in the prognosis of such affections is liable to certain sources of error. And the theoretical question still remains under discussion, whether the poison of the soft chancre is or is not in its origin independent of syphilis. It is hardly necessary now to allude to the confusion which at first prevailed in consequence of the way in which Ricord and those who followed him formulated their doctrines. Instead of speaking of the "duality" or "unity" of the chancre, they insisted on the *duality of the syphilitic virus*. The inevitable result was that they drove from their camp all those who regarded syphilis as a single specific disease, comparable with the contagious fevers. And yet for such persons there was no place among Ricord's opponents, who advocated the *unity* of the local soft sore with that which is followed by constitutional symptoms. This, however, is scarcely more than a verbal difficulty.

The chief considerations bearing upon the theoretical question as to the origin of the contagious principle of the soft chancre are the following:— In 1854 Clerc met with a case in which an indurated chancre on the scrotum, followed by secondary symptoms, had inoculated itself upon the skin of the penis with which it came into contact, producing there a sore which remained free from induration. Another patient under his observation had an indurated chancre, the grey surface of which suggested that its secretion would be inoculable; the experiment was tried on the patient himself, and the result was a soft sore which reached the size of a franc-piece. It is evident that these facts are easily explicable on a theory of Rollet's, to which I shall presently refer, namely, that "mixed sores," containing both the virus of syphilis and the contagion of the soft chancre, are not uncommon. But the view which Clerc adopted was that the soft chancre is nothing else than the product of inoculation from an infecting chancre upon a person who is suffering or has suffered from constitutional syphilis. And, as if there were not already sufficient confusion, he proposed to limit the use of the term chancre to the indurated primary lesion of syphilis, and to call the soft sore a "chancroid." Shortly afterwards Mr Henry Lee showed that an indurated sore, when irritated by savine ointment or powdered savine, could be made to pour out a purulent fluid which, when inoculated upon the bearer, or upon other patients, produced without any incubation a sore having the characters of a soft sore and capable of being inoculated again and again.

And, lastly, Pick, of Prague, found that pus from a case of scabies, or pemphigus, or acne, was also capable, when inoculated upon syphilitic subjects, of generating a similar sore, although it failed to affect healthy individuals in the same way. From these experiments Clerc's hypothesis derives far more support than from his own observations, because the alternative theory of Rollet can be altogether set aside. And if it is really the case that the sores obtained by Lee and Pick are identical with soft chancres, I think that one may be justified in concluding that such chancres are a kind of bye-product of syphilis, and that their contagious principle is generated *de novo* in persons suffering under that disease. As I shall presently point out, there is reason to believe that in mucous patches (or flat condylomata), themselves due to syphilis, there often arises a virus which is capable of transmitting them, independently of all other symptoms of syphilis, to healthy individuals. And such facts are fairly comparable with those which seem to show that in various morbid states of the human body animal poisons may be developed which may set up erysipelas, or even perhaps diphtheria, in other patients. But I cannot agree with Mr Hutchinson in speaking of soft chancres as "abortive inoculations," since what creates all the difficulty in explaining their origin is that their contagion possesses a virulence of a certain kind which is wanting in the secretion of a true infecting chancre. That it is not a mere question of the presence or absence of pus is shown by Ricord's observation that the matter from the interior of a suppurating gland produces a soft sore when inoculated, whereas this is not the case with the matter which is formed in the connective tissue outside it.

From a practical point of view, however, the origin of the soft chancre is of minor importance in comparison with the question whether it contains the virus of syphilis: that is, whether it remains local and can only propagate itself, or whether it is liable to be followed by constitutional symptoms. In principle I believe that the first of these statements is the correct one, but unfortunately it is open to exceptions, which interfere with the application of it in the prognosis of individual cases. I have already alluded to the theory of "mixed chancres," promulgated by Rollet, of Lyons, in 1858. Now, if a person is infected at the same time with the poisons of the soft chancre and of syphilis the necessary result must be the immediate production of what appears to be nothing but a soft chancre, which will not assume an indurated character until the lapse of three or four weeks, corresponding with the incubation of syphilis. Such sores, I believe, constitute an actual majority of the primary syphilitic lesions which are seen in the hospitals of large cities. It is, however, a question whether the rapid spreading of the base and sides of a soft sore, which sometimes occurs, may not lead to the destruction of any syphilitic poison that may be present in it, and thus save the patient from further ill-effects. In this way it might be possible to account for an occasional failure in the transmission of syphilis from a person suffering under that disease to one who has never had it, notwithstanding the occurrence of a chancre in the latter individual. Bäumler states that there are only seven experiments on record in which the artificial inoculation of unquestionably syphilitic secretions upon healthy persons has not communicated the constitutional affection. On the other hand, it not unfrequently happens that a male patient derives syphilis from a woman in whom a soft sore is the only discoverable primary lesion; and the best observers have been obliged to allow that they have seen instances in which a sore that has at no period been indurated was followed in the same individual

by secondary symptoms. Mr Morgan, of Dublin, has especially insisted on the fact that in the Lock Hospital of that city he scarcely ever saw indurated chancres in the very class of women from whom the men who come to the institution derived their venereal diseases. But this proves less than might at first sight appear, now that we know that the secretions of moist secondary eruptions contain the specific virus. And Mr Cooper Forster has stated that at Guy's Hospital he generally succeeded in finding a characteristic primary affection in female patients when he carefully looked for it. It is usual to explain away all difficulties in the matter by saying that the induration is sometimes very slight and parchment-like *(parcheminée)*, so that it can be detected only by taking the sore between the finger and thumb in a particular manner, which is impracticable in the case of a female with a sore situated within the vaginal orifice. But this is in reality equivalent to the admission that the primary lesion of syphilis does not invariably present the distinctive characters which are assigned to it. Conversely, the typical induration is sometimes closely simulated by inflammatory thickening of the floor of a soft chancre as the result of irritation by caustics.

Thus it appears that in practice, although it is easy to say that certain sores are almost sure to be followed by secondary symptoms, it is seldom, if ever, possible to assert that any sore will *not* be so followed. It must consequently be unadvisable to attempt to limit the use of the term "chancre" to sores from which the syphilitic virus is absent, as has been proposed by some writers, including Mr Forster. We had much better keep to its original meaning, and apply it to all *ulcers*, whether syphilitic or not, which are directly due to venereal infection. But I think that when an indurated papule constitutes the primary lesion of syphilis it should not be called a chancre.

Constitutional symptoms of syphilis are sometimes seen in persons in whom no primary affection is known to have occurred. One source of fallacy in these cases is that a primary lesion sometimes assumes the appearance of a secondary one. Thus, a parchment-like glazed papule on the glans penis may be undistinguishable from the elements of a general eruption among which it lies, while on the labium the affection may be so modified as to simulate a "broad condyloma" or "mucous tubercle." In other instances, perhaps, it is so inconspicuous that the patient never notices its presence, and it quickly subsides, leaving no mark. But some observers admit a true "*syphilis d'emblée*," *i. e.* the invasion of the general malady without a local lesion.

Premonitory stage.—Early syphilis is further characterised by swelling and induration of those *lymphatic glands* which bear an anatomical relation to the seat of the primary chancre or papule. So constant is this symptom that Fournier failed to detect it in only three out of 265 cases in males, and in the same number among 223 cases in females. The glands which correspond with the genital organs are, of course, those in the groins. An indurated sore upon the finger leads to enlargement of glands in the axilla, or at the bend of the elbow, or in both places. One upon the lip affects those near the lower jaw. These "amygdaloid" glands reach the size of filberts, or may-be a little more, but they never approach the dimensions of the suppurating bubo which accompanies a soft chancre. They feel hard, are freely moveable, and are seldom tender or painful. There is generally no reddening of the skin over them. They scarcely ever suppurate. Sometimes

along the dorsum of the penis an inflamed lymphatic vessel can also be felt like a cord.

This "indolent bubo" of syphilis, as it is termed, appears a few days later than the primary lesion. It commonly includes a chain of glands, and may be present in both groins at the same time. It runs an exceedingly slow course, so that it often does not begin to subside until six months have elapsed. Thus it may be very useful in diagnosis, by indicating the probable seat of a chancre or papule, the marks of which might otherwise have been overlooked. One must, however, remember that syphilis is not the only possible cause of a similar affection of the inguinal glands. Mr Cooper Forster has insisted on the frequency of such an occurrence in those who habitually irritate the skin of the buttocks in rowing. I think I have also seen it in young men accustomed to take much walking exercise. Whether glands in other regions are liable to become enlarged apart from syphilis I do not know.

The appearance of the primary papule, or chancre of syphilis, is followed by an interval during which we may suppose that the virus is undergoing multiplication in it and in the adjacent glands. And as the lesion itself now and then subsides in the meantime, the period is sometimes spoken of as the "second incubation." Its length is generally six or seven weeks; but it is liable to wide variations, as is shown by the fact that where the disease has been inoculated experimentally it has been found to range from twelve days to twenty weeks. Probably it is shorter in persons who are weakly, or whose habits are dissolute or intemperate, than in those whose general health is good. During its course some patients look and feel well. But others—it is said, particularly women—complain of malaise and depression, and acquire a pale and haggard aspect.

Secondary stage.—The general or *constitutional* symptoms of syphilis are variously ushered in in different cases. In some there is well-marked fever, the temperature rising suddenly, or gradually, until within a few days it may reach 104°. With the appearance of an eruption it sometimes declines, but it may run on for several weeks, assuming, as Bäumler has shown, an intermittent type resembling that due to malaria. The proportion of cases attended with febrile disturbance is stated by Güntz, who has specially studied the question, at 20 per cent.; but Bäumler thinks that it is really higher.

Another, and a very characteristic, symptom of this period is what French writers term "bitemporal neuralgia;" the occurrence of more or less violent pains, which shoot upwards along each temple. They are not felt at all periods of the day, but come on, often with surprising regularity, in the evening or at night. A similar tendency to nocturnal exacerbations, indeed, belongs to all other painful syphilitic affections. Ricord used to ascribe it to the warmth of the bed, and said that in persons whose occupations compelled them to sleep during the day, the time at which the pains returned was reversed. But, whether or not this is the fact, there can be no doubt that his explanation of it is incorrect. For the hour at which the bitemporal neuralgia sets in is often early in the evening, while the patient is still up. And Bäumler is probably right in thinking that it coincides with an increase of fever, and is in some way dependent upon augmented vascular excitement. Pains in the back and limbs may be present. There may even be swelling of joints. I have seen a distinct, though slight and painless, enlargement

of some of the articulations of the fingers; and Bäumler speaks of patients seeking advice for an affection of the metacarpo-phalangeal joint of the thumb, but leaving it to the physician to discover that they have syphilis.

Syphilis of the skin.—Unlike all other specific poisons, the virus of syphilis produces not merely one or two definite cutaneous affections, but an immense variety of them. They are known as "syphilides" or "syphilodermata" (more properly syphilodermiæ); but in consequence of the way in which they shade off into one another they almost defy description, and are classified with the greatest difficulty. With regard to the causes which lead to the evolution of one of these eruptions, rather than another, all that we at present know is that a patient who is in a bad state of health is most likely to have those forms which suppurate and ulcerate. The differences between them are by no means attributable to tendencies towards particular non-syphilitic cutaneous diseases; as, for instance, that a person liable to ordinary psoriasis is specially apt to be affected with a squamous syphilide, or one who has lichen with a papular one. We should, therefore, avoid using such names as "syphilitic lichen," "syphilitic roseola," or "syphilitic psoriasis." Indeed, however difficult the diagnosis may occasionally be in individual cases, syphilitic affections are entirely distinct from the non-syphilitic eruptions which may simulate them, in pathology, in prognosis, and in treatment.

There are certain features which belong, more or less, to all the syphilides. (1) One of them is a peculiar colour, which is commonly said to resemble raw ham, or to be "coppery;" according to Bäumler, the former comparison has been traced to Fallopius, who wrote about three hundred years ago; the latter only to Swediaur, at the commencement of the present century. The cause of the tint is probably a chemical change in disintegrating blood-discs which have been extravasated into the tissues, and therefore it is not present at first; while, on the other hand, a very similar appearance is sometimes displayed by non-specific eruptions, of chronic course, especially in the legs, where the venous circulation is apt to be embarrassed. (2) A second character of syphilitic cutaneous affections is their multiplicity or "polymorphism" in the same patient, and at the same time. Sometimes macules, papules, pustules, scaly patches are so intermingled that we cannot say which of them preponderates. Or there may be little more than crusts and indefinite patches, to which no positive character can be assigned. In my opinion, it is only in such cases that one can be justified in setting down an eruption as syphilitic for no better reason than the fact that it does not correspond with any known skin disease; but I believe that this method of diagnosis was formerly often adopted, even when the characters of the affection were obviously peculiar. (3) A third point, which is somewhat special to the syphilides, is their tendency to arrange themselves in circles or semicircles, or less completely annular forms. (4) Unless their development is very rapid, they are seldom attended with itching; in many cases, the patient experiences nothing whatever to draw his attention to the skin. (5) The earliest rash, or any universal syphilitic eruption, may be symmetrical just as the exanthem of measles or smallpox is symmetrical, because it affects the symmetrical human body. But syphilides do not show the characteristic symmetry of psoriasis, eczema and some other cutaneous diseases, in picking out corresponding parts of the trunk or limbs. They are irregular in distribution. Their favourite localities are the forehead, the soles of the feet, and

the palms of the hands, and they often affect regions rarely visited by other cutaneous affections.

In most cases, however, a correct diagnosis must be based, not so much upon the recognition of characters common to all the syphilides, as upon an accurate acquaintance with each of them individually. It is, therefore, absolutely necessary that we should in some way classify them. Now, there is one main division which but seldom fails to be applicable, namely, between those which are of *early*, and those which are of *late*, occurrence. The former, to which some writers limit the name of *secondary* affections, may relapse several times, but the first appearance of any one of them is very rarely postponed beyond twelve months from the time of infection. The latter, which are often called *tertiary*, seldom appear within the first year, and they may break out for the first time after an interval of many years.

As a fundamental difference between them, Mr Hutchinson insists, perhaps too exclusively, upon the bilateral symmetry of the early syphilides, which is certainly absent in the case of the late ones. It is, in fact, impossible to strip a patient suffering under one of the early eruptions, the "exanthem" properly so called, without seeing the correspondence of its pattern on the two sides of the body and on the opposite limbs, just as we see it in the diffused rashes of scarlatina. Other features are: the large number of the secondary spots or patches, their isolation from one another, their comparatively superficial seat in the skin, their little tendency to ulcerate, and the fact that, as a rule, they leave no cicatrices behind them. On the other hand, the so-called tertiary eruptions consist of comparatively few separate lesions, but they generally run together; they affect the deeper layers of the integument; they destroy the tissues, and are followed by scars. They may be regarded as "sequelæ." These distinctions are not absolute, nor do they apply equally to every form of syphilide belonging to the early and the late group respectively, but the exceptions to them are few.

The chief early syphilides are the following:

1. *The macular or exanthematic syphilide* ("syphilitic roseola"). It consists of rather ill-defined, pale or dark, rose or "copper" coloured spots; irregular in form; of small size, or approaching that of a threepenny-piece, or even larger; scarcely if at all raised above the surface; generally disappearing completely under pressure. Biesiadecki and Kaposi have each investigated their histology, and have found the capillaries surrounded by cells, and an excess of nuclei in their walls. The external coat of the larger vessels also showed round and spindle-shaped cells. Kaposi further detected wandering cells in the papillary layer of the cutis.

This form of syphilide is most constantly seen upon the sides of the chest and abdomen, but sometimes it covers most of the trunk, and appears on the neck and face; on the limbs it is less common, affects the flexor rather than the extensor surfaces, and avoids the hands and feet. As a rule, it takes a week or more in coming fully out, but sometimes it is developed so rapidly that the case may be mistaken for one of measles. Or an opposite error may be committed, and a rash regarded as syphilitic which is really due to copaiba. On the other hand, where there are but few spots, the affection most likely to be mistaken for the syphilide exanthem is Tinea versicolor. A pale and scanty eruption often fades within a fortnight. One which is dark coloured and abundant may remain visible for several weeks. It may then after a time assume a squamous or papular character; otherwise it sooner or later disappears, even when no treatment has been adopted. Probably this

syphilide always comes out for the first time within a few months after infection, but relapses of it may occur at any time during the first, or even (according to Kaposi) during the second year. The maculæ, however, are described as being then larger and darker, and often annular in form.

2. *The follicular syphilide.* Under this name I group together two less common eruptions, which have generally (except by Bäumler) been separated from each other, the first being placed with the papular syphilide under the name of "miliary syphilitic lichen," the second with the pustular syphilides as "syphilitic acne." The present arrangement, however, is much more natural, since the affections in question bear no close relation towards the other forms, whereas they are intimately allied to one another.

One variety consists of small, pointed, dry elevations or papules, each corresponding with the mouth of a hair-sac. They are generally more or less scaly at the summit, and their bases may be surrounded by a minute white collar of desquamating cuticle. They may either be scattered irregularly or collected into groups or clusters. Histologically they appear to be characterised by an exceedingly superficial infiltration of cells into the papillæ close to the orifice of the hair-sac, and by an extrusion of this infiltration downwards along its walls. This eruption is exceedingly slow in its course, developing itself by successive crops at considerable intervals of time, and subsiding only after several weeks or even months. Sometimes the papules pass into vesicles, or become pustular; but even when this is not the case they always leave minute white cicatrices.

The other variety consists from the first of pustules, which, like the papules, are small and pointed, and which have swollen, reddish-brown bases, and are each traversed by a hair. They may be present in immense numbers not only upon the face, but also upon the chest and back and limbs. They dry up into little yellowish-brown crusts.

It is worthy of notice that the minute punctured scars which (contrary to the rule as regards early syphilides in general) result from these follicular affections are often exceedingly useful in the diagnosis of the disease at a later period.

3. *The papular syphilide* ("lenticular syphilitic lichen"). This consists of red shining elevations, firm and solid to the touch, often presenting a marked coppery tinge. In size they vary considerably, some being scarcely bigger than millet-seeds, others as large as peas or even beans. Thus the larger and more typical of them can scarcely be termed papules according to the usual practice of dermatologists, being what would rather be called "nodules" (or formerly tubercles). They develop themselves very rapidly, reaching their full magnitude in a few days. They do not, however, generally all come out at once, but rather in successive crops over a period of some weeks or even months. Histologically, each of them consists of an infiltration of cells into the superficial layers of the cutis, so dense that the boundary-line between the papillæ and the rete mucosum is almost, if not quite, obliterated. The cells themselves are of unequal size and are supported by a stroma with fine meshes. Towards the sides and the base of the papule the infiltration is sharply limited, the deeper strata of the cutis containing no cells, and not being even œdematous. As Kaposi remarks, this strongly suggests that the pathology of the affection differs from that of a mere inflammation.

The eruption is sometimes scattered irregularly over the whole body, sometimes the papules are grouped together in clusters. They may be

more numerous on the neck and on the forehead than elsewhere; in certain cases they are thickly crowded in the naso-labial grooves, at the angles of the mouth, and about the genital organs. They generally remain for some weeks and then subside, leaving dark stains, which in their turn slowly disappear. But not unfrequently they desquamate, in which case the papules may be made to pass into what will be described as the squamous syphilide, and in other instances their summits soften down, and become covered with yellow or brown crusts.

3. *a. Palms and soles.*—In consequence of the thickness of the cuticle of the palms and soles, these parts show, not raised papules, but flat round horny plates, each with a reddish-brown border. And after a time the plates may become detached, forming so many little ulcers, or the adjacent surface may become rough and scaly, and cracks or fissures may be formed. Thus a very complicated affection arises, constituting the chief form of what was formerly called "syphilitic palmar and plantar psoriasis." At this stage the disease is still generally bilateral.

3. *b. Mucous patches.*—Another modification of the papular syphilide is seen on parts of the skin which are delicate in structure, and which habitually touch other parts; and also upon the mucous membranes of the various orifices. In either case the lesion which results is known as a "mucous tubercle" (*C. latum*)—a "*plaque muqueuse*," or patch, a "flat, broad or moist condyloma," the epithet being needed by way of distinction from the "pointed condyloma" (*C. acuminatum*) or "wart" of the genital organs, which is non-syphilitic in origin. This affection consists of one or more broad raised patches, each from the size of a lentil to a half-crown, with a sharply defined edge, and a surface which is sometimes dry and warty-looking, but much more often moist and coated with a dirty grey secretion of a peculiarly nauseous, pungent odour. Histologically they are characterised not only by the usual cell-infiltration of the cutis, but also by a great overgrowth of the papillæ and by a branching out of their summits.

Mucous patches occur, sometimes in large numbers, about the genitalia, and also along the perinæum and round the anus, in the fold of the nates, in the groins, at the umbilicus, at the folds of the axillæ, beneath the breasts, in the neck, between the toes, at the angles of the mouth, and elsewhere. They may become confluent so as to affect an extensive surface almost uniformly; and where this is the case, it is sometimes possible to mistake the affection for an infiltrating eczema. Not infrequently they are so placed upon opposed parts of the skin as strongly to suggest the idea that they spread by a local infection. But, if this is the case, it is one of great theoretical interest, since a patient who already has syphilis is believed to be absolutely protected from the further influence of the virus (cf. pp. 309, 311); so that flat condylomata would possess an independent contagion peculiar to themselves. We shall hereafter see that they are often the means by which syphilis is transmitted to persons previously healthy. In such cases they generally give rise to a typical primary indurated papule, which is followed after the usual interval by constitutional symptoms. But, on the other hand, it is certain that in some cases the affection which they set up in non-syphilitic individuals is itself undistinguishable from a flat condyloma, and cannot be proved either at the time or afterwards to contain the syphilitic poison. As much as this is admitted by Kaposi, but he passes it lightly by with the remark that secondary symptoms sometimes fail to appear even after an indurated primary sore. Yet so common is the occurrence of flat condylomata, apart from all

other indications of syphilis, that careful observers (including several of my surgical colleagues) have in some cases attributed them to mere irritation of the surface by dirt and moist secretions. Moreover, in some countries they have been known to prevail endemically in such a way that their connection with syphilis has been altogether a matter of inference. These considerations seem to me to point strongly to the conclusion that, although the affection owes its origin to syphilis, it yet acquires a contagious principle of its own, which is capable of surviving in full activity after the original syphilitic virus has in some way disappeared or become exhausted. As I have already remarked, this tends strongly to corroborate the doctrine that the soft chancre arises and spreads in a similar manner.

4. *The pustular syphilide* ("syphilitic ecthyma and rupia"). This is rather a collection of several different varieties of eruption, than a single, well-characterised form. It consists of pustules of all sizes, each of which is generally seated upon a firm red base. They are sometimes present in immense numbers, especially upon the face and trunk. They come out rapidly and with much febrile disturbance, but in successive crops, which may be prolonged over many weeks; and they may relapse even after a year has passed. They dry up into brown, or dark green, or black scabs, and leave large stains which ultimately pass into shallow, flat, white cicatrices. But in many cases their inflamed bases continue to spread long after their summits have thus scabbed over. Under such circumstances, as the crusts increase in size and in thickness, the older parts of them are continually being pushed upwards by the aggregation of fresh material beneath; the necessary consequence is that they assume a conical shape and in fact look very like limpet-shells. Some of them may thus acquire an enormous size. This particular variety is commonly called rupia; there is, I believe, no similar affection of non-syphilitic origin. It is to be observed that it resembles the late syphilides not only in being attended with more or less deep ulceration of the cutis, but also in being unsymmetrical. Indeed, it has often been described among the tertiary eruptions.

5. *The squamous syphilide* ("syphilitic lepra or psoriasis"). Contrary to what once used to be taught, this, as an independent eruption, is infrequent. We have seen, however, that the macules and papules of syphilis often after a time become scaly. Now, if such spots go on spreading still further, there arises an affection which may resemble psoriasis. The features which have generally been relied on as distinguishing it are the dirty grey appearance of the scales, their rather scanty development and small size, the copper-like tint of the patches, and the absence of a definite localisation upon the points of the elbows or knees, or upon the extensor surfaces of the limbs generally.

A particular variety, which has been called *the early circinate syphilide*, is characterised by thread-like scaly rings, from the size of a split pea to that of a shilling; they may be formed in very large numbers, especially on the face.

9. *The vesicular syphilide.* That syphilis very rarely produces an eruption of vesicles is well known. Hutchinson, however, speaks of a form which is attended with clusters like those of shingles, but which is bilateral and widely distributed over the body; and Hardy gives three varieties, which he terms respectively eczematous, varioliform and herpetiform syphilide. I am not myself acquainted with any of them.

7. *The pigmentary syphilide.* This also is a somewhat rare affection, which was first described by Hardy. In the museum of Guy's Hospital we have

models and drawings taken from a case which was under Dr Barlow's care in 1856. The patient, a woman, had brownish-black maculæ scattered over the chest, shoulders, and arms. The commencement of the syphilis dated from eight months previously. Under treatment the stains faded to a considerable extent. Hardy speaks of this syphilide as being almost peculiar to the female sex, as occurring principally on the neck and front of the chest, and as consisting of irregular spots, of a *café au lait* colour, of the size of half-franc or franc pieces, situated close to one another, or running together.

Concomitant early affections.—One of the points in which syphilis resembles the acute exanthemata is its strong tendency to affect the *fauces*, apart from all other mucous surfaces, in conjunction with the skin. Indeed, the throat sometimes suffers before any cutaneous eruption can be discovered ; and in other instances an inspection of it reveals morbid changes, which had caused neither pain nor discomfort, so that the patient had no suspicion of their presence. The acute angina of early syphilis appears as a diffused redness, and not in the form of distinct maculæ, contrasting in this respect with the throat affection of measles. There is often a good deal of swelling, particularly of the tonsils. The follicles become enlarged and prominent ; or they may rupture, giving rise to shallow excoriations. In more chronic cases flat condylomata, assuming the appearance of grey patches or ridges, are present on the tonsils or the palatine arches ; or they may be yellow ulcers, with sharply-defined red borders. But the most curious affection of all is one which is seen, not only upon the fauces, but also upon the hard palate, the inside of the cheeks, and the lips. It consists of scattered milk-white spots, which have been termed *plaques opalines*, and which are perhaps best compared with the effects of the application of nitrate of silver upon a mucous membrane. Their shape may be round, oval, or indefinite ; they vary in size, and may run together so as to cover a large surface with an irregular pattern. Sometimes they are slightly puckered, and parts of their surface may be reddened, with only a little white opacity here and there. All their varieties are deserving of careful study, for they are very characteristic of syphilis, although I can hardly agree with Kaposi in thinking that no similar affection ever occurs in those who have not had that disease. They run an exceedingly slow course, and may break out again and again, not only during the early stage, but also long afterwards.

In many cases the *eyes* suffer. The affection most commonly observed is an iritis, attended with the formation of yellowish-red nodules near the edge of the pupil. This is generally bilateral. Mr Hutchinson says that in his experience it arises within the first six months of the disease, if at all. It is generally associated with one of the more severe forms of eruption. Later attacks are said to be always relapses ; they are often limited to one eye at a time. Or retinitis may occur, in which case the fundus of the eye has a hazy appearance, when seen with the ophthalmoscope ; the disc is reddened and swollen, and its margin is indistinct ; there may be many small extravasations of blood. The recognition of this marked change is very important, because of its insidiousness ; Mr Hutchinson speaks of the patient as noticing "nothing, except that his sight is very dim ; he has no pain, no congestion of the front of the eye, no intolerance of light."

The *lymph-glands* in various parts of the body become swollen at this stage of syphilis ; we are especially accustomed to look for enlargement of those which lie one above each elbow, and of those that are seated at the

back of the neck, close to the occipital bone. Whether this is dependent upon any previous affection of skin or mucous membrane is at present doubtful.

Another symptom is *falling off of the hair;* it may come away with the comb so freely that the patient is apprehensive of becoming prematurely bald. I have, however, never myself seen anything like a complete alopecia from this cause. The short hairs from the limbs are shed, as well as those of the scalp.

As to early syphilitic lesions of the viscera, nothing positive is as yet known. Mr Hutchinson has ably insisted on the strong probability that such affections may occur, and he mentions transitory albuminuria as not uncommon. I myself had one patient whose urine was albuminous (and who, I think, had dropsy) at a time when it could hardly be supposed that the vessels had already become lardaceous. I have also occasionally seen jaundice which in one case quickly subsided, but which in another instance ended in acute atrophy of the liver. To this subject, however, I shall return hereafter.

Later syphilides.—After the lapse of the six months, or the year, or two years, during which one or more of the early syphilides has developed itself once or oftener, the disease often becomes altogether latent. In many cases, indeed, it finally ceases, and the patient henceforth remains perfectly free. But in other instances he continues to be troubled, at varying intervals, with slight manifestations of it. Perhaps small scattered pustules appear on the scalp, which are scratched by the comb, and, scabbing over again and again, cause him great annoyance. Perhaps the palm of one hand now and then becomes scaly and fissured in its centre. Perhaps some of the nails grow rough and thick and discoloured. Affections such as these may go on for a very great length of time.

But in some cases, possibly many months or even years after the subsidence of the early syphilides, there appears an eruption presenting definite characters of its own, and yet differing from any that would have arisen during the secondary stage of the disease. I have already indicated what are the chief features common to such late or *tertiary* syphilides, namely, that they are less symmetrical, generally cohere together, affect the deeper layers of the integument, destroy the tissues, and are followed by scars. Their special characters vary widely in different cases.

In 1869 a woman was under my care in hospital who had been infected by her husband twenty years previously, and in whom the disease assumed the form of thin reddish-brown glazed patches, covering the greater part of the face; they were not at all raised, and there was only the slightest possible desquamation from them; in fact, they were scarcely more than maculæ. In other cases, thick white scabs appear upon the circumference of a bold red ring or festooned line. Such an affection, perhaps, deserves to be specially designated as squamous. Or again, immense patches of skin may become thickened, raised, of a reddish-brown colour, and rough with a bran-like scurf; so that the disease looks somewhat like dry eczema. But, as a rule, what constitutes the essential feature of the late eruptions of syphilis is the presence of lurid reddish-brown nodules; these are like those of the early papular syphilide; except that, instead of being scattered, they now cohere in rings or patches, which, again, are often fused together, so as to cover a large surface or to form straggling festooned lines. It is also, I think, a

peculiarity of such late nodules that they invariably leave cicatrices even when they have not ulcerated. But in most instances they either become covered over with crusts or eaten away so as to form small deep ulcers with vertical edges. It is no uncommon thing to see patches, a foot or more in diameter, some parts of which have already cicatrised, while other parts show recent nodules, or serpiginous lines of scabs, spreading over the healthy skin around them. All these varieties are worthy of the most patient clinical study, for they are absolutely characteristic of syphilis, and the patient has often no suspicion of their nature. They have a special tendency to affect the face (particularly the forehead, nose, and lips), the nape of the neck, the shoulders, the back, and the extensor surfaces of the limbs.

As an illustration of the importance of the correct diagnosis of such affections I may mention the case of a gentleman aged fifty-five, who came to me for the most obviously syphilitic ulcerating patches, one on each shoulder, which had been steadily getting worse for a year. His medical adviser had put no question to him as to venereal disease, but when a young man he had some complaint of this nature, attended with swelling of the testis. He had been married for many years; his wife had had three miscarriages but no children. Iodide of potassium cured one of the patches in a fortnight, and the other within six weeks.

Later lesions of mucous membranes.—The tertiary period of the disease is also characterised by lesions of the throat and mouth, most of which differ from those of the secondary stage. *Plaques opalines* may, indeed, continue to form on the cheeks or lips. But the surface of the tongue now for the first time becomes affected in a variety of ways. Sometimes more or less extensive patches appear unnaturally smooth and glossy. Sometimes there arise oval greyish ulcers, especially on its centre or edges. Sometimes its mucous membrane becomes greatly thickened, with deep grooves intersecting it in all directions. All these changes render the contact of salt and pepper exceedingly painful to the patient, and make him glad to confine himself to the blandest possible diet.

In other cases, again, the soft *palate* becomes affected with deep sharp-cut ulcers, which very rapidly perforate it, and eat away a considerable part of its substance. Such ulcers, when they heal, leave well-marked cicatrices. Some years ago a woman died in Guy's Hospital whose velum had long before been extensively destroyed on one side, so that the uvula was held in its place by two thread-like processes of mucous membrane, which looked as if they could not possibly have escaped being torn through during deglutition; the preparation is now in our museum. Sometimes the palate may become adherent to the pharynx, so as to cut off the communication between the cavity of the nose and the air passages below.

*Gummata and nodes.**—That I have not yet mentioned gummata as occurring in the skin and mucous membranes will probably surprise many of my readers; for some writers characterise the tertiary period as the "gummatous stage" of the disease. In the subcutaneous tissue, or in the sub-

* The terms *gumma* and *gummositas* are of considerable antiquity. Fallopius, in the sixteenth century, spoke of tumours of bones as having been called "*gummata gallica*," on account of their containing a matter resembling *gummi eliquatum*. Ernst Wagner proposed to substitute the name "syphiloma" for gumma; but the change is not worth making. "Tubercles" is rightly abandoned, for *tubercula syphilitica* have nothing to do with tubercle in the modern sense of the term, although v. Bärensprung supposed that they had. "Node" is still often used as a synonym of gumma, especially in superficial regions.

stance of the tongue, such tumours often acquire a very considerable size; and the skin or the mucous membrane over them may at length become ulcerated through, so as to expose a grey degenerating mass, of the most typical kind. But it is, I think, impossible to say that tertiary eruptions in general deserve to be called gummatous, to the exclusion of the early ones. I have already remarked that the scattered lenticular papules which may constitute one of the first manifestations of constitutional syphilis have a structure which seems not to be merely inflammatory; and recent observers have shown that the nodules which are found in iritis are small gummata, instead of consisting merely of fibrinous exudation. Again, Mr Hutchinson has mentioned a case in which definite gummata were found in both testes and also in the spleen, although a secondary rash was still out on the skin of the patient, who died of "syphilitic disease of the heart—myocarditis with a gumma."

The gummatous affections of internal organs are, indeed, by far the most important of the effects of syphilis, since they produce various symptoms, and not rarely destroy life. But I shall leave them to be described among the local visceral diseases, since I could in no other way bring their clinical relations into sufficient prominence. In this place I shall content myself with insisting on the necessity of carefully examining the bones and the testes whenever a syphilitic taint is suspected.

It is believed that those *bones* which, like the skull-cap, clavicles, ulnæ, and tibiæ, are but thinly covered with soft parts are more than others liable to be affected. By running one finger along the surface of these bones it is easy to discover whether there is any swelling of them, or whether the patient shrinks from pressure. The enlargement caused by syphilitic periostitis generally rises gradually from the surrounding surface, but I remember an instance in which the frontal and the parietal bones presented bold hemispherical tumours that at first appeared as if they must be malignant growths, especially as one upper eyelid was enormously swollen and in part gangrenous; the effect of treatment with iodide of potassium in this case was wonderful. When such osseous affections are recent, they often feel soft and semi-elastic, and at an autopsy one may find that there is a grey succulent material, more or less extensively caseating, which can be shelled out of the excavated substance of the bone. At a later stage the soft substance may be absorbed, and there may be left a central depression, with a thickened zone around it. But upon the bones of the limbs, I think that the whole of the gummatous growth is more often converted into a uniformly dense, raised, bony mass.

The *testes*, when affected with syphilis, often present firm yellow gummata, of irregular form, which are embedded in the proper substance of the organ, or in the epididymis, and which can often be easily detected during life. But in many cases a diffused fibrous thickening, with atrophy of the secretory structure, is all that one discovers at an autopsy. This, no doubt, suffices to account for the fact that so many of these patients are unable to beget children. But I suppose that one could hardly in the living subject recognise such an affection by palpation, distinguishing it from the flaccidity which may accompany any serious disorder of the general health. Nor am I satisfied that it is in itself a proof of syphilis; it is certainly found in the *post-mortem* room in many cases in which there is no other evidence of that disease. Still more doubtful is the significance of closure of the extremities of the Fallopian tubes in women.

Diagnosis.—From the foregoing description of syphilis it must be sufficiently evident not only that the recognition of a disease of which the symptoms and effects are so numerous and varied must often be very difficult, but that to attempt to lay down any rules for its diagnosis would be of very little use. Sometimes the first glance at a patient reveals to a skilled observer the nature of the case beyond a possibility of doubt; and he is bound to treat it actively, particularly in a married woman, without asking questions which might cause endless domestic misery. In other instances, even when all inquiries can be pushed without the least reserve, one is unable to hazard a positive opinion until one has watched the effects of remedies for a considerable time. It is important to remember that the body should always be stripped as thoroughly as possible, and that every part of it should be looked at before one pronounces as to the character of a doubtful eruption. Some of the most puzzling cases are those in which several cutaneous diseases are present at the same time. I well remember one instance in which a macular syphilide was almost hidden among the nodules of acne indurata and the papules and pustules of scabies.

Transference of contagion.—With regard to the ways in which syphilis is transmitted, it is perhaps worth notice that among married women belonging to the lower classes the disease is often traced back to a confinement, when in all probability the patient really derived it from her husband after her convalescence, he having become infected as the result of adulterous intercourse while she was in childbed.

Few theories in medicine have been better disproved than the doctrine of Ricord that primary syphilitic lesions alone possess the power of propagating the disease. Clinical experience has furnished numerous instances of contagion from flat condylomata, and experiments have been made which show that it is possible to convey the virus to healthy persons by inoculation with the blood of syphilitic patients, or with matter from pustules of a secondary eruption or from an ulcer of the tonsils. Whether pus from a late or tertiary affection of the skin contains the poison seems to be still doubtful. There seems to be no doubt that the surface of the skin must be abraded, or fissured, to allow of the penetration of the virus, and the most recent writers think that this is true even of mucous membranes.

There are undecided questions as to the contagiousness, apart from admixture with purulent discharge, of the natural secretions of the salivary, mammary, or lachrymal glands; of the semen, without impregnation of an ovum; or of inflammatory exudation from a mucous membrane, as, for instance, the gonorrhœal pus in one who has also had syphilis. With regard to this last point it is suggested that if pus-cells are wandering leucocytes from the blood, they must almost certainly contain any virus which the blood itself contains. But it is generally held that the saliva and the milk are not in themselves capable of conveying the disease. That mixed fluids from the mouth may form the vehicle by which the virus passes from one person to another is shown by cases of infection in the act of kissing, in glass-blowing, when the same metal tube is employed by different workmen in turn, in smoking, if one cigar or pipe is used by several individuals, or (as in a case related by Dr Baxter) in cleaning the teeth with another person's brush. Nor must we forget that we may unintentionally infect our patients if we omit thoroughly cleansing a Eustachian catheter or a laryngoscopic mirror, after using it in a syphilitic case. The disease has

also been transferred by one person biting another in the hand. Lastly, accoucheurs and midwives have acquired primary sores on the finger when they have overlooked a slight abrasion upon it during their attendance on a woman with flat condylomata or other specific affections of the genitalia.

Special pathological points.—I have already set forth the analogy between syphilis and the specific fevers (p. 308), an analogy which has been particularly well stated by Mr Hutchinson, and I must now briefly discuss certain questions with regard to its pathology, some of which are of great practical consequence, while all of them have important theoretical bearings upon the study of contagious diseases in general.

Concerning the nature of the primary indurated papule or chancre two opposite opinions are entertained. Some think that it is itself a local expression of an infected state of the blood. Others, following John Hunter, suppose that the virus does not reach the blood until after it has multiplied itself at the seat of inoculation and has passed along lymphatic vessels, so as to be discharged with their contents into the great veins. In support of the second view, attention has recently been drawn to the fact that, in the ordinary indolent inguinal bubo, the glands which lie along the iliac vessels become swollen as well as those which lie superficially in the groin. This is said to be shown by three preparations at the Lourcine Hospital in Paris, each of which was taken from a woman who died of some intercurrent disease while suffering from sores upon the vulva. But, as Bäumler remarks, if such were the mode of transmission of the syphilitic poison, one would expect that under ordinary circumstances the constitutional symptoms should be longer delayed than when, the seat of the primary affection being on the lip or on the finger, only a single group of glands would have to be traversed. Moreover, even if the *irritation* which causes the glands to swell does spread from one gland to another, it by no means necessarily follows that *infection* of the blood takes place by the same slow and circuitous route. The analogy of vaccination seems to suggest that it is quite possible for an indurated papule or chancre to be the direct result of inoculation of the specific virus *in situ*, and yet for the blood to be already poisoned from the moment when such a lesion begins to be developed. The rarity of multiple primary syphilitic sores, and the fact that the secretion of a primary sore is scarcely ever inoculable upon the patient, point strongly in the same direction. One might have expected to settle the point at issue by excising indurated chancres, or destroying them with caustics at their very commencement, but at present the evidence as to the effects of such operations is conflicting.

Another question concerns the relation between the secondary and the tertiary stages of syphilis. Dr Wilks has long thought that visceral lesions are most frequently met with in cases where cutaneous eruptions and other conspicuous symptoms of the disease had been absent or but slightly marked. But, although I am inclined to adopt this opinion, I do not know that it has been positively proved to be correct; and, if it were, we should still have to ask whether it is not the omission of early specific treatment in the cases in question which determines the occurrence of remote effects. An opposite doctrine is maintained by Mr Hutchinson, namely, that the tertiary phenomena—being, as we have seen, unsymmetrical—ought properly to be regarded rather as sequelæ than symptoms of syphilis. His expression is that they are "regrowths," in morbid structures left behind from the secondary period. Of the points which he makes in support of his suggestion, the most

important seems to me the fact, that the liability to outbreak of tertiary symptoms sometimes continues after the patient has produced a family of healthy children, and when one can therefore hardly suppose that his blood still retains the virus.

It must not be imagined that this matter is one of merely theoretical interest. A patient affected with syphilis is apt to be exceedingly pertinacious in his inquiries as to whether the disease will leave a permanent taint. He has heard of instances in which, after the lapse of many years, tertiary symptoms have appeared; and he has acquired a notion that the syphilitic poison differs from the contagious principles of all other specific diseases in being never eradicated. But I think we are justified in assuring him that the distinction is one of degree rather than of kind. In the immense majority of cases a person who has had syphilis is after a few years free from it in every sense in which it can be said that one who has had scarlet fever or smallpox is free from those diseases. In each instance the only appreciable difference between the individual who has been attacked and other people is that he is no longer capable of taking the infection. And since we are not accustomed to speak of the exanthemata as remaining uncured during the whole lifetime of the patient, or until he falls exceptionally ill with them a second time, I do not see why we should adopt a modern fashion and use language of this kind with regard to syphilis.

Opinions probably differ very widely as to the extent to which the presence of a syphilitic taint *modifies* other diseases. Some surgeons have held that it may interrupt the natural course of almost every morbid process; that a broken bone may fail to become united, and a wound refuse to heal, until the virus is neutralised by the administration of specific medicines. But I very much doubt the fact of such occurrences, unless, indeed, the general health of the patient is profoundly impaired. On the other hand, it is well established that an injured part may gradually take on the characters of a syphilitic sore; but then it is also the case that psoriasis or eczema may develop itself upon spots which have been bruised or lacerated in a person liable to one or the other of these diseases. There is no reason to believe that the effects of definite diseases like scarlatina or diphtheria on the throat, rheumatism on the joints, or psoriasis, scabies, and eczema on the skin, are the least modified by the presence of syphilis. The two diseases may occasionally be seen in the same person, each unaffected by the other.

With regard to *protection*, it is well known that a person very rarely has an indurated chancre and afterwards a secondary eruption and sore-throat on two different occasions; but several instances of such an occurrence have now been recorded. In a case of Mr Hutchinson's the interval of health was only three years; it has generally been much longer. The second attack has always been very mild. On the whole syphilis protects against itself more perfectly than measles or scarlatina, though probably not so perfectly as variola.

Prognosis.—This must be discussed with the treatment of syphilis, if we are right in believing that the administration of medicines has immense power in cutting it short. To adults, indeed, it is scarcely ever directly fatal. Bäumler mentions, as the only two ways in which it can destroy life, hæmorrhage dependent upon spreading ulceration (*phagedæna*) and exhaustion appearing when the early symptoms are very acute and severe. The only case that I know to have come down to the *post-mortem* room at Guy's Hospital

during the last twenty-five years, in which death was attributed to syphilis, apart from any visceral lesions, is that of a man, aged thirty-one, who lay in one of the wards for seven months, with a rupial eruption on the skin, and complaining of pain and swelling in his joints. The most exteme wasting took place; his knees and his left arm were rapidly contracted; his wrists and ankles were enlarged. Towards the last he was drowsy and listless, and at the autopsy the brain appeared to contain an excess of fluid. Indirectly, however, as we shall see in the chapters on diseases of the brain, larynx, lungs, liver, kidneys, syphilis is the cause of a very large mortality. If, therefore, I say that the prognosis is almost always favourable, it is because I hold that by proper treatment we can not only cure the early symptoms, but prevent their remote after-effects which are so dangerous.

Treatment.—Mercury has been employed as a remedy for syphilis during nearly four hundred years, but unfortunately the experience which appeared to justify its use was long vitiated by a most obvious source of error; the natural course of the disease, when unmodified by drugs, had never been carefully studied. Moreover, the metal was generally given so freely as to cause profuse salivation, with the most pernicious results. Thus, when certain English army surgeons, in the early part of the present century, introduced a "simple treatment," in which mercury found no place, they had the immense advantage that their patients remained free from many evils to which persons suffering under syphilis were then commonly subjected by their medical attendants. And what made the difference the more striking was that the real nature of syphilis as a specific disease being then altogether unknown, the same methods had been employed indiscriminately for all venereal sores. Is it matter for surprise that those who advocated this great revolution in medical practice should have overshot their mark, and attributed to mercury not only the injurious effects which it really does cause, but also many of the remote consequences of the disease itself, such as are now known to occur when no medicine has been administered?

It is now an established fact that it is possible for not only the primary lesion of syphilis, but also the various secondary and tertiary eruptions to subside spontaneously without any specific treatment; indeed, this result would occur (sooner or later) in most cases, if the patient is placed under favourable conditions as regards rest of body and mind, good diet, and pure air.

Nevertheless, experience tends irresistibly to the conclusion that a patient who has syphilis derives immense advantages from a course of mercurial treatment, provided that it is judiciously managed. Among those who are best qualified to form a judgment upon this matter there is at the present time hardly any difference of opinion; and what adds to the force of this consensus is that it has been arrived at by a variety of methods, and that some of those who now most warmly advocate the use of mercury were originally in doubt or in opposition.

1. It is certain that mercury, properly administered, is in no way injurious to the patient's general health, if he is otherwise sound. Even in the case of a person with a tubercular tendency, the drug may almost always be given with perfect safety, although one should perhaps be more cautious with it. Nor would the actual existence of visceral disease prevent my prescribing it; except that one naturally would refrain from treating a mild form of syphilis at all, if the patient were obviously about to die in a few months of

phthisis, cirrhosis of the liver, or renal dropsy.* It is generally thought that syphilitic affections attended with much ulceration should, if possible, be treated by other remedies rather than mercury, but Mr Hutchinson is now inclined to doubt the correctness of this opinion. The cachexia resulting from syphilis, so far from being a reason for avoiding the use of mercury, is generally quickly curable by it.

2. Mercury has a most marked effect upon the induration of a primary infecting papule or chancre. The treatment of such affections belongs to surgery rather than to medicine and I shall not further touch upon it.

3. Mercury if administered systematically during the existence of a primary indurated sore will often prevent the development of secondary symptoms. This is perhaps more important than any other point connected with the treatment of syphilis, and it deserves the most careful verification. At present the best evidence in favour of it is the statement of Mr Hutchinson in a paper read before the Hunterian Society in 1874, especially when contrasted with his previous utterance on the same subject in Dr Reynolds' 'System of Medicine.' Formerly he thought that secondary symptoms were for the most part inevitable whatever treatment might be adopted. But in a series of eleven cases of vaccino-syphilis in which he began to give mercury two months after contagion and about a fortnight after the appearance of the specific induration not only did every patient quickly get rid of chancre and glandular enlargement but only about half of them ever showed secondary symptoms: these, moreover, were almost always of an exceedingly mild type, and did not appear until many weeks later than they would have done if no medicine had been taken. Now, where syphilis has been intentionally inoculated upon healthy persons constitutional effects have scarcely ever been wanting; and the same may be said of cases of vaccino-syphilis in which early mercurial treatment has not been adopted.

4. Mercury renders the secondary symptoms of syphilis mild and of brief duration in proportion as its administration is begun early and is carried on regularly and without intermission for a considerable time. Upon this point also Mr Hutchinson has recently spoken very decidedly. Formerly he cited instances in which a patient, while actually salivated for iritis in one eye, became attacked with the same affection in the other eye, as tending to establish an opposite conclusion; but now he says that in such cases the failure is probably due to the fact that the "salivation is premature," that is, that the remedy has not yet excited its full influence upon the rest of the body, notwithstanding that it has affected the gums so severely. "The secret of success is to avoid any interruption of this kind," and he insists upon the fact that cases in which the secondary symptoms of syphilis are exceptionally severe are almost always cases in which mercury has not been given. My own experience leads me to confirm this statement very strongly.

5. Mercury if administered efficiently during the primary and secondary stages of syphilis will probably diminish the frequency and severity of tertiary symptoms. Here, indeed, we come to a point as to which it is and must be difficult to obtain direct proof, for there are no certain data as to the proportion of cases of syphilis which result in tertiary symptoms when

* Even in such cases the chance of our having made a false diagnosis would probably lead to antisyphilitic treatment. For the supposed "phthisis" might be syphilitic peribronchitis and interstitial pneumonia, the supposed "cirrhosis" syphilitic gumma of the liver, and the supposed "Bright's disease" lardaceous degeneration from syphilis.

no treatment is used; nor do we yet know to what extent their occurrence may depend upon the severity and character of the secondary symptoms. But there is at least a strong presumption that if the drug can prevent the earlier and more constant of the phenomena of syphilis, it will not have less power over the more remote and exceptional effects of the disease.

If these statements should prove to be correct, it would evidently be the bounden duty of a medical man to prescribe mercury to all patients suffering under primary or secondary syphilis who should place themselves unreservedly under his care. And I entirely agree with Mr Hutchinson that we cannot insist too strongly upon the necessity of continuing the remedy regularly for many weeks after the entire subsidence of every visible symptom of the disease.

There has been some discussion as to whether mercury should be called an "antidote" to the syphilitic virus. Mr Hutchinson is disposed to give it this title. But, for my own part, I should prefer to avoid using any expression which would imply that the cure of syphilis must depend upon, and be preceded by, the disappearance of the specific contagion of the disease. I think it is at least as likely that mercury acts directly upon the living tissues, depriving them of their susceptibility to the particular kinds of morbid action which syphilis induces. The notion of an antidote is, we believe, inapplicable to such a case as the cure of psoriasis or pemphigus by arsenic; and I do not think that it could properly be extended to include the cure of gout by colchicum. But surely the use of mercury for syphilis has a far more obvious relation to these therapeutic measures than to the treatment of cases of poisoning by antidotes or of parasitic diseases by germicides.

With regard to the relative advantages of the various preparations of mercury, differences of opinion prevail. But I think it is clear that we should employ in preference those which are mild; it is rarely advisable to prescribe calomel or blue pill, in frequently repeated doses, except, perhaps, when it is necessary to produce a rapid effect, as when iritis or retinitis is present. Plumer's pill is very useful in these not infrequent cases. I cannot believe that the hypodermic injection of the bichloride of mercury, has any advantages which counterbalance its pain and its tendency to set up inflammation. I have generally been content to administer this preparation by the mouth in doses of one sixteenth to one twelfth of a grain, three times a day. Another preparation which may often be used with advantage is the *hydrargyrum cum cretâ*, in doses of two or three grains; when the gums appear likely to be too quickly affected, further inconvenience may be obviated by letting the patient at the same time take the chlorate of potass; but, as a rule, it is not desirable to mask the effect of the mercury in this way.

Sometimes, when no mercurials, taken internally, could be borne, the inunction of blue ointment has succeeded perfectly. But best of all, in really difficult cases, is the calomel vapour-bath of Mr Lee. About fifteen grains may be volatilised by a spirit lamp over a water bath, the apparatus being placed beneath a chair on which the patient sits, naked, and wrapped round with blankets. The bath should last from a quarter of an hour to twenty minutes.

Except during the cold seasons of the year, the patient may go out, and even continue at his occupation, while he is carrying out a mercurial treatment. He should, however, keep early hours and take plenty of rest, his clothing should be made of flannel, and other warm materials; he should have good food, but should generally abstain from stimulants. Pure air is important to him; there are greater advantages in residence at the seaside

than at Aix-la-Chapelle or other continental spas, where, if report says true, the real method of cure is neither the baths nor the waters, but mercury.

Even when syphilis has reached the tertiary stage, mercury should, I think, as a rule, be prescribed, if the patient has not already gone through a systematic course of it. But in the majority of cases the remedy which is now most serviceable is Iodide of Potassium. This is, indeed, often employed with advantage, even during a protracted secondary period; but over the early macular and papular eruptions it has little if any power. With regard to the dose of it, there has been a good deal of difference of opinion. But of late it has become the general practice to give from ten to thirty grains three times a day. It is generally prescribed with spiritus ammoniæ aromaticus and some bitter tincture or infusion. But some persons take it more readily in milk, which they keep on a table beside them and sip from time to time.

Even when iodide of potassium does not finally eradicate the disease, it often exerts so marked an influence over the symptoms that the patient will go on with it more or less continuously for years. In such cases it is generally desirable to interrupt its use from time to time; and important services are then rendered by the Chlorate of Potass. I have repeatedly seen obstinate forms of cutaneous eruption removed by this salt, given in doses of ten grains, three times daily. Another medicine which may now be prescribed is the dilute nitric acid, and still more valuable is, I believe, Sarsaparilla. I have used the fluid extract with results which appeared to me to be incontestable. Of the value of guaiacum, which three centuries ago had so high a reputation, I know nothing.

It is not generally worth while to apply remedies directly to the eruptions of syphilis, but some of the more localised tertiary affections are very rapidly brought to subside when they are covered with the diluted blue ointment, and calomel should always be dusted upon mucous patches (*i. e.* flat or moist condylomata). The severe nocturnal pains produced by nodes may often be relieved by blistering the skin over them.

Congenital Syphilis.—This is a modification of the disease, which, instead of being acquired by direct infection, is transmitted to the patient from one or both parents. It differs in important respects from ordinary syphilis. Some modern writers prefer to call it "hereditary" or "inherited," because it does not usually manifest itself by well-marked symptoms until some weeks after birth. Recently, however, the discovery has been made that the bones in the fœtus are often affected by syphilis, and I do not think that there can be any doubt that the malady, even if latent, is really present from the very commencement of extra-uterine life, just as it is in an adult during the period of incubation, or in the intervals between successive eruptions. The term hereditary may well be reserved for diseases such as gout and phthisis, which usually begin at a later age, the tendency to them being all that is handed down.

Method of transmission.—At one time it was thought that infection to the fœtus *in utero* must necessarily come from the mother; even when the father had been the original sufferer, the idea prevailed that she always received the virus first and gave it to her offspring. But observation proved that in a large majority of cases the mother neither showed any sign of the disease nor knew of any symptom of it. All writers admit that the semen may convey syphilis directly to the ovum; and the assertion is frequently made

that the father is the parent from whom the disease is most often derived. But instances must be rare in which it would be possible to declare positively that at no period since her marriage had a woman gone through even a slight form of syphilis, especially as it is not certain whether the semen may not also infect her, independently of the occurrence of impregnation. But little stress, therefore, can be laid upon the well-known fact that a man sometimes transmits the disease to his children notwithstanding that he did not marry until long after the disappearance of all secondary eruptions, nor until a period when, if any further symptoms were to arise, they would be regarded as tertiary and as incapable of being directly contagious. It would seem, moreover, that such cases are in the highest degree rare and exceptional. If one is consulted about the propriety of marriage on the part of the patient who has had syphilis, one should perhaps never declare it to be impossible for the offspring to be tainted: until two years have passed after the complete subsidence of the secondary symptoms of the disease marriage must be altogether forbidden. But lapse of time seems, as a rule, quickly to diminish the risk. Thus, as Mr Hutchinson has pointed out, each succeeding child of the same parents is less likely to suffer than the previous one; sooner or later the taint wears out, and the children subsequently born remain free. Sometimes, however, one infant may escape, notwithstanding that both older and younger ones are attacked.* There are, moreover, modifying circumstances which may cause the later children of a married pair to suffer more than the earlier, even when the syphilis had been contracted long previously. According to Mr Hutchinson, such a result sometimes happens when the general health of the infecting parent becomes more and more broken down from year to year; but the more frequent cause is supposed to be the fact that a healthy woman, impregnated by a syphilitic husband, gradually becomes herself poisoned, so that she forms a second source of infection for the children whom she afterwards brings forth. For there is reason to believe that the offspring are much less likely to escape, and that they are more often affected severely, when both parents are tainted than when only one is.

The full extent and subtlety of the contamination which affects the mother of a syphilitic child were first pointed out by Colles, of Dublin, in 1837. He formulated the remarkable law which now bears his name, that "a newly-born child, even although it may have symptoms in the mouth, never causes ulceration of the breast which it sucks, if it be its mother who suckles it, though it is still capable of infecting a strange nurse." In other words, although the mother may have shown no sign of syphilis, she has yet undergone a modification of the disease, bearing somewhat the same relation to the ordinary form that vaccinia does to smallpox, and no less protective in its action. The *choc en retour* (as French writers have termed the infection of a woman through her fœtus) is not, however, always unattended with symptoms. She not infrequently becomes pale and thin, her hair may fall off, perhaps certain lymphatic glands become swollen, or there may be some swelling of bones. Mr Hutchinson has observed that symptoms may first appear at the menopause in the form of so-called "psoriasis palmaris," sores on the tongue, or induration in the legs. He suggests, as an interesting

* The case is of course different when a husband or a wife contracts syphilis after having already had children; their next infant will then be the one most severely affected. It is generally said that a woman acquiring the disease during her pregnancy may convey it to the fœtus at any period up to the seventh month.

point for inquiry, the question whether a woman infected in this way *per fœtum* can transmit the taint to children subsequently born to a healthy father.

Effects on the ovum.—The results of syphilitic infection on the fœtus vary greatly in different cases. Very often it dies *in utero* and is thrown off after an interval in a more or less decomposed condition, with its cuticle peeling in large flakes as the result of maceration in the dark and fœtid liquor amnii. Thus abortions and miscarriages, especially if several times repeated, afford very valuable indications of a syphilitic taint; so that not only is it important, whenever a woman suffers from any complaint that could be attributed to this cause, to inquire as to the mode of termination of her former pregnancies; but also, when a married man is the patient, to make similar investigations with regard to his wife.

The occurrence of a specific lesion of the *placenta* has long been suspected, but until recently nothing definite was known about it. In 1863 Dr Wilks cited in the 'Guy's Hospital Reports' some observations of Mr Wilkinson King, in which the chorion had been found thickened and coriaceous, and the amnion lined by a false membrane, as the result, it was supposed, of syphilis; in almost every instance abortion had taken place at the third month. Virchow, however, afterwards described the decidua and the maternal part of the placenta as the structures liable to morbid changes in this disease. If his view had been correct, it would have followed that the changes in question must be absent whenever the fœtus derives its infection solely from the father; but there is reason to believe that most of the hard yellow masses in the placenta which have been taken for gummata have been mere residues of accidental hæmorrhages. At any rate, gummata spreading from the maternal into the fœtal part of the placenta were only once found by Fränkel, of Breslau, who in the 'Archiv für Gynäkologie' for 1873 has recorded a series of investigations with regard to this subject. In sixteen cases, however, he discovered a peculiar affection of the villi themselves, a fact of great interest, since it is obviously compatible with an entire absence of infection from the mother. The lesion in question consists in a dense growth of round- or spindle-cells in the substance of the villi, attended with a more or less complete destruction and disappearance of their vascular loops, and ending ultimately in a process of fatty degeneration. Villi so altered are less easily isolated from the structures in which they are embedded; they are swollen and opaque, and have irregular outlines and bulbous extremities. Sometimes the whole placenta is uniformly affected; it is then remarkably large and heavy (even to the weight of two pounds), close, tough, and of a pale greyish-yellow colour. In other instances, only certain portions of it are diseased; these appear as opaque wedge-shaped masses, surrounded by zones of congestion; in the healthy portions there are often hæmorrhages. Once Fränkel found that the cell-growth had extended from the villi into the maternal part of the organ. He is in doubt whether the premature expulsion of the fœtus, which in his cases took place at varying periods from the sixth month onwards, is dependent upon the placental lesion directly or rather upon the death of the fœtus. In some instances gestation appeared to have gone on to its natural limit, and when the morbid change was partial the child was sometimes born alive. The earliest period at which the affection of the villi was detected was at the sixth month; it often seemed to be only just commencing. Thus we have still to learn whether a similar lesion exists when abortion occurs at the third or the fourth month.

Occasionally a syphilitic fœtus shows gummata in the *liver*, but in the

great majority of cases what alone characterises the placental disease as syphilitic is a peculiar morbid condition of the *bones*, which is rarely absent. This affection, first described by Wegner, of Berlin, in 'Virchow's Archiv' for 1870, but independently observed by a French physician, Parrot, at about the same time, seems to begin at a very early period of fœtal life. Like that which constitutes rickets, it has its chief seat at the meeting lines between the shafts of the long bones and their epiphysial cartilages, and, in the case of the ribs, between their anterior extremities and the cartilages which tip them. The meeting lines in question become much thicker than natural, uneven and irregular. But here the resemblance ceases. There is in syphilis comparatively little increase of the normal "zone of proliferation," whereas the "zone of incrustation of cartilage" is enormously exaggerated. It forms a thick layer, dense and homogeneous, but friable, white, and opaque, like mortar; long processes project from it into the substance of the cartilage beyond. As the affection advances, this layer becomes separated from the shaft by a soft, or even semi-fluid, greyish-red or yellowish material, consisting of granulation tissue which may shade off into pus. Another feature is the formation of new bone (osteophyte) round the outside of the shaft in the neighbourhood of the epiphysis.

Wegner terms this lesion a "syphilitic osteochondritis." It is widely diffused throughout the body, but it is said to be always most marked where the growth of osseous tissue is naturally most active; and what is interesting is that precisely the same distribution is stated to obtain in the case of rickets. Thus the lower end of the femur is the favourite seat of both diseases, while the corresponding part of the humerus is least often affected by either. Parrot, however, says that in syphilis the osteophyte is most abundant just where the other changes are least developed; and this corresponds exactly with a remark long ago made by Mr Hutchinson to the effect that periostitis in congenital syphilis is more often met with just above the elbow-joint than anywhere else.

In a few exceptional instances, when an infant infected with syphilis is born alive, the soft material between the shafts of the long bones and their epiphyses increases to such an extent as to detach them from one another, and even to form fluctuating purulent swellings beneath the periosteum. There is then a complete loss of power in the limbs, attended with so little pain that it has actually been mistaken for a paralysis. The child's hands are described by Parrot as lying pronated by its side; its legs are extended, and when it is lifted up they hang helpless and swing backwards and forwards. After death, almost every one of the principal epiphyses may be found separated from the bone to which it belongs.

Henceforth, there ought to be little difficulty in diagnosing cases of this severe kind, but it is otherwise with the ordinary form of the disease, in which there is seldom a sufficient degree of enlargement of the ends of the bones to justify one in speaking positively about it, at least in very young infants. Indeed, Köbner has shown that even after death the microscope is often required to reveal the presence of the lesion.

These lesions of congenital syphilis are to be carefully distinguished from those of Rickets, as will be explained in the chapter on that disease in the second volume of this work. (See also below, p. 335.)

Effects at birth.—As a rule, a child infected with syphilis does not manifest symptoms of the disease when it is first born.

One exception is, a peculiar and very rare form of bullous eruption, which

is known as *pemphigus neonatorum*, and which may either be present at the time of birth or come out a few days later. In 1851 there was a discussion upon this affection in the Academy of Medicine in Paris; Paul Dubois declared that it was syphilitic, while Cazeaux maintained the contrary. The question, so far as I know, has never been cleared up; but probably an examination of the bones in future cases would easily settle it. The bullæ are flaccid and contain opaque serum or pus; their favourite seats are the soles of the feet and the palms of the hands. I have never myself seen a case. This eruption generally proves rapidly fatal.

The *thymus* has been found in a state of suppuration by Dubois; there was no abscess, but the organ when squeezed emitted drops of yellowish pus, easily distinguishable from the opaline liquid which it may contain when in a normal state. Another morbid condition, first pointed out by Depaul, as occurring in the bodies of syphilitic children, consists in the presence of yellowish-grey indurated nodules in the *lungs*, softening in their centres into cavities. Lastly, Gubler has described a peculiar change in the *liver;* the organ is not only enlarged, hard, and elastic, but its cut surface shows a number of small, white, opaque grains on a uniform yellowish ground. Dr Wilks showed a specimen of this affection at a meeting of the Pathological Society in 1866; it came from an infant one month old. I once saw suppuration of the thymus in a child who had lived for some months. Acute peritonitis, or pleurisy, or meningitis has sometimes been found to be the cause of death. In cases of abortion from syphilis, the state of the skin may suggest the cause of the death of the fœtus.

Effects after birth.—The more common symptoms of congenital syphilis generally begin to show themselves towards the end of the first month of extra-uterine life or in the course of the second month, rarely after the end of the third month.

The earliest is, in most cases, a *nasal catarrh*, producing what is popularly called the "snuffles." This is attended with the discharge from the nostrils of a fluid, at first thin and serous, but afterwards viscid, so that it dries up into crusts which obstruct the passage of air. Consequently the child is no longer able to breathe while it is at the breast; it takes the nipple into the mouth only to drop it again, and, as Diday pointed out, it fails to get enough food, and rapidly loses flesh. Indeed, although syphilitic infants are sometimes at birth well-grown, plump, and fat, and may even remain so throughout the whole course of the disease, the rule is that they are from the first, or soon become, pale and wasted; they have a dull, opaque, yellow, and wrinkled skin; to use a favourite expression, they "look like little old men."

Soon after the catarrh there appears an *eruption*. Its favourite seats are the nates and the face, but it may cover the whole of the body and limbs. It consists of maculæ, blotches, or flat papules, which may be bright red, brownish, or copper-coloured. They are sometimes isolated, sometimes so confluent that they have been described as having an "erysipelatous" character. It is often difficult to distinguish between syphilis and the effects of the nurse's negligence in allowing the parts about the anus to remain soiled with excreta or in drying them insufficiently after having washed them. As a rule, the red blush produced by mere irritation is ill-defined and fades away at the edge, whereas at the corresponding part of a specific rash there are to be seen a number of isolated, sharply-outlined blotches. The papules themselves may have smooth

and glazed surfaces, or they may be dry and horny, or raised here and there into flaccid bullæ, or superficially ulcerated. At the corners of the eyes, about the angles of the mouth, in the folds of the neck or of the groins, or round the anus, the papules often become covered with a moist opaque layer, and assume more or less the appearance of flat condylomata. But I am inclined to agree with Mr Hutchinson that really typical "mucous patches," occurring chiefly at the anus, are comparatively seldom seen in children less than eight or ten months old. Sometimes the eruption takes a pustular form; the greater part of the body may then become covered with moist scabs, separated by cracks, from which a sero-purulent fluid is constantly oozing.

In the interior of the mouth ulcers are often present, or there may be a diffuse *stomatitis*. The latter affection involves the gums and dental sacs; Mr Hutchinson has even seen it lead to necrosis and exfoliation of the alveoli. The mucous membrane of the nose may likewise ulcerate, and discharge a sanious fluid, perhaps containing fragments of bone from the septum, which becomes perforated.

Another but a rare symptom is *iritis*. Of this Mr Hutchinson, some years ago, cited twenty-three cases, the majority of which occurred in girls. It was usually seen at about the age of five weeks. Sometimes it was limited to one eye, sometimes it affected both eyes. It was attended with irregularity of the pupil, alteration of colour in the iris, and the exudation of white, yellow, or red lymph. But it required to be carefully looked for, as the cornea was generally clear, and had but a very faint pink zone round its margin.

Transference from the child.—I have already remarked that although, in accordance with Colles's law, a child suffering under congenital syphilis is incapable of infecting its mother, it may give the disease to any other woman who suckles it. Diday even records cases in which women advanced in years appear to have contracted syphilis by kissing such infants; and the use of a spoon that had previously been employed in feeding them has been followed by the same terrible consequences. Probably the source of the virus is always the secretion of an ulcer about the lips or within the mouth. Hence Diday lays great stress on the importance of searching for such ulcers, when a child born of syphilitic parents is to be placed with a wet-nurse. But as it is practically impossible to be sure that the back of the throat is healthy, French writers advised that as soon as any cutaneous eruption, or even a nasal catarrh is discoverable, one should, for the nurse's sake, insist that she should no longer suckle such an infant. If one knew a wetnurse who had had syphilis she might be engaged for the service; but otherwise recourse must be had to the feeding-bottle. The primary lesion on the nipple of a nurse is described by Diday as a small red papule, slightly desquamating on the surface. When once infected, she in her turn may convey the disease to other infants. That the milk is not then the vehicle of the poison is shown by an observation of Mr Henry Lee's. A woman, who was nursing a syphilitic child with one breast, acquired an ulcer upon that breast, and afterwards an eruption; but her own infant, for whom she reserved the other breast, went on sucking for six weeks and remained healthy.

I do not find any evidence that syphilis contracted by an infant from a wet-nurse differs from the congenital form of the disease in its symptoms, as might be expected.

Treatment.—We have seen that congenital syphilis seldom proves directly fatal, except soon after birth. This, at least, is my decided opinion. In very mild cases the eruption often subsides in a few weeks when left entirely without treatment; and in the more severe forms of the disease specific medicines have immense power. I have generally been accustomed to prescribe the *hydrargyrum cum cretâ*, in doses of a grain, or two grains, two or three times a day, according to the age of the infant; a minute quantity of Dover's powder, or of carbonate of soda may be given with it, according to circumstances. Many prefer, however, to use the diluted *unguentum hydrargyri* externally; it may be applied on the inside of a flannel band which is sewn round the knee, as Sir Benjamin Brodie recommended; or ten or fifteen grains of it may be rubbed into the child's armpits once or twice daily for a few minutes at a time. Or the *liquor hydrargyri perchloridi* may be ordered in doses of ɱxx to ɱxxx three times a day. The effect of these various measures is generally to remove the visible signs of the disease in from one to three months. Sometimes relapses occur; and in such cases, or where the improvement is slow, it may be advisable, in addition to the mercury, to administer the *iodide of potassium* in doses of two or three grains. Mucous patches about the anus may be dusted over with calomel.

Later effects.—The subsidence of the usual symptoms of congenital syphilis is occasionally followed by the development of others, which are obviously analogous to those that constitute the "tertiary" stage of syphilis in the adult. Thus nodes may appear. Mr Hutchinson says that the bone most often affected by them is the humerus at its lower end, and that they often reach such a size as to impair the movements of the elbow-joint. I have seen extensive periosteal swellings upon the tibia and upon the ulna in a girl who must have been eight or ten years old; she originally came to Guy's Hospital on account of large ulcerating or rupial patches, covered with thick brown crusts, on her trunk and limbs. Under iodide of potassium they healed with marvellous rapidity, but she nevertheless kept returning to me for two or three years. Another form of eruption of which I have seen several instances in not very young children, consists of raised red rings or zones, somewhat like the "circinate squamous syphilide" of adults. At the Evelina Hospital I once saw a girl, eight or ten years old, with a sloughing ulcer which had destroyed the soft palate, and with great enlargement of the metacarpal bones of one hand. Mr Hutchinson cites the case of a boy, aged eight, almost the whole of whose calvaria was involved in disease at first regarded as "strumous," but which proved to be syphilitic; he was the son of a clergyman, but his mother had contracted the disease from a former husband, an officer in the army. I have once or twice met with nodular swellings, doubtless gummata, upon children's testes. With regard to affections of the cerebral arteries, or of the brain itself, I shall have something to say hereafter. Of lardaceous changes in the viscera, as the result of congenital syphilis, I at present know nothing.

The most interesting of the remote effects of the hereditary disease are, however, some which differ altogether from those of ordinary acquired syphilis. Mr Hutchinson has shown that syphilitic children often grow up with a special physiognomy. One peculiarity is that on each side of the forehead *the frontal eminence is protuberant.* This he formerly regarded as the result of antecedent hydrocephalus from chronic meningitis. But M. Parrot has recently maintained that in addition to the intra-uterine

osseous lesions already described, syphilis gives rise during later infancy and childhood, to a morbid change in the bones bearing a somewhat close relation to rickets; and Mr Hutchinson now accepts such a change as accounting for the appearance in question. A character on which Parrot has further insisted is the presence of four eminences upon the bones forming the sides of the anterior fontanelle; from their resemblance to the buttocks he calls such a calvaria "natiform.' He also claims for congenital syphilis the affection known as Craniotabes, which has hitherto been deemed rachitic. But it appears to me very doubtful whether Parrot's cases ought not to be regarded as examples of the accidental presence of rickets along with syphilis; and the more so, as he is disposed altogether to ignore the former affection as an independent malady. Further observations will no doubt soon settle this question.*

The remaining points originally detected by Mr Hutchinson are far more distinctive than the shape of the forehead. One is a broad, and *sunken bridge of the nose*, resulting from long-continued swelling of the parts within, or from loss of support by exfoliation of the septum. Another is the presence of radiating *linear cicatrices* at the angles of the mouth, running outwards towards the cheeks. A third is a thick, pasty, opaque condition of *the skin* generally, the hair being scanty, and the nails sometimes broken and disposed to split into layers. But most characteristic of all is a peculiar change in certain of the permanent teeth, and particularly in the *central upper incisors*, which for this purpose Mr Hutchinson terms the "test-teeth." Not only are they much smaller than usual, but they are "notched" and "pegged;" the former peculiarity consisting in a single deep crescentic excavation of their free edge, the latter in a gradual convergence of their sides towards this edge. It is supposed that this alteration in their form is the result of their having been disturbed in their growth by the stomatitis which is so marked a feature of the disease during infancy. When they first protrude from the gums, the notch is not present; in its place there is a row of minute projections; these soon afterwards break off. Similar changes may sometimes be observed in the case of the other incisors. It is important to note that the above single deep notch is alone indicative of syphilis. Horizontal grooves on the surface, and numerous small notches in the edge, are common enough in persons who are altogether free from any congenital taint.

The appearances just described enabled Mr Hutchinson to identify as syphilitic an affection of the eye, the real nature of which had not previously been suspected, and which was always known as "strumous corneitis." This disease, *interstitial keratitis* as it is now called, occurs chiefly in children between eight and fifteen years old, but sometimes in adults up to the age of twenty-five or twenty-six. Mr Hutchinson has only once seen it as the result of acquired syphilis. It is more common in girls than in boys. It seldom remains limited to one eye, but commonly attacks the two eyes in succession at an interval of a few weeks. It begins as a dotted haziness near the centre of the cornea, which spreads until almost the whole of it is densely opaque, like ground glass. The patient often complains but little of intolerance of light. There is not generally at first much congestion of the conjunctiva or of the sclerotic. Ulceration never occurs, but at a certain stage the cornea may become so vascular as to be uniformly pink or salmon-coloured. When the affection is at its worst, vision is often reduced to a bare perception of light. But in the course of a year or eighteen months

* See on this and other points Fourinu's 'La Syphilis héréditaire tardive,' 1886.

a surprising degree of improvement takes place. The opacity very slowly clears up, until perhaps only a few hazy patches remain; and the patient's vision may ultimately be but little impaired. Sometimes iritis occurs as a complication, and sometimes choroido-retinitis.

Another morbid condition which Mr Hutchinson has seen in the subjects of congenital syphilis is a bilateral *deafness*, dependent apparently on some disease of the deeper parts of the ears. It comes on at about the same age as does interstitial keratitis.

Two questions in regard to the after-effects of congenital syphilis still require to be briefly considered.

One is, whether a person who has passed through it in infancy is less liable than others to acquire the disease later on in life. Mr Hutchinson has recorded some instances in which venereal sores have been contracted under such circumstances, and one in which an outbreak of constitutional symptoms occurred; and his opinion is that the protection conferred by the fact of a patient's parents having had syphilis probably renders the acquired disease milder rather than prevents it altogether.

The other question is whether it is possible for the taint to be handed down to a third generation, so as to produce definite symptoms. In answer to this, Mr Hutchinson says that he has about eight times had opportunities of examining the children of persons undoubtedly the subjects of congenital syphilis. With one exception they appeared to be healthy. The exceptional case was that of the infant of a woman who came to him for interstitial keratitis, and who had also notched teeth and a characteristic physiognomy. She said that nothing ailed her baby; but on inspection it turned out to be covered with coppery blotches, and to have condylomata at the anus and snuffles in the nose. Mr Hutchinson had had the child's father under his care for a long time, and was convinced that he had never had any venereal disease. He was therefore inclined to believe that the virus had descended from the infant's maternal grandparents.

AGUE*

Intermittent Fevers—Incubation—The cold, hot, and sweating stages—Regular and irregular varieties—Remittent Fever—Malarial cachexia—The spleen, liver, and other organs in ague—Melanæmia—Nature and laws of malarious contagion—Diagnosis—Treatment.

We have now to discuss a disease which seems to stand apart from the specific fevers in some very important respects. In its most typical forms its course is interrupted by definite intervals of apyrexia, so that instead of appearing as a continuous fever it consists rather of a succession of independent paroxysms or "fits," which recur with marvellous regularity. For such cases the name of *intermittent fever* is commonly employed. But they must not be separated from others which are due to the same cause, and in which the subsidence of the pyrexia is only partial, so that they are said to be examples of *remittent fever*. And yet further varieties are met with, to which neither of these terms is applicable. I therefore prefer the homely word "Ague," as including them all. Another name sometimes used is that of *paludal* or *marsh fevers*; but it is better to avoid such expressions, for reasons which will appear when I have to speak of the ætiology.

The poison which excites ague is best called *malaria*, for this term can now be kept strictly to that meaning. It enters the human body from without; but, unlike the virus of most other specific diseases, it never passes directly from one person to another. In other words, ague, though in a broad sense one of the "infective" diseases, is not "contagious," nor even "infectious" in the narrow sense in which that epithet is generally employed.† Whether the poison multiplies in the blood, in such a way that the direct inoculation of the blood of a patient affected with ague upon a healthy individual would under certain conditions convey the disease, is a problem that has not, so far as I know, been hitherto solved.

* *Synonyms.*—Intermittent Fever, Periodic, Malarial, Miasmatic, or Paludal Fever.—*Fr.* Fièvre paludéene, *Germ.* Wechselfieber. The English word "Ague" is the French Fièvre aigue, *Febris acuta.*

† Vaccinia and Syphilis are contagious from person to person, *i. e.* inoculable by direct contact, but not infectious, *i. e.* not conveyed to persons at a distance. Typhus and Measles are contagious and infective, *i. e.* conveyed from person to person by direct contact and at a distance. Cholera is probably, and Enterica almost certainly, conveyed from person to person, but not at a distance through the air nor yet by direct contact, but only by conveyance of the virus or contagium further developed after its discharge from the body. Ague is not conveyed from person to person at all, but from locality, probably soil, to persons. According to the parasitic theory of specific diseases, proved in a few and more or less probable in several, and without any evidence but analogy in the rest (cf. *supra*, pp. 22–25), the microphyte of vaccinia and syphilis can only be transferred by direct inoculation into the blood or lymph; that of typhus, plague, or scarlatina can be carried by the air or on fomites and introduced through the stomata of the air-vesicles into the lymphatics by means of the inspired air; that of Enteric Fever is harmless when first discharged, but after certain stages of growth outside the body of the host can be introduced into the alimentary canal, and there reproduce like the ovum of a tapeworm or a trichina; and lastly, the microphyte of Ague is not solely parasitic, but lives and grows outside the body, and possibly, like many parasitic worms, can only complete its cycle of development when it is at one stage parasitic as well as at another stage free.

It is a curious circumstance that malaria appears to be almost without influence upon domestic animals, at least in those countries where its effects have been most studied. In Italy, however, a few instances have been recorded in which horses or oxen have had intermittent attacks of fever, or have suffered from cachexia attended with enlargement of the spleen, such as we shall see to be another result of the ague-poison.

Descriptions of the different varieties of ague are to be found in the writings of Celsus and other ancient writers, for various forms of intermittent fever were, and still are, the commonest disease in Greece and Italy. The first observer who took a clear view of malaria, as a noxious effluvium given off by marshes, appears to have been Lancisi, in a work published at Geneva in 1716.

Incubation.—That ague has a period of incubation is not always apparent in its history; for, as a rule, those who are attacked by it have resided for some little time in a region infested with malaria, and seem to succumb to the influence of repeated doses of the poison, instead of taking the disease on any particular occasion. But when a single exposure alone is known to have occurred, an interval of from six to twenty days is said generally to elapse before the paroxysms begin. Dr Maclean, however, in 'Reynolds' System,' mentions the case of three German missionaries, fresh from Europe, who passed a night in an uncleared and unhealthy spot, at the foot of the Segoor Pass in India; next morning they pursued their journey, but within less than twenty-four hours three out of four of the party were stricken with fever. And Hertz, of Amsterdam, writing on this subject in 'Ziemssen's Cyclopædia,' declares that on several occasions, having purposely placed himself in a marshy ditch at a time when it was drying up, he has been attacked within half an hour by giddiness, shivering, nausea, and other symptoms, which ended in a slight paroxysm of fever a few hours afterwards. On the other hand, it is well known that the onset of the disease may be much longer delayed. Sailors have fallen ill upon the open sea, weeks or months after leaving a port infected with malaria. In London the Irish poor often apply for hospital relief in the spring, on account of an ague which, although only just declared, must have been contracted during their autumnal migrations into Kent for hop-picking.

Premonitory symptoms.—Among strangers who have recently entered a malarial district, and also among persons who have left such a district before being attacked, the occurrence of a regular paroxysm is, according to Hertz, in most cases the earliest symptom of ague. He says that it is chiefly when the patient has been living for a long time in the same place, so as to have repeatedly run the risk of catching it, that a prodromal stage is to be observed. It lasts a week or more, and consists of vague malaise, fatigue, headache, and pains in the limbs, in association with nausea, fulness at the epigastrium, loss of appetite, and other symptoms of disordered digestion. There may also be occasional sensations of chilliness, alternating with slight flushes of heat.

The attack.—The actual ague-fit itself is divided into three periods, which have long been known as the "cold," the "hot," and the "sweating" stage respectively.

1. The *cold* stage begins with the patient's feeling tired, weak, and listless. He yawns and stretches out his limbs. He complains of an un-

comfortable sensation at the epigastrium, of headache, or of giddiness. Before long, these symptoms merge into those of a "shiver" or *rigor*. He experiences a chilly feeling along the spine, which quickly spreads all over him. He shudders, his teeth chatter, his knees knock together, his whole frame may be so violently agitated that the bed into which he has crept shakes beneath him. His voice is feeble and his speech is interrupted by the quivering of his lips. At the same time his appearance undergoes a remarkable change. He grows pale, his features shrink, his ears and nose and finger-tips turn livid, his skin becomes dry and rough—*cutis anserina* or "goose-skin." Yet the thermometer, as De Haen pointed out more than a century ago, shows that in the central parts of the body the temperature is not lowered, but raised several degrees above normal. It must not, however, be supposed that the patient's sensations deceive him as to the condition of the surface, and especially of the more distal regions. One has only to feel his nose or his fingers in order to assure oneself of the fact that they are really cold. And a surface-thermometer, or an ordinary thermometer held in the patient's hand, will remain far below the natural standard. But in the axilla, the mouth, or the rectum it shows marked pyrexia. The temperature begins to rise even before any symptom of the onset of the ague-fit is experienced by the patient. At first its increase is gradual, but with the development of the rigor it makes a sudden leap upwards, perhaps passing through four or five degrees Fahrenheit in the course of an hour. The patient during this stage feels dry and parched; his tongue is white; he often complains of nausea, and sometimes vomits. His pulse is quick, small, and sometimes irregular. His respiration is short, hurried, and distressed. His urine, although pale, is often very irritating, so that Dr Maclean has found it desirable to administer bicarbonate of potass and even tincture of opium for the relief of this symptom alone. Sir Thomas Watson spoke of the urine as scanty, although passed frequently; but the careful measurements and analyses of Redtenbacher and of Ringer have shown that it is in reality considerably increased in quantity, when compared with the amount passed each hour during the apyretic interval, and that there is also a marked augmentation of the urea and of the chloride of sodium. Indeed, the excess, both of the water and of these solid ingredients, not only lasts throughout the whole length of the fit, but begins before the patient feels chilly, and even a little before the commencement of the rise of temperature.

2. The *hot* stage gradually succeeds. The patient ceases to shiver, and begins to experience flushes of heat about the face and neck, which presently diffuse themselves over his body. But for a time if he attempts to throw off the bedclothes which have been piled up over him, slight rigors reappear. At length, however, even the extremities become permanently hot and glowing. His aspect is now again altered. His face is flushed, his eyes are injected, his skin becomes smooth and turgid. The temperature, as measured by the thermometer, still continues to rise for some time, and may now reach its maximum, which is generally 104°, but sometimes 106·5°, or even higher. The surface of his body, and even of the distal parts, now feels pungently hot to the hand. Evidently, there is not only an increased generation of heat, but it is now freely carried to the periphery, and abundantly dissipated. Accordingly, the pulse is not only rapid, but full and throbbing; and the breathing is deep as well as frequent. The patient complains more than ever of headache, which is now of a throbbing character; and he is exceedingly restless and uncomfortable. The urine during this

period is described by most writers as differing in appearance from that of the cold stage, being now high coloured and concentrated; and both Watson and Hertz speak of it as passed in but small quantity. There is often an eruption of herpetic vesicles upon the lips, or the nose, or the tongue.

3. The *sweating* stage follows in its turn. The surface gradually becomes soft; then a little moisture breaks out upon the forehead and the face, and before long the whole skin is bathed in the most copious perspiration, which saturates the patient's linen and bedclothes. It is said that the thermometer sometimes still continues to rise for a little while, so that the maximum temperature may occur during this, and not during the hot stage. But, as a rule, the pyrexia begins to decline from the moment when the skin ceases to be dry. The fall is at first slow, but after half an hour or an hour it becomes more rapid, and goes on until the normal temperature is reached. According to Wunderlich, this takes place by a series of steps, there being a drop of from one fifth to one third of a degree Fahr. about every fifteen to thirty minutes. All observers speak of the urine during this period as differing from that passed in the previous stages in showing a thick deposit of lithates. The breaking-out of perspiration is attended with complete relief to the patient; he loses his pains, the throbbing of his arteries ceases, he is no longer tormented with thirst, and his tongue beomes moist. After a time he perhaps falls asleep; and, when he wakes, he feels perfectly well, except that he may be somewhat exhausted.

Recurrence.—The subsidence of the ague-fit or paroxysm by no means brings the disease itself to an end. As we have already stated, its most remarkable feature is that precisely similar attacks are repeated again and again, recurring at perfectly definite intervals. Some patients have a fit every day; the ague is then said to be *quotidian.* In other cases it occurs every other day; and this form of intermittent fever is styled *tertian,* for according to the Latin way of counting it falls on every third day, or as we should say on every second day. If there are two entire days between the paroxysms the disease is a *quartan* ague. In other words, an interval of about twenty-four hours characterises a quotidian, one of forty-eight hours a tertian, and one of seventy-two hours a quartan. Nor does this exhaust all the possible varieties. In some cases in which there is an attack every day, each paroxysm differs in severity or duration or in the hour of its occurrence from that of the preceding day, and resembles exactly that which occurred two days before. Thus a tertian type is clearly perceptible, and the affection is then called a *double tertian.* So, again, it is possible to have a *double,* or even a *treble quartan* ague. Or a patient may have two ague-fits one day, and a single fit on the following day, this series being regularly repeated; and one may then suppose that a quotidian and a tertian are combined. Such a case constitutes the ἡμιτριτᾶιος of Celsus, or, as subsequent Latin writers have termed it, a *semitertian.*

The return of the paroxysms of ague is often so regular that it can be foretold with absolute certainty. But even then the hour at which the fits begin is not always the same on each occasion. Sometimes it is earlier, each time by a constant difference; sometimes it is later. In the former case, the disease is said to *anticipate;* in the latter case to *postpone.* And whereas the one is a sign that the severity of the case is on the increase, the other indicates that it is becoming milder. Such variations may even bring about a change of type; thus an anticipating tertian may at last pass into a quotidian, and a postponing quotidian may be gradually converted into a tertian. Some-

times the attacks of a quotidian fever are so prolonged that before the sweating stage of one fit is over the cold stage of the next one begins; the disease is then said to be *subintrant.*

With regard to the relative severity of the three principal forms of ague, writers state that quartans are peculiarly obstinate.* This variety, which is comparatively rare, is especially apt to occur in the autumn; and it has long been well known that autumnal agues are worse than those contracted at other seasons of the year.† On the other hand, it is said by Hertz that in the tropics the disease never assumes the quartan type; and yet ague is certainly in every other respect far more severe there than within the temperate zone. In hot countries the quotidian appears to be the most common form of ague, whereas in milder climates the tertian is more frequent.

There are some other differences in the course of the several varieties of intermittent fever. Thus the fits of a quotidian are said generally to set in during the morning, those of a tertian at about noon, those of a quartan still later in the day. It is stated that the average duration of a paroxysm of quotidian ague is from ten to twelve hours, that of a tertian from six to eight hours, that of a quartan from four to six hours, but that in the last-named variety the cold stage is more prolonged than in the others.

Subsequent course.—Ague, in its intermittent forms, appears to be never directly fatal. Its duration is very variable. Sometimes, even though the patient remains in the place where he acquired it, it ceases after a few paroxysms. In other cases they come to an end when a change of season reduces the malaria to a state of inactivity. In others, again, ague recurs for an indefinitely long period, until the victim is compelled to remove to some other district. There is always a strong tendency to relapse, not only under a fresh exposure to the ague poison, but even independently of it; and it is a very remarkable fact that, whether the type be tertian or quartan, the paroxysms of a relapse often occur on those very days on which they would have been due if the disease had gone on without break from the first. Graves has related an instance of quartan ague which he watched for twenty-seven months, and in which the periodic rate was maintained through thirteen out of sixteen intervals, some of which lasted more than two months. A similar observation had previously been made by Dr James Gregory in the case of a relative, who had marked on an almanac the days on which the fits of a tertian ague might be expected to recur, and who found that for a long while they did so whenever the East wind blew. Since the introduction of the thermometer into clinical practice, however, it has been shown that there is sometimes a distinct rise of temperature at the proper time for the paroxysm, although the patient himself may be altogether unconscious that anything is the matter with him. Ringer states that in spite of the absence of all febrile disturbance the periodicity of the disease may be indicated by the voiding of urine undue in quantity, and containing an excess of urea.

Varieties.—Ague-fits are not, indeed, always characterised by the whole series of phenomena described above. Sometimes the cold stage is absent, sometimes the sweating stage. It is said that sometimes the usual order is reversed, as in a case cited by Watson from Maugenet. The patient in

* Nevertheless the Latin couplet runs:

Pro febre quartana
Rara sonat campana.

† On the contrary:—"An Ague in the spring is Physick for a King." Yet James I died of a tertian fever in March.

question was always first attacked with profuse sweating; then he became dry and hot; finally he felt cold and had distinct rigors. As an unusual symptom may be mentioned the presence of albumen in the urine, accompanied by casts and even by blood; this has been observed by Griesinger and Hertz.

In certain exceptional cases the paroxysms differ *in toto* from those which are ordinarily seen, and they may be attended with great danger to the patient's life, so that Trousseau and Hertz both classify them apart under the name of *pernicious malarial fevers*. The most strange form of all is that which has been termed *syncopal*, in which there is a condition of suspended animation, so that the patient runs a great risk of being buried alive. Trousseau relates two instances of this. One occurred in a station-master on the Avignon Railway. He had been subject for some time to paroxysms of intermittent fever, and had repeated fainting fits; once he became pulseless, was supposed to be dead, and was carried to the mortuary. After some hours a servant happened to enter the place and found him groaning; he was therefore taken back to his bed, and under large doses of cinchona he regained his health. It is an extraordinary circumstance that Dr Chauffard, the physician who observed this case, also met with another one of the same kind. A man had fallen into a faint, was taken for dead, and had his face covered with a sheet; Dr Chauffard, however, detected slight movements of his heart, although the radial, axillary, and carotid arteries had ceased to beat; he immediately administered a quinine enema, and the man was saved.

Scarcely less remarkable is another variety which is attended with *coma*. Hertz says that instances of it are from time to time sent into the hospital under his care for apoplexy, and Graves placed on record a case in which he made this mistake. A gentleman awoke at about 4 a.m. with sensations of malaise, chilliness, nausea, and headache. After an hour he became extremely hot, the pain in the head was intense, and he passed from a drowsy condition into one of complete coma, with deep snoring, so that he "appeared to be labouring under a severe apoplectic fit." He seemed to derive much advantage from bleeding and other remedies, and in the evening he was perfectly well. The day but one after the same symptoms returned, and were removed by the same treatment, but when a third attack came on, Graves saw that it was an example of *tertiana soporosa*, and cut it short by large doses of quinine.

In other cases it is said that epileptiform convulsions or tetanic spasms have been present, or that the symptoms have simulated those of hydrophobia. It even seems that the paroxysms of ague may imitate various abdominal or thoracic diseases—cholera, dysentery, peritonitis, pneumonia, pleurisy, are among those enumerated by writers. Hertz tells us of a man who was attacked at two o'clock in the morning with shivering and pain in the left side; at eight there was a distinct pleural friction sound, but twenty-four hours later he was perfectly well. However, on the following night, at twelve o'clock, all the symptoms returned, and by nine the friction sound was more evident than before; at four in the afternoon he was sweating and free from pain; full doses of quinine were therefore given to him, and he had no further attacks. But it seems to me that it is impossible to be too cautious in accepting the doctrine that such a case is the mere result of malarial poisoning, for it is an established fact that in districts where ague prevails all kinds of diseases assume a more or less distinctly intermittent character.

Nor is it less difficult to determine the real nature of what have been termed *masked agues*, in which the paroxysms are stated to be of the most diverse descriptions, and attended with scarcely any febrile disturbance, or even with none at all. The most important of these is neuralgia, occurring especially in the region supplied by the first division of the fifth nerve (and thus often called "brow-ague"), but sometimes assuming all sorts of other forms. Not only have cases of sciatica and of other neuralgic affections of the limbs been attributed to ague, but the same view has been taken of painful affections of the mammary gland or of the testicle, and even of alarming seizures attributed to cardialgia or to neuralgia of the vagi; but, for my own part, I must confess that I do not know what evidence in favour of such interpretation of clinical facts could be taken as conclusive. Obviously, it is not enough to show that the attacks recur with a certain degree of regularity, and pass off under the influence of quinine, for as much as this may be said of almost every kind of neuralgia. The observance of a tertian or quartan type would no doubt be very significant, but Hertz says that masked agues are usually quotidian. It must be borne in mind that the more extensive a man's experience in regard to intermittent fevers, the more likely he is to be led astray by tradition, or by personal bias until he has recourse to the same convenient hypothesis for the solution of every obscure and indeterminate affection, such as occur frequently enough in clinical practice, even in districts where no ague is met with. What, for example, is to be said of the case of Dr Macmichael, cited by Sir Thomas Watson, who "caught an ague many years before his death by sleeping on a rock somewhere in Greece, and was ever after subject to occasional attacks of periodic headache and other aguish symptoms, for which he was obliged to have recourse to bark or arsenic"? And what value can be assigned to Trousseau's statement that there are cases in which insomnia, unaccompanied by fever and not preceded by rigors, recurs every two or three nights, that these cases are of the nature of masked ague, and that they are cured by similar treatment? This writer also mentions as manifestations of ague, spasmodic cough recurring in paroxysms at the same hour, and certain periodical mucous or sanguineous fluxes from the nasal fossæ, the uterus, or the intestines.

Remittent fever.—Malaria is not limited in its effects to the production of the different forms of intermittent fevers. In hot countries, and during the hot seasons of the year even in temperate climates, it causes fevers which are either continuous or at most interrupted only by *remissions*, intervals in which, instead of there being a complete apyrexia, there is only a partial lowering of the temperature and of the pulse, with some degrees of abatement of the other symptoms. For cases of this kind the technical name is *remittent fever*, but in India a common term for them is *jungle fever*, and another is *bilious remittent fever*. They are, in fact, characterised by a series of phenomena which do not belong to the milder effects of the marsh poison, as well as by the absence of some of the more distinctive features of ordinary ague. Thus the cold stage is slight and ill defined, and the hot stage is followed by little or no sweating. The remissions themselves, which often begin about midnight or in the morning, and which last for twelve hours or longer, may be so slight as to require careful clinical observation for their detection. A feeling of oppression at the epigastrium is present from the commencement of the patient's illness, and vomiting is usually a marked

symptom throughout its course. The vomited matters may at first consist of food, but afterwards a watery fluid is ejected, often in surprising quantity, and it may ultimately become greenish yellow, brown, or even in rare cases black. Headache is generally a prominent symptom, but not delirium.

In the worst cases, in which the disease is said to be of an "adynamic" character, the patient rapidly becomes very prostrate and insensible, his skin is yellowish and covered with petechiæ, his tongue is black, his teeth are covered with sordes, and hæmorrhages may occur from the nose, the mouth, and the limbs; or hæmaturia may be a prominent symptom. French writers have described such cases under the title *fièvre bilieuse hématurique*. Maclean, from whose description of remittent fever in 'Reynolds' System' I have taken most of the preceding details, says that except in such cases the urine seldom contains albumen; it is often abundant, and sometimes contains but little pigment.

The duration of remittent fever is said to be generally from five to fourteen days. Even its worst forms ought, according to Maclean, to do well in most cases, if seen early and skilfully treated. Sometimes it ends with a critical perspiration, sometimes it subsides gradually, sometimes it passes into one of the regular types of intermittent fever. Death seldom occurs before the seventh or the eighth day, a point which may distinguish this disease from yellow fever in countries where they both prevail.

Malarial cachexia.—In persons who have suffered from protracted ague, or who have had repeated relapses, a chronic condition of ill-health is apt to develop itself which is known as malarial cachexia. And a precisely similar condition is not infrequently seen in those who have lived for a long time where the disease is prevalent, even though they may have never had any febrile symptoms. One of its most characteristic signs is a peculiar earthy sallow complexion; another is a damp, clammy state of the skin, especially noticeable in the hands. The patient is depressed in spirits, and wakes unrefreshed by the night's sleep; he often suffers from giddiness, noises in the ears, tingling in the hands and feet, pains in the lower part of the back, palpitation, loss of appetite, a furred tongue, and digestive disorders.

In all cases of this kind enlargement of the *spleen* can be made out on percussion. Very often the organ is to be felt beneath the left costal cartilages, extending down to the umbilicus or even lower. Sometimes it forms a conspicuous tumour, which used to be called an "ague-cake." Although in some very rapidly fatal forms of malarial fever the spleen has been found of unusual size, the rule appears to be that it swells with every paroxysm of the disease, and subsides more or less completely during the intervals. It is generally supposed that the yielding capsule of this viscus allows it to become the receptacle of an undue proportion of the blood which is driven inwards from the surface of the body in the cold stage. Nor do I think that Hertz is right in rejecting this hypothesis altogether, on the mere ground that a malarial cachexia in which there have been no febrile attacks is also attended with splenic enlargement.

As might be expected, the state of the spleen is very different under different circumstances. In acute cases it is soft or diffluent. It may rupture from the slightest accident, and pour a quantity of blood into the peritoneal cavity so as to bring the case directly to a fatal issue. Hertz states that infarcts are sometimes formed in it, which may lead to suppuration, or to gangrene, followed by intense peritonitis. On the

other hand, in cases of chronic ague or of malarial cachexia, the spleen becomes extremely indurated. Not only is the hardness of it discoverable by palpation during life, but after death it is found to have little or no pulp, while the fibrous elements of its substance have undergone an immense increase. Its capsule is often very much thickened, and it may be fixed by adhesions to adjacent parts.

In cases of death in malarious cachexia the *liver* is often found enlarged and indurated, and there appears to be some doubt whether it may not undergo a change identical with cirrhosis. Clinically this condition is further indicated by the presence of pain and tenderness in the hypochondriac regions, and by the occurrence of slight ascites with which œdema of the legs may be associated. German writers commonly mention lardaceous changes in the abdominal viscera among the results of chronic poisoning by malaria; but the only authority that I have found for such statements is the Swedish physician, Key, who in 1862 described a form of renal disease, consisting mainly in lardaceous degeneration, as a sequela of ague.

Maclean cites Parkes as having often observed chronic Bright's disease in those who had suffered from ague, and confirms the statement from his own experience. But can one be sure that sufficient care was taken to eliminate other possible causes?

There is, however, another affection which results from severe or protracted ague, and which is altogether peculiar to malarial cachexia. It is called *melanæmia*, and consists in the presence of a brown or black pigment, partly free, partly enclosed in leucocytes, which is found not only circulating in the blood, but also lying in the substance of the solid tissues. The pigment in question is no doubt derived from the colouring matter of the blood. Klebs states that it yields Prussian blue when tested with ferrocyanide of potassium, so that it must contain iron which no longer forms part of an organic compound. It occurs either in minute granules or in larger masses, irregular in shape, and sometimes semi-crystalline in appearance. Where there is a large quantity of it, the organs are conspicuously discoloured. Thus the liver and the spleen and the kidneys assume a slaty-grey colour, the medulla of bones becomes chocolate-brown, and the cortex of the brain may appear as if rubbed over with black lead.

A most striking example of pigmentation of the brain, no doubt malarial in origin, occurred at Guy's Hospital in 1829 in the practice of Dr Bright, who figured it in his 'Medical Reports.' The case was that of a man who, with his wife, died of severe fever immediately after they had walked up to London from Horncastle in Lincolnshire. I do not know of any other similar instance as having been observed in this country. In Germany, too, melanæmia with abundant pigmentation of the organs appears to be very rare; all the well-known cases recorded by Frerichs belonged to an epidemic of ague which arose in Silesia after an inundation in 1854. But more than one observer has stated that he has constantly been able to detect pigment in the blood, by microscopical examination, in cases of "pernicious" ague, or of malarial cachexia. In 1877 Dr Stephen Mackenzie made a similar observation several times in the case of a man who had brought ague with him from India. The red discs were normal; but many leucocytes contained granules which were arranged round their nuclei, or sometimes completely filled them so as to give them a uniform brown or black colour. After treatment with quinine, the melanæmia was no longer discoverable. The blood of the portal vein has been said to be specially affected with this

change ; Dr Hammond has even punctured the spleen during life, and drawn off a few drops of blood from it for examination. The pigment is generally supposed to be originally formed mainly in the spleen, and to pass from this organ into the blood, so as to be carried all over the body. It often blocks up the capillaries, but it is also found in the walls of larger blood-vessels, and even outside them in their sheaths. These facts do not prove that it may not have come from elsewhere, for wandering leucocytes may have carried it with them. But many pathologists think that, in some cases at least, it is developed *in situ* from extravasated red corpuscles. Whether melanæmia directly gives rise to any symptoms appears to be doubtful. Dr Mackenzie's patient had hæmorrhages into each retina, and the same thing has been observed in some other cases of ague. It is natural to suggest that they might have resulted from plugging of minute vessels with pigment, but the anæmia which is so commonly present in such patients would equally account for them. In 1880 a man who had caught ague in the Black Sea was in Guy's Hospital under my care ; he had a large hæmorrhage in one retina ; but no pigment could be found in the blood. Capillary hæmorrhages in the brain have, however, been attributed to pigmentary embolism of this organ ; and a variety of cerebral symptoms have been supposed to result. But Hertz asserts that there is, in individual cases, no close correspondence between the occurrence of such symptoms and the demonstrable presence of the pigment in the minute vessels of the brain. Perhaps the most interesting question of all is whether there may not be something in common between melanæmia and that remarkable affection, paroxysmal hæmatinuria.

Ætiology.—We have now to consider what is the nature of the *malaria* or exciting cause of ague, under what conditions it is developed, and how it gains access to the human body.

That the disease is especially apt to prevail in marshy districts is universally admitted, and the fact could not possibly be disputed. Yet it is no less certain that something more is needed than the mere saturation of the soil with water. For countries like Ireland, which are widely covered with peat bogs, do not breed malaria abundantly. Nor, according to Parkes, are salt marshes infested with ague, when they are regularly overflowed by the tide ; and this, notwithstanding that for a large part of each day their surface is exposed, which (as we shall presently see) might favour the development of malaria. Again, the most virulent forms of ague have sometimes been met in districts that appeared to be perfectly dry and arid. Such, according to Hirsch, are the table-land of Castile, the plain of the Araxes, and the lofty plateaus of Northern India and Persia, all of which are highly aguish ; he even says that careful inquiry has proved that the malarial regions of Italy are in large parts of their extent devoid of water and sterile. Again, instances in which British troops have been attacked with the disease while encamped upon dry sandy soils, both in Holland and in Spain, are cited by Watson from observations made by Dr William Ferguson nearly a century ago. And Maclean lays stress upon the development of malaria in Hong Kong and in other places, situated upon granite rocks which are undergoing disintegration. These exceptional cases are fatal to a hypothesis which at one time was generally upheld, namely, that the poison of ague is nothing more than a product of putrefying vegetable matters. But there is no real difficulty in bringing them into harmony with cases in which the disease is developed in marshes, or along the estuaries of rivers. For accurate

investigations have shown that even there the malaria is developed, not in the wet part of the year, when the ground is entirely flooded with water, but rather during those seasons at which large parts of it are exposed to the air, and become more or less dry. And as Maclean has pointed out, it is tolerably certain that, in every instance supposed to be of an opposite kind, water might very easily have been present, either in the form of subterranean streams, or upheld by a bed of clay or other impermeable material, or only saturating the soil up to a certain level. So, again, it has been noticed that in Italy an occasional overflowing of fresh-water marshes by the sea has been followed by a great development of malaria; but this is not inconsistent with the fact that marshes which are always saturated with salt water are healthy, since the conditions in the two cases are clearly different. One way in which a hot sun probably favours the development of ague is by cracking the dry surface of the ground and allowing emanations to escape from the moist layers beneath. And so, again, it has repeatedly been found that the disease has reappeared in places that had long been free from it when the soil has been extensively disturbed for building purposes, or during the construction of canals or of fortifications. Conversely, much can be done towards preventing the exhalation of malaria by draining the ground thoroughly, and by then spreading a layer of fresh soil over its surface, or, in the case of inhabited villages or towns, by paving it. Of course, many districts are decidedly damp without being aguish. But this merely proves that other conditions besides a wet state of the soil are necessary for the production of malaria.

At certain times intermittent fevers spread to regions which lie far beyond their usual limits, and assume somewhat of a *pandemic* character. The years 1558, 1678–79, 1718–22, 1807–12, 1824–27, 1845–48 are mentioned by Hertz as having been characterised by such a wide diffusion of ague. Watson says that he never knew ague at all widely prevalent in London except in 1827. From 1866 to 1868 there was an epidemic in Mauritius, which island had previously been so free from malaria that the sufferers from Indian fevers had been accustomed to resort there. And in 1869 it broke out for the first time in Réunion.

Climatic conditions.—With regard to the nature of the various conditions which favour the development of the ague-poison, we at present know very little. It is a curious circumstance that there has often been an antecedent or simultaneous epidemic of influenza or of some contagious disease such as typhus, cholera, or the plague. One very important feature is undoubtedly a high temperature. Ague prevails with far greater intensity in the tropics than in temperate climates, while towards the poles it is unknown, its boundaries being about 63° N. and 57° S. of the equator. Hirsch has endeavoured, upon very defective data, to determine the corresponding limits of temperature, and finds that mean summer temperatures rather than mean annual temperatures must be taken into account. In this hemisphere the highest range of malaria toward the North is fixed by him at a line between the isothermal lines of 60° and 61° Fahr. Again, in particular districts, the occurrence of malarial fevers is governed in a very marked manner by variations of temperature. During the winter they disappear entirely; and, other things being equal, they are always more severe in proportion as the season happens to be a hot one. In the tropics the exact period of the year at which ague is most prevalent varies in different localities; generally speaking, it closely follows the rainy season. Hertz lays down the rule that, in order to escape malaria, one should arrive in the East Indies between November

and January, in the West Indies between January and March. In temperate climates malaria is usually more active in the spring and in the autumn than in the height of the summer. Wenzel made the important observation that among the men employed upon the works at Wilhelmshafen the development of ague followed the summer-heat by an interval of twenty to twenty-five days, of which from six to eleven days appeared to be occupied in the generation of the poison in the soil, while the remaining fourteen days correspond with the period of its incubation within the bodies of those infected. The liability to ague is greater near the sea-level than in cooler places higher up; but Parkes mentions some instances in which marshes at elevations of five thousand to six thousand feet have, in hot countries, been found to give off malaria. It is, of course, quite a different question to determine what is the lowest point on the hills above a marsh at which one may reasonably expect immunity from the disease. The fact is well ascertained that the wind sometimes carries the ague-poison to considerable distances from its source, not only on level ground, but also up the slope of a range of mountains. Parkes says that in temperate climates an elevation of at least five hundred feet above a malarial spot should be reached in order to escape its influence; in the tropics, one of one thousand to fifteen hundred feet, or even higher still. On the other hand, at the level of a marsh itself, the injurious emanations seem to have but little tendency to rise and diffuse themselves in the air. There is evidence that, both in barracks and in private houses, persons sleeping on the ground floor are more apt to be attacked than those who occupy upper stories. It is at night, and for a short time after sunrise, that malaria is especially to be feared; probably the poison is harboured by the stagnant mists which are so apt to lie over the surface of damp ground, until the sun disperses them. The presence of actively-growing vegetation appears to be adverse to the development of malaria. There is also reason to believe that the inhabitants of houses in the neighbourhood of a marsh are sometimes protected by the interposition of a belt of trees. The poison also seems to be incapable of being carried across a surface of water without undergoing absorption. When English troops occupied Walcheren and other parts of Holland, it was several times noticed that only those soldiers who disembarked were attacked by ague; those who remained on board ship, even in narrow channels, escaped.

Chorography.—I have already incidentally mentioned many of the countries in which ague prevails. In England its chief seats are along the eastern coast; Romney Marsh in Kent, the estuary of the Thames in Kent and Essex, the fens of Cambridgeshire and Lincolnshire, and the marshy lands of the East Riding in Yorkshire. In all these districts the disease has become far less frequent than formerly, since population has increased and improved drainage has brought more and more land under cultivation. Not many years ago cases were common in London. James I and Oliver Cromwell* died of ague contracted in this city. At present its occurrence is altogether exceptional, even in the low-lying parts of Southwark. In Scotland I believe that there is now but little malaria; Sir Robert Christison, I think, stated that it had begun to disappear before any drainage works were carried out. On the continent of Europe the regions in which the ague-poison is most prevalent are the

* 'The Court and Character of K. James,' by Sir A[nthony] W[eldon], 1651, p. 160. Dr Bates gives the following account of the spleen at the autopsy of the Protector:—"In naturalibus fons mali comparuit; liene, licet ad conspectum sano, intus tamen tabo instar amurcæ referto." Elenchus motuum nuperorum in Anglia, 1662, p. 417.

following: the great plain of North Germany, with some of the Baltic provinces of Russia; Holland, with the adjacent parts of Germany and Belgium; the south-western departments of France; the western side of Spain and Portugal; almost the whole western side of Italy; a great part of Greece and Turkey; the plains of Hungary; and the shores of the Black and of the Caspian Seas. Nowhere is it so severe as in Italy, especially in the Maremma of Tuscany, the Campagna of Rome, and the Pontine Marshes. And what is of especial interest is that in these districts it has greatly increased in intensity within the last four centuries. Places which were at one time thickly populated and well cultivated have become waste and deserted; and coincidently with these changes they have grown in the highest degree unhealthy.

In Asia, malarial fevers abound not only in many parts of India, but also in Persia, and in China. In Africa the west coast is but too well known for the malignant character of the agues which prevail there; and they are also of frequent occurrence in Algeria and in Egypt, as well as in Mozambique and Zanguebar. On the American continent, the States which surround the Gulf of Mexico are those in which the effects of malaria are most frequent and severe; they are also seen in parts of Brazil and of Peru, as well as in many of the West Indian islands. It is a remarkable fact that in Australia ague is everywhere of a very mild type, if it prevails at all; it is said to be altogether absent from New Zealand and from Tasmania.

Infection by water.—Hitherto I have assumed that malaria enters the body with the air which is breathed; and no doubt this is usually the case. But there is good evidence that the poison is sometimes introduced in drinking-water. The most striking case of all seems to be that recorded by M. Boudin, of the one hundred and twenty soldiers conveyed by the French transport ship "Argo" from Algiers to Marseilles in 1834. In the hurry of embarcation, the water which was to be supplied to these men was taken from a marshy place near Bona. Thereupon, all but nine of them became attacked with various forms of ague; and it turned out that these nine, instead of drinking the same water, had purchased wholesome water from the crew of the vessel, all of whom remained well. In two more ships, which made the voyage at the same time, there were six hundred and eighty other soldiers, and they too escaped. Another instance is that of a farmer's family at Houghton near Bedford, who drank well-water, and who were at one time almost the only persons free from ague in the parish, the other inhabitants of which had only ditch-water. In another village close by, it was noticed that the digging of wells, by which a supply of fresh water was yielded, was followed by a great diminution of the disease. And it seems not unlikely that to the same cause may be attributed whatever part of the decline of ague during the last few years, both in England and in Scotland, cannot be accounted for by the improved condition of the soil. I doubt, however, whether statements with regard to the prevalence of malaria in former times can be universally accepted, since it is certain that until recently its effects were frequently confounded with those of enteric fever.

Predisposition.—Of the conditions that may render one person more susceptible to malaria than another we at present know but little. That more cases of ague are seen in men than in women or in children is doubtless due to the circumstance that men are more exposed to its exciting cause. A similar explanation may be found for the fact, if it be a fact, that during pregnancy women possess a certain degree of immunity. Sir Thomas Watson, however, cites an instance in which a child *in utero* had tertian ague with

its mother, who herself shook one day, and felt the child shake within her on the following day. And Hertz speaks of infants born with enlarged spleen, under similar circumstances.

It seems to be well ascertained that negroes are proof against ague; so that black soldiers are invaluable for field service in certain parts of the West Indies. Even if this fact be due to a kind of acclimatisation, transmitted by inheritance, it is not the less remarkable. For, although ague has sometimes been noticed chiefly to attack persons who had recently arrived in a malarious district, yet those who have lived there for a longer time have commonly suffered in other ways, being, in Watson's words, "puny, sallow, and sickly." The negro, on the contrary, is said to enjoy life and health in such regions. It seems to be certain that over-exertion and fatigue render a man more susceptible to the action of malaria; and that those who are convalescent from other diseases, or recovering from the effects of wounds, are particularly apt to suffer. It would be worth while to investigate whether persons whose general state of circulation is such that they are prone to the disease known as paroxysmal hæmatinuria are not more sensitive than others to the ague-poison.

Contagium vivum.—It appears to me impossible to ponder over the various conditions which favour or modify the development of malaria, without coming to the conclusion that no hypothesis accords so well with the facts as that which assumes it to be a living organism. In 1866 an American physician, Dr Salisbury, declared it to be an alga of the genus *Palmella*, which he had found in marshy soil in Ohio. But he did not attempt to show that the spores of this microscopic plant were really capable of generating the disease; and the same alga has since been discovered in regions where no ague exists, and even in the Alps. Nor are any direct proofs brought forward by other observers in Italy and elsewhere, who afterwards took up the subject, and by each of whom some different species was assumed to be the essential agent in propagating the disease. It was, therefore, a step in advance when in the spring of 1879 Prof. Klebs, of Prague, and Prof. Tommasi-Crudeli, of Rome, carried out together a series of experimental investigations which they believe have determined not only that what constitutes malaria is a particular kind of bacillus, but also that a fever of intermittent type can be generated in rabbits by subcutaneous injection of liquids containing this organism. They constructed a machine by means of which a large quantity of air could be rapidly made to pass over the surfaces of glass slides moistened with glycerine jelly. This they set to work in the Pontine Marshes and in Rome, taking the air always from very near the surface of the ground. Direct microscopical examination of some of the glass slides afterwards revealed the presence of rod-shaped bodies and delicate threads; and by suitable methods of cultivation these were made to undergo further development into jointed filaments, with spores in their interior. Similar structures were found in mud taken from the borders of lakes in the same region, but not in the waters of the lakes themselves. I must confess, however, that after reading carefully the details of the experiments in question, and studying the temperature charts which Klebs and his Italian coadjutor give, I fail to see that there is anything definite or characteristic about the fever which occurred in their animals after injection of fluids containing the so-called "*Bacillus malariæ*." Further points on which these observers lay stress are that the spleen, without being softened, was constantly found enlarged after the rabbits were killed, and that this

organ often contained black pigment, analogous with that which is seen after severe ague in man. They also detected the bacillus both in the spleen and in the medullary tissue of the bones of the animals. Finally, they cite Dr Marchiafava as having succeeded in discovering the spores and jointed filaments of the same parasite in the same structures, and in the blood of two persons who had succumbed to pernicious malarial fever in Rome.*

It is, I think, a significant fact that the other disease in which the presence of a peculiar organism in the blood has been most clearly traced,—relapsing fever,—should itself be characterised by a definite interruption in its pyrexia, which may fairly be taken as bearing some relation to the intervals between the successive paroxysms of an ague. And the analogy tempts one to hazard the hypothesis that during each paroxysm the bacilli, if they really constitute the malarial poison, are in great part destroyed, and that their reaccumulation is in some way the cause of the next paroxysm. But, if so, it is difficult to understand how the periodicity can be maintained when no well-marked ague-fits occur. With regard to the succession of phenomena that constitute the typical paroxysm, I do not think any special explanation is needed. The same order of events, more or less modified in details, is seen in the rigors of pyæmia and under a variety of other conditions. And there can be no doubt that, like an epileptic seizure, the ague-fit rests upon a physiological basis. Its recurrence at the same hour on successive days, or every third or fourth day, is doubtless, as Cullen originally suggested, connected with the diurnal habit of body which is so plainly manifested in other ways in both health and disease. This is not merely acquired, but has probably been transmitted by long inheritance to every existing member of the human race. One therefore need not wonder, if, as is stated by Hertz, Griesinger and Duchek failed to modify the times at which ague-fits returned by changing the patient's hours of meals and his other conditions of life.

The Italian physicians, Crudeli, Marchiafava, and Celli had described and figured remarkable pigment granules in the blood-corpuscles of malarial cases.† Laveran and Richard discovered pigmented amœboid bodies within the blood-discs and flagellate organisms ('Comptes-rendus' for 1882). Dr Osler, of Philadelphia, confirms these observations, and in addition finds, outside the blood-discs, crescentic bodies containing granules, and pigmented corpuscles furnished with one, two, or more flagella in active motion.‡

* Mr North has since with great perseverance and devotion followed the investigation of malaria at Rome, and his results differ in many particulars from those of Klebs and Crudeli.

† See Professor Crudeli's paper, with drawings of blood-discs after Marchiafava and Celli, in the 'Report of the International Congress at Copenhagen,' vol. ii, p. 23.

‡ "The Hæmatozoa of Malaria," with figs., 'Brit. Med. Journal,' March 12th, 1887. Independently of these observations, flagellate organisms have been described in the blood of mules in India suffering from a serious disease known in the Punjab as "Surra," by Dr Griffith Evans and Mr Steel (in Burma), and also by the late Dr Timothy Lewis in Indian rats ('Quart. Journ. Micr. Sci.,' 1879 and 1884). Dr Crookshank has found the same organism in the blood of rats in England ('Journal Roy. Micr. Soc.,' 1886). At present it is quite uncertain whether these, apparently polymorphic, bodies are connected with malaria as cause or effect, or whether they are physiological, as Dr Lewis believed them to be in rats. The Indian species were *Mus decumanus* and *M. rufescens*, the organisms in their blood (named Herpetomonas by Saville Kent in his 'Manual of the Infusoria') have been compared to the *Spirillum Obermeieri*, to spermatozoa, to Gaule's 'Cytozoa' in frogs' blood, previously described by Lankester (*Drepanidium ranarum*), and to stages of Gregarinidæ or other forms of Leuckart's Sporozoa.

Diagnosis.—The recognition of ague is for the most part very easy, but it may present difficulties which have to be considered from two points of view, according as the disease is or is not known to be prevalent in a particular district. In places which are free from malaria, there is sometimes no little risk of the real nature of a patient's illness being overlooked, when it has been acquired elsewhere during a visit, or while he was travelling in a country where the existence of the poison was perhaps unknown to him. Again, it is an extremely difficult point to settle whether or not some of the milder and less characteristic effects of malaria may still continue to appear from time to time in places (such, for instance, as the Borough of Southwark) in which the regular forms of ague have long been extinguished. Many very experienced physicians have undoubtedly held this belief; but I cannot determine how far their judgment was biassed by the fact that the affections in question are curable by quinine and the other remedies for ague, a fact which certainly is in itself of no decisive significance.

On the other hand, in regions notoriously infested with malaria, the physician has constantly to keep in mind not only that ague is apt to complicate all other diseases, but that it is capable of simulating an immense variety of them, as I have already pointed out (p. 343). Further there is always danger of his carelessly mistaking for results of malaria other affections which are attended with recurrent rigors or with paroxysms of febrile disturbance. Thus Graves relates an instance in which the intermittent hectic of *phthisis* was set down as ague, and some years ago I saw the very same error committed. Another disease which is very apt to be overlooked under such circumstances is *pyæmia*. In reference to this, Hertz remarks that the pyæmic rigor generally sets in more gradually than that of ague; that its subsidence is more rapid than its onset, whereas in ague the reverse is the case; and, lastly, that in pyæmia there is very seldom an interval of complete apyrexia, continuing for twenty-four or even for twelve hours. But, as we have seen, ague itself may be merely remittent. The fever attending *internal suppuration* may also be attributed to malaria. Not long ago I saw a case in which a medical man was firmly convinced of the correctness of such a diagnosis, until his patient voided a quantity of horribly fœtid pus with his urine, after which a rapid recovery took place. Some years ago, a gentleman who had stricture was placed under my care by Mr Cock, on account of the recurrence, at tolerably definite intervals, of rigors which seemed to be due to the formation of a small abscess in the neighbourhood of the urethra. Ulcerative *endocarditis*, too, must be thought of.

Finally, there are the continuous forms of ague, with which other diseases, and especially *enteric fever*, have often been confounded. Thus I happen to know that in Romney Marsh no kind of fever used to be recognised except that due to the endemic malaria; and in many parts of India it is only quite recently that enteric fever has been recognised. The *fièvre bilieuse hématurique* is not always easily distinguished from yellow fever in countries where both diseases prevail.

Brass-founders' "ague."—This is perhaps the most convenient place for brief mention of a complaint to which brass-founders are liable. It was studied by Dr Greenhow in Birmingham in 1858 and named by him "brass-founders' ague" on account of the resemblance between its symptoms and those of a paroxysm of intermittent fever ('Med.-Chir. Trans.,' vol. xlv, p. 177). It is in no way dependent upon malaria, but is caused by the dense white

fumes of oxide of zinc, the result of deflagration of the metal, which, at a certain stage in the formation of brass, fill the casting shop, unless it is very well ventilated. Exposure to these fumes is followed by malaise and weariness, by a sense of constriction at the chest, and sometimes by nausea. Afterwards shivering comes on, and even chattering of the teeth; and this is succeeded by a more or less marked hot stage, and ends in very profuse sweating. Next day the man feels unwell, but not enough to be unable to work. There is no regularity in the recurrence of the attacks, which come on from time to time under the operation of their exciting cause, but especially when the patient has been off work for a few days. They seem not to impair the health, and it is not known that they shorten the life of the patient. Drinking milk is said to be preventive of the brass-founders' disease.

Treatment.—With regard to the course taken by the various forms of ague if left to themselves, I have nothing to add to what has already been stated. But the *prognosis* in this disease is not based directly upon its natural course, for it always is, and always should be, submitted to treatment as soon as it is recognised. Indeed, we have so potent an antidote to malaria in cinchona-bark, and in the Quinine which is derived from it, that writers have sometimes been disposed to class this remedy apart from all others, as the most signal instance of a specific. And I imagine that it really does occupy a unique position in one respect, namely, as being the only medicine of which the efficacy has never, in our day, been challenged by anyone, however rash and inexperienced. The bark itself is not now prescribed on account of its causing nausea and sickness in the large doses required; quinine or one of the other alkaloids is substituted for it. Even as a preventive of ague, quinine has been found very efficacious for sailors exposed to malaria when sent ashore for a day, for travellers passing through, or for soldiers bivouacking in, a marshy district. From two to five grains are given twice or three times a day. It is less desirable to place the permanent residents in an aguish region constantly under the influence of quinine, since the organism appears to become somewhat blunted to its action in course of time; but they may at least take it at those seasons during which the disease is most prevalent.

With regard to the best method of administering the remedy in the intermittent forms of ague there has been much difference of opinion. There is reason to believe that the paroxysm itself cannot be modified by quinine taken after its commencement, except perhaps when it is injected subcutaneously. Probably, therefore, if one is called to a patient in an ague-fit the best plan is to prescribe a full dose of some ten or fifteen grains, as soon as possible after its cessation, repeating the same dose more or less frequently during the interval, until an hour or so before the next fit is expected. Maclean suggests, as a rule of practice, that the patient should never have less than thirty grains during this period. It is, he says, best given in solution with a little sulphuric acid and tincture of orange-peel. As is well known, pills containing quinine, besides being clumsy, are exceedingly apt to become hardened by time so as to pass through the stomach undissolved. If vomiting should occur, the alkaloid may be administered per rectum in a dose of fifteen grains suspended in beef-tea or thin starch.

When the stomach is loaded an emetic is often a useful adjuvant, and if there is constipation a purgative; but it was altogether a mistaken practice

which formerly prevailed of putting off the use of quinine until the tongue had been made clean by other treatment and the digestive organs regulated.

For the actual paroxysm of ague but little treatment is needed. The patient should go to bed and have plenty of blankets, and hot bottles or hot bricks wrapped in flannel may be applied to his limbs. Baths are better avoided, and he should not be encouraged to drink warm fluids in any large quantity. Frictions with stimulating liniments, or with a mixture of soap liniment and laudanum, have been recommended. It is only when threatening symptoms of collapse are present that recourse should be had to coffee, wine, ammonia, and ether. As the hot stage comes on most of the bedclothes may be removed, the patient may have cooling drinks if he wishes it, or he may suck a little ice and be sponged with vinegar and water. At the end of the sweating stage he should be carefully dried and have a change of linen. He may then get up, but if he should afterwards go out of doors he must be very careful not to take cold.

It very rarely happens in recent cases of ague where quinine is given in the manner indicated above, that the patient has any subsequent attack of the same degree of severity as before; and within a few days, or in a week or two at latest, they almost always cease. But in certain cases, especially such as are of long standing and of quartan type, the remedy seems altogether to fail. The drug which then proves most successful is Arsenic. The tincture of Eucalyptus may sometimes, it is said, be prescribed with advantage. The application of cold douches to the left hypochondrium is recommended by Hertz as a valuable accessory to other treatment.

For the dangerous *remittent* and continuous forms of ague, and for those cases in which the symptoms are those of apoplexy or of some other cerebral disease, Quinine is still the remedy, but it must be employed much more boldly. Maclean insists on the importance of watching for even a slight remission, using it as an opportunity of giving fifteen or twenty grains of quinine by the mouth, and repeating the dose in two hours' time. If the stomach rejects it, a similar quantity may forthwith be administered per rectum, but in the most severe cases of all he does not wait a moment, and not only prescribes quinine energetically, but also gives stimulants and plenty of nourishment. This writer also speaks in high terms of Warburg's tincture, a remedy at one time secret, but now known to contain, in addition to quinine, aloes, rhubarb, camphor and opium. It appears to be a powerful sudorific.

The *malarial cachexia* requires the administration of iron as well as quinine, but even these remedies are of secondary importance in comparison with the removal of the patient to a healthy locality. Maclean speaks strongly of the value of the ointment of red iodide of mercury in the treatment of chronic enlargements of the spleen and of the liver. He directs that a piece of the size of a nutmeg should be rubbed into the skin of the hypochondriac regions, and that these parts should be then exposed to the heat of a fire.

YELLOW FEVER*

History—Incubation, course, and events—Morbid anatomy—Diagnosis—Ætiology and pathology—Question of its contagious or miasmatic nature—Preventive, climatic, and curative treatment.

I HAVE now briefly to describe an infectious febrile disease which is almost limited to tropical America, the West Indian Islands, and the West Coast of Africa. It has sometimes been imported into Europe, but never maintains itself among us. In the East it is altogether unknown. The first epidemic on record was in 1647, when it appeared in Barbadoes. A destructive pestilence of the same kind appeared at Philadelphia in 1699, and again in 1762 and 1793 and 1802. It visited Mauritius in 1815, and Gibraltar in 1804, 1814, and 1828. It is endemic in the island of Hispaniola (Haiti and San Domingo). Its range is certainly wider than it was in the last century. There was an epidemic in Lisbon in 1857 ; and another at Swansea in 1865, introduced from Cuba in the "Hecla" barque ; this happily did not spread.

Course.—The incubation of Yellow Fever is said to be usually from one day to fourteen or fifteen, but there are doubts whether it may not be considerably longer in certain cases. There are sometimes slight prodromal symptoms of malaise, headache, &c. ; but very often it sets in suddenly with rigors, extreme depression, and all the other symptoms of severe pyrexia. Sometimes it proves fatal in a few hours with collapse and lividity. More often the temperature rises for two or three days, until it reaches 105°, or, as was once recorded, 107° F. In such cases the face becomes flushed, the conjunctivæ are red and ferrety, and the eyes bright and staring. Frequently there is pain on one side of the forehead, or an agonising pain in the back and sacrum ; or there may be pains in all the joints. The stomach becomes very irritable, and rejects its contents ; the epigastrium is tender to pressure. The palate is reddened and œdematous ; the gums may be swollen and inclined to bleed ; the tongue is of a bright crimson colour, or thickly furred ; at a later stage it becomes clean, smooth, and raw looking. Constipation is more frequent than diarrhœa. The urine is very scanty, with deficient urea, and it generally contains albumen.

About the third day, or not until the fourth or the fifth, the next stage succeeds ; the skin becomes jaundiced ; the urine is bile-stained ; but the fæces are seldom clay-coloured. Hæmorrhages now occur from various surfaces ; epistaxis is frequent ; sometimes there is cutaneous purpura ; the vomited matters, from being "white" and consisting of an acid watery liquid, become "black," containing dark-brown flocculi or masses of altered blood-corpuscles. It is to be observed, however, that this symptom of "black vomit" occurs

* *Synonyms.*—Typhus icterodes—Bilious Typhoid Fever—Specific diffuse Hepatitis—Icterus gravis with parenchymatous hepatitis—Calentura vomitonegro—Coup de barre—Mal de Siam (probably a bilious remittent, not Yellow Fever).

only in the more severe forms; and chiefly in such as end fatally. Thus during an epidemic at Gibraltar in 1828, Louis found that of the patients who died, all but one had it, and of those who recovered very few. According to Alvarenga, however, at Lisbon, in 1857, there were no fewer than forty recoveries among cases in which black vomit was present.

Haenisch, the writer on this subject in 'Ziemssen's Cyclopædia,' who has himself seen the disease in the West Indies, states that there is usually on the fourth day a remission of the pyrexia, so that the temperature in twelve hours may fall nearly to normal. At the same time the patient loses his headache, and experiences so much relief that he fancies himself out of all danger. The stomach, however, still remains irritable; and the urine, if not previously albuminous, now becomes so. In some cases the improvement is permanent, and goes on to complete convalescence. But more often, after a few hours or within two days at the longest, matters change again for the worse. The temperature may now rise until it reaches 104°; but sometimes remains normal, as, for instance, in the case of a gentleman who died of yellow fever in London in 1878, under the care of Mr Leggatt ('Clin. Trans.,' vol. xi).

The symptoms at this period seem mainly to depend upon disturbance of the renal secretion. The urine is often entirely suppressed for several days together; it always contains albumen and often casts. The patient now falls into an apathetic condition, or he may become violently delirious. The pulse is small and thread-like, sometimes rapid, sometimes slow. The jaundice increases until the skin has a dark mahogany colour. Hæmorrhages continue, blood being passed from the whole length of the intestine, from the female genitalia, from the external ear, and from the respiratory surface. Death is usually preceded by coma, but sometimes it occurs suddenly, the patient falling backwards upon his bed in syncope, after a state of violent excitement. But recovery may take place even when the case has appeared most threatening; the temperature then falls quickly to normal, with profuse sweating.

Prognosis.—The mortality from the disease seems to vary widely in different epidemics, being sometimes as low as 15 per cent., sometimes as high as 75 per cent. The symptom of most value as regards prognosis at an advanced stage is said to be albuminuria; if the amount of albumen in the urine diminishes as the case goes on, the patient is likely to do well; if it increases, a fatal termination is to be apprehended. Ordinary bilious vomit is accounted a good sign.

Among the *sequelæ* observed are suppurative parotitis, sloughing of the scrotum and penis, and the formation of boils containing blood mixed with pus. Convalescence is always slow, and several weeks elapse before the patient regains his strength.

It must be added that during an epidemic of yellow fever very mild cases occur, in which the symptoms are but slightly pronounced. The pyrexia is moderate in degree; there is but slight jaundice, or it may be altogether absent, or may appear only when the attack is passing off.

Morbid anatomy.—Rigor mortis is early and well marked; and, according to Nielly, putrefaction is retarded, which is certainly contrary to what one would have anticipated. The body is deeply jaundiced. Mr Macdonald (in 'Reynolds' System') says that the discolouration is sometimes more marked than it was during life. The heart is often pale and soft,

and the fibres in a state of granular degeneration. The pleuræ are ecchymosed, and the lungs may contain infarctions. Intermuscular and meningeal hæmorrhages are found. There is usually acute catarrh of the whole length of the alimentary canal, and the stomach sometimes shows hæmorrhagic erosions; but in Mr Leggatt's case the stomach was pale and its mucous membrane was not swollen. According to Macdonald, the stomach appears perfectly normal when the digested black blood is washed off.

The liver may present patches of a bright yellow colour, or its tint may be that of coffee. The only histological change which is usually described in it is that the hepatic cells are loaded with fat globules; but in Mr Leggatt's case, Dr Greenfield, who made the autopsy, found that the portal canals were crowded with leucocytes, that many of the bile-ducts were filled with swollen epithelium, and that the greater part of the hepatic cells were undergoing disintegration. They were swollen and fused together, or broken up into irregular fragments. It may, however, be a question whether in this case the state of the portal canals was not an accidental cirrhotic change, due to habitual indulgence in alcohol. Dr Greenfield's observations are of great interest, as tending to support a doctrine, upheld by Liebermeister in his work on 'Hepatic Diseases,' that yellow fever is nothing else than a "parenchymatous hepatitis" dependent upon a specific infection. This view is supported by Dr Wickham Legg in his work on the 'Bile and Jaundice' (p. 546). He cites the description of microscopical appearances by Dr Alonzo Clark, of New York, and several other observers. Acute fatty degeneration with atrophy is constant. The jaundice in this disease is described as "hæmatogenous" by those who distinguish sharply between hæmatogenous and hepatogenous forms of jaundice. The biliary passages are found free from obstruction; the gall-bladder contains dark green or tarry bile.

The spleen is but slightly if at all enlarged; its tissue is often firm and healthy looking, and is not unfrequently shrunken.

The kidneys are enlarged and may show points of suppuration; in the case already referred to, Dr Greenfield found that the convoluted renal tubes had their epithelium swollen and proliferating, and that the straight tubes contained hyaline casts; but it is to be observed that the kidneys were in this instance affected with chronic disease, and that therefore even the acute changes may have been unconnected with the yellow fever. According to Dr Legg, however, a condition of cloudy swelling like that found in the early stage of parenchymatous nephritis is the rule.

The *diagnosis* of yellow fever is not always easy except when it is known to be prevalent. The affection most apt to be mistaken for it is the bilious form of remittent fever. Other diseases that must be borne in mind are relapsing fever and the various forms of local jaundice attended with pyrexia.*

The "bilious typhoid" described by Griesinger in Egypt was probably bilious remittent fever, but it has been also identified with yellow fever.

Ætiology.—With regard to the origin of yellow fever there have been great differences of opinion, and perhaps the time has not even yet arrived when final conclusions can be arrived at. One of the most striking features of the disease is that its infectious principle is often transported by vessels from one place to another. I need only cite a few instances which have become historical. Thus in 1823 H.M.S. "Bann" carried yellow fever from Sierra Leone to

* Laroche, 'Yellow Fever' Philadelphia, 1855, 2 vols.

the island of Ascension. In 1845 the "Eclair" steamer brought it from the African coast to Boà Vista, one of the Cape de Verde Islands. In 1861 t was conveyed by a wooden sailing-ship, the "Anne Marie," from Havannah to St Nazaire in France; in 1865 by a similar vessel, the "Hecla," from Cuba to Swansea. Moreover, the disease frequently passes from one ship to another, not only where they have been lying side by side in the same port, but also on the high seas. The "Anne Marie," for instance, infected seven other vessels which happened to be brought near her; and during the local epidemic at Swansea, a smack, the "Elinor," which took in cargo near the "Hecla," had her crew attacked after they had left that port and had gone to Llanelly. So, in 1795, the "Hussar" frigate captured a French ship, the "Raison," on which yellow fever was prevailing; only those prisoners who were believed to be in perfect health were transferred to the English vessel, but notwithstanding this precaution the disease soon broke out on board her.

These facts are readily explained by the hypothesis that yellow fever, like typhus and the exanthemata, is propagated by a contagious emanation from the bodies of the sick; but it is to be observed that the infection has often been found to cling to the hull, or perhaps to the cargo, of a particular vessel, after the crew have been paid off. In the 'Med.-Chir. Review' for 1848 and subsequent years, there appeared a series of very able articles, which are now known to have proceeded from the pen of the late Dr Parkes. He professes to furnish unequivocal proof that the disease is sometimes if not always contagious. He relates in full detail the circumstances which attended the diffusion of it in Boà Vista from two soldiers, belonging to the fort, who were lodged while ill in the chief town of the island, Porto Sal Rey, as well as from a labourer, who brought it direct from the "Eclair" into another town, Robil; and in each case he shows that the next persons to be attacked were those who lived close to the patients and visited them.

On the other hand, many of those who have had the largest acquaintance with yellow fever have disbelieved in its contagiousness, in the ordinary sense of that term, and many facts have been adduced in support of their view. One is that, as Griesinger has pointed out, the disease often remains localised upon the seashore, or in close proximity to the banks of a navigable river; it may even confine itself to a small part of a seaport town in the immediate neighbourhood of the harbour. Thus, when it prevailed at Lisbon in 1857, one hundred and eighty-two persons are said to have left the city for different places in Portugal, carrying with them the disease, and eighty-six died, but in no instance was it communicated to other persons in the places whither they went. In 1865 Dr Buchanan, having investigated with great care the local epidemic at Swansea, came to the conclusion that "the evidence tending to negative personal contagion was about as strong as such evidence can by its nature ever be."

Pathology.—Yellow fever is a specific disease, and either miasmatic-contagious or miasmatic only (cf. p. 21). The first of these hypotheses is held by some recent German writers, but I am not acquainted with any facts which support it; I do not know of any instance in which yellow fever has been traced to drinking water, nor does there seem to be any reason to suppose that the evacuations of the sick contain a poison which enters the soil, and then undergoes a further stage of development which fits it for re-entering the human body. But there seems to be much to be said in support

of the view that the infective principle is a miasm. By this I do not mean that it is identical with malaria. Dr Parkes, indeed, in the articles above referred to, maintains that yellow fever is, or may be, developed out of ague, which, in fact, undergoes conversion from a non-contagious into a contagious disease. He considers it to have been proved, for example, by Dr Bryson that when the "Eclair" left Sierra Leone on her way to Boà Vista, there was no yellow fever in the former place, so that the crew of that vessel must be supposed to have derived the fever from the usual "endemic sources," to which they were "exposed in a most intense degree both in their boat-services and in the unhealthy anchorages to which the nature of their duties drives them." So, again, he cites the case of a party of men detached from H.M.S. "Syren" in the Banana Islands in the summer of 1847; among these men, according to Dr Bryson, a series of cases of fever arose, of which "the first was of a purely endemical character, while the later ones merged into yellow fever with black vomit."

The ultimate decision of pathologists as to this question will doubtless be governed to a great extent by their judgment as to the development of contagious erysipelas or of diphtheria from simple non-specific affections. But there is one fact which seems to point strongly in the direction of regarding yellow fever as a specific contagious disease; namely, that it protects against itself, so that a person seldom has it a second time. I must admit, however, that this circumstance is deprived of some of its force by the further facts that mere residence in a hot climate seems also to impart a considerable degree of immunity from yellow fever, and that negroes are scarcely, if at all, more susceptible to this disease than they are to ague.

A particular instance, in which yellow fever is supposed to have been set up by the emanations from swampy ground in Barbadoes among the men of two regiments in 1841, is recorded by Mr Lawson in the 'Lancet' for 1879. And it is obvious that the fact cited by Dr Parkes with regard to the spread of the disease at Boà Vista are quite consistent with the hypothesis that the infective principle of the disease, having once been introduced into the island from the "Eclair" upon clothing or upon the persons of those who landed from the ship, or having even been carried into Porto Sal Rey by the soldiers who were taken ill in the fort, afterwards established itself in the soil, and flourished as a pure miasm. And it seems very probable that when infection clings to the hold of a ship, the requisite nidus for a specific microphyte may be afforded by the bilge-water.

Whatever the decision as to the miasm or contagion, the question remains as to the relation of yellow fever to other forms of malignant jaundice (icterus gravis). Grisolle, Garnier and Liebermeister maintain that the two are identical, and that the cases of acute yellow atrophy of the liver which occur in Europe are specific cases of yellow fever.

This can scarcely be. The absence of fever in the former, and its presence and height in the latter; the atrophy as well as softening and degeneration of the liver, the swollen spleen, and the entirely non-contagious character of the former; to say nothing of the absence of hæmorrhagic vomiting and the presence of leucin and tyrosin in the urine—all these facts prevent our identifying the two diseases. But the icterus, the hæmorrhages, the rapid course, and the histological change in the liver, with the affection of the kidneys, show a close relation between them.

Prophylaxis.—If yellow fever is a miasmatic disease, the precautions

which should be adopted in order to prevent its transport from one country to another may be greatly simplified, in comparison with the regulations laid down until recently by the best authorities, as, for instance, by M. Mélier in France after the St Nazaire epidemic in 1861. As Mr Simon remarks in his 'Eighth Report,' the segregation of persons arriving from an infected town, and their confinement in a lazaretto for a definite period of time, is, on this view, superfluous, and in a trading country like England, all but impracticable. The points to which the whole energies of a port sanitary officer should be directed are the isolation and disinfection of the vessel which is known or suspected to contain the virus of the disease. It should be compelled to anchor at a distance from all other vessels; and every part of the hold should be thoroughly cleansed. It would now be advisable to use carbolic acid for this purpose, rather than the chloride of lime which M. Mélier recommended. The cargo, and the clothes and other effects of the sailors and passengers, should be disinfected at the same time.

The necessity for stringent precautionary measures against the development of yellow fever in England and in other temperate climates, is, however, very much diminished by a circumstance which I have yet to mention, namely, that a high external temperature is believed to be a very important, if not essential, factor in its ætiology.

Climate.—It has generally been said to be incapable of maintaining itself except in hot climates; and I may remark that this affords a further reason for supposing that its infective principle multiplies itself outside the human body and where it is freely exposed to atmospheric influences. The regions in which the disease commonly prevails are all situated near the equator; and the occurrence of a local epidemic within the temperate zone seems constantly to be associated with an exceptionally sultry state of the weather at the time. This was the case at St Nazaire, in France, when it developed itself there in 1861; and also at Swansea, in 1865. The latitude of Swansea is 51° 37′, which is beyond the geographical limit usually laid down for the disease. So, again, upon low coasts and near the mouths of rivers, the worst months are generally July, August, and September; although in small rocky islands within the tropics, the period of the year from October to February is stated to be the most dangerous. Of late, however, some exceptions have been recorded to the rule that the infective principle of yellow fever cannot resist cold. Thus Dr Archibald Smith recorded, in the first volume of the 'Transactions of the Epidemiological Society,' the fact that in 1855 it prevailed at Cuzco, in the Peruvian Andes, where (as he was informed) the temperature of summer rarely, if ever, reaches 65° Fahr. in the shade; and also in Cerro Pasco, with a mean temperature of 44° by day. So, again, Mr Leggatt's patient, who died in London, was taken ill on March 21st, 1878, the mean temperature of the four previous days having been 54°. He had arrived at Southampton from Rio (where the disease was epidemic) on March 17th. There had been three cases of yellow fever on board the ship on her voyage home; at that time the days are said to have been hot, but the nights cool. Afterwards the weather had been cold.

Treatment.—The treatment of yellow fever is mainly symptomatic. Quinine has no specific influence on the disease. A laxative, or enema, is employed at the beginning. Some experienced physicians give a purge of calomel, jalap and ginger. The cold-pack seems to be useful in many

cases, but more constantly useful, especially at the onset, appears to be a hot foot-bath. For the relief of sacral pains dry cupping may be used. The irritability of the stomach may be checked by a hypodermic injection of morphia, or by the administration of a few drops of chloroform or of chlorodyne. Lime-water is also mentioned as serviceable in this direction. The patient's strength must of course be kept up as much as possible by beef-tea; but bland liquids, such as thin arrowroot, barley-water, or chicken broth, are often best retained by the stomach. Gum-water, made by dissolving three drachms of pure gum-arabic in six ounces of cold water, has been recommended; a tablespoonful to be given every hour or two when the state of the tongue indicates that the mucous membrane below is raw and irritable. Ice is of course to be used, if it can be obtained; and it seems to be the usual practice to allow the patient brandy well diluted with water, or some effervescing wine, such as champagne. In one case recorded by Mr Macdonald the cook of H.M.S. "Icarus" very nearly succumbed to yellow fever, "but rallied immediately on the administration of a stout glass of rum and water and recovered steadily."

DENGUE*

History—Names—Course and symptoms—The fever and the interval—The exanthem—Sequelæ—Pathology—Contagion—Diagnosis—Prognosis—Treatment.

In 1824 an epidemic of a kind hitherto unknown to Indian surgeons broke out at Rangoon in Burmah, and quickly spread to Calcutta and to various places in Bengal or in Madras. In 1827 the same malady appeared in the West Indies in the Isle of St Thomas, and a few weeks later in the adjacent Isle of Santa Cruz. Here also it was at first regarded as a new disease; but it has since been found that Dr Rush, of Philadelphia, described it as prevailing in that city in 1780. During the last fifty years it has from time to time attracted attention both in the Eastern and the Western Hemispheres. The latest outbreak in India seems to have been in 1871–72, when it is said to have been derived from Zanzibar, passing to Bombay by way of Aden; in 1873 it spread to China and to Cochin China, and also to the islands of Mauritius and Réunion. In the West Indies it was last seen at Martinique in 1874 and in 1875. The only spot in Europe in which this malady has hitherto appeared is Cadiz, where it was observed by Poggio in 1867; an earlier epidemic is said to have occurred in the same town as far back as 1784.

In St Thomas the English negroes called it "dandy fever," apparently in ridicule of the attitude and step assumed by those who were seized with it. Other absurd names for it have been "breakbone fever," "broken-wing fever," and "giraffe," the latter because the neck is held in a stiff position. Several later terms have been applied to it, but it is now universally known as Dengue, not only in England, but also in France and in Germany; this is a Spanish word, analogous to dandy in its meaning, except that it is applied to women and not to men.

Course.—Dengue sometimes sets in with lassitude, drowsiness, vertigo, a sensation of chilliness down the back, and other symptoms of commencing pyrexia, but more often it begins suddenly with pain in some particular part of the body, perhaps while the patient is walking about, or during the night, waking him from sleep. If the pains affect the knees while he is standing, he may suddenly fall down. Dr Stedman, who in the 'Edinburgh Med. Journ.' for 1828, gave an excellent account of the disease as it occurred in St Thomas, says that the first thing noticed was often a stiffness in one finger, especially the little finger; this would increase and be accompanied by intense pain, which spread over the whole hand and up to the shoulder, and in a few hours the fingers of both hands would be swollen, stiff, and painful, and their joints incapable of being bent. I do not find that any

* *Synonyms.*—Dandy Fever—Breakbone Fever—Three-day Fever—Scarlatina Rheumatica—Febris exanthematica articulosa. The word *Denguis* has been coined as a Latin name of the disease.

writer states that effusion can be detected in the articular cavities, as in rheumatic fever; but Hirsch says that, in those cases in which autopsies were made, serous infiltration was found to have occurred into the connective tissue round certain joints, and that in one instance the crucial ligaments of the knee were reddened. Cotholendy, in describing dengue in the Isle of Réunion, suggests that there is an exudation into the sheaths of the tendons, and especially of the extensor tendons, and that it is this which renders movements of the joints so exquisitely painful. He also speaks of "a slight fulness, a sort of œdema," of the hands and feet. There is often violent pain in the eyeballs, which feel too large for their sockets and as though they would start from the head. Before long every part of the body becomes the seat of the most extreme suffering, which is aggravated by restlessness, compelling the patient to be constantly changing his position, while he also feels extremely prostrate and distressed. Sleeplessness is a marked symptom, and children are sometimes delirious.

In the meantime the temperature rises until it reaches 102°, 103°, 104°, or even (though very rarely) a higher point still. Observers who have employed the thermometer during some of the more recent epidemics have attached some importance to the occurrence of remissions, which follow one another three or four times in the twenty-four hours; but I should doubt whether this is so remarkable a feature of the disease as they seem to think. Most writers have described the pulse as very rapid, 120 or even 140 in the minute, but a writer in the 'Arch. de Méd. Navale' for 1874, says that he often found it not much over 80, lower in fact than might have been expected from the temperature. Twining long ago noticed that the countenance was flushed and of a scarlet hue, and of late it has become the fashion to speak of an exceedingly fugacious *initial rash*, consisting of bright red patches upon the face, the chest, the palms of the hands, and elsewhere, which, however, subside after the lapse of a few hours. There is anorexia; the tongue is thickly furred, with bright red edges. The stomach is extremely irritable, and often rejects everything that is swallowed. The bowels are constipated.

The condition of the patient at this period might well cause considerable anxiety to a medical man unacquainted with the characters of the disease; but at the end of twenty-four or thirty-six hours the pyrexia begins definitely to subside, without any marked critical sweat, and the temperature soon falls to normal or slightly below it. At the same time the pains in the limbs cease, and soon there is nothing for the patient to complain of except a sense of general weakness, loss of appetite, and backache. At this time it is said that the glands in the neck, the axillæ, and the groins may be felt to be slightly enlarged. The parotid is often swollen and salivation is frequent.

The duration of the interval is about three days. At the end of it an eruption—a *second rash*, according to recent observers of the disease—appears upon the skin. It is first seen upon the palms of the hands, and it also affects the feet and the knees, and may in exceptional cases spread all over the body. In character it is usually intermediate between the rash of scarlet fever and that of measles. It has also been compared with the erythema that sometimes accompanies rheumatic fever. Or it may resemble urticaria, or in some cases be attended with the formation of bullæ as well as wheals. It gives rise to a distressing sensation of tingling, which presently passes into still more intolerable itching. Its development is sometimes

associated with a return of pyrexia, but recent observations seem to have shown that, as a rule, the temperature remains normal at this period of the disease. Consequently the fact that an eruption has made its appearance is not seldom altogether overlooked; whether it is ever really absent is doubtful. After a few hours, or two or three days, it subsides and disappears, and with it the fever, if any has been present. Afterwards, however, the cuticle begins to desquamate, usually as a branny powder, but sometimes in very large flakes, and this may be attended with considerable discomfort and soreness, especially of the feet.

The second eruption is often associated with a repetition of the articular pains, which are, however, less severe than at the beginning of the disease. Or there may be a respite of three or four weeks, and at the end of that time the pains may return, compelling the patient to take to his bed again and to be fed like a child. Dr Stedman speaks of these pains as always most severe in the morning and as wearing off in some measure towards evening. They are felt chiefly in the joints of the fingers and of the toes, in the wrists, in the ankles, and in the knees. The affected articulations may be stiff and swollen to such a degree as to produce deformity. After a few days the secondary pains in their turn begin to subside, and one joint after another becomes free from them; but months may elapse before they have entirely disappeared. It is even said that there may be a permanent partial anchylosis of some particular joint. Long-continued weakness is a frequent sequel, and jaundice an occasional one.

Pathology and causation.—The true place of dengue among infective diseases has yet to be accurately determined. In the rapidity with which it spreads over a population it resembles influenza more than any other malady.

A remarkable point is that scarcely anyone escapes, even among the inhabitants of a large city, as, for example, among the half million of residents in Calcutta in 1824. It attacks persons of all ages, including even infants a few days old, and the coloured races are as liable to it as whites. When it has existed for a little time in a place and patients are recovering, one might think that there were none but cripples in the place, so many are seen limping about the streets on crutches, with bodies half-bent or with arms in slings. The whole duration of an epidemic is generally short, from two to seven months, according to Hirsch.

The disease is said to prevail chiefly with sultry cloudy weather or at the time of heavy rains. Indeed, in temperate climates it occurs only during summer and autumn, and disappears when frosts set in; but in the West Indies vicissitudes of weather seem to have no way interfered with its course. Its diffusion from one country seems plainly to be effected by human intercourse. Whether it is contagious in a narrow sense, passing from the sick directly to the healthy, appears not to be known with certainty. Dr Stedman, however, says that in 1827 it was introduced from St Thomas into Frederickstadt, a town of Santa Cruz, by some young ladies who went to reside at the house of a Major S—; all the members of his family were attacked, and a few days later it appeared in the next house and affected everyone there. Cotholendy relates that an infant, which had taken the disease while with the family of its nurse, was brought home to its mother; four days afterwards she fell ill; the grandmother and the aunt did not see the child until the day after its arrival, and they were attacked a day later than the mother, each at the same hour.

This writer also gives several other instances which indicate that the *incubation* of dengue lasts four days.

Diagnosis.—The epidemic character and the rash sufficiently distinguish this singular disease from rheumatism, the articular pains and absence of sore-throat from scarlatina or any other exanthem. The endocardium seems never to be affected. It is not malarial, for it affects the whole population equally, it is unattended with splenic enlagement, and is unaffected by quinine.

Prognosis.—Dengue is scarcely ever fatal, but infants sometimes die with convulsions during the primary pyrexia, and old people may succumb to exhaustion towards the end of the disease, or to bronchitis occurring as a complication. Nielly, in his 'Éléments de Pathologie Exotique,' mentions that pericarditis has been observed in severe cases. When untreated, recovery is said to be very slow.

Treatment.—It is recommended to give an emetic at the commencement of this disease, and then a purge of calomel and scammony or jalap, repeated each day. After the bowels have been freely opened, a dose of laudanum, or Dover's powder, completes the cure. Nielly suggests, on theoretical grounds, the use of salicylate of soda or of colchicum, but I have not met with any recorded evidence of the value of these medicines. Liniments of chloroform, belladonna, or cajeput oil are often useful. Dr Stedman found that the application of blisters or of sinapisms to the neck or to the loins gave great relief to the pains in these parts. When one or more joints remain stiff and painful after the subsidence of the disease, sulphur baths are said to be very efficacious.

EPIZOOTIC DISEASES

OCCASIONALLY TRANSFERRED TO MAN

ANTHRAX: *Nomenclature—Distribution—Varieties in animals—The Bacillus—Ætiology—Modes of infection—Varieties in man: Charbon, Intestinal and Thoracic Anthrax.*

GLANDERS: *Its history and nomenclature—Origin—Acute form—Eruption, ozæna, &c.—Pyrexia—Fatality—Chronic form.*

FOOT-AND-MOUTH DISEASE: *An epidemic disorder among cattle—Occasionally occurs in man—Its mode of conveyance—Its symptoms.*

ACTINOMYCOSIS: *Its occurrence in cattle—Its appearance in the human subject.*

THERE are certain epizootic maladies which sometimes pass from animals to man. These may conveniently be described together in this last chapter on the specific contagious epidemic maladies, of which pathologically they are examples.

ANTHRAX.*—It seems probable that the name of Anthrax, which has hitherto been used as a synonym for carbuncle, will in the future be reserved for a very different disease which human beings derive by infection from one that is epidemic in cattle, horses, sheep, and other herbivorous animals. This disease has until lately been very imperfectly understood in England, and has generally been termed *Malignant Pustule* or *Splenic Fever*. Certain forms of it have, in all European countries, been commonly known under the name of *Anthrax*, and others have been described as *Anthracoid*.

With regard to the *geographical distribution* of anthrax, the parts of Europe in which it is most common are said to be Poland, Hungary, the countries on the Lower Danube, Prussian Saxony, and certain departments of France. It is very prevalent in Siberia ("Jaswa"), and in the western part of Asia, in Lapland, in India (Loodiana plague), in South Africa (Horse-sickness), Australia, Mexico, and South America. It occurs not only in domesticated animals, but also in deer, reindeer, buffaloes, and even elephants. It also affects swine. It is easily inoculable upon rabbits and guinea-pigs. Carnivorous animals are much less susceptible of it; but cats are more so than dogs. It is endemic in Catalonia, in the Romagna, and in Courland.

Varieties in cattle.—In different kinds of animals the symptoms of infection by the specific virus of anthrax differ considerably. But, according to Bollinger, three principal varieties may be recognised:

1. Sometimes the animal (generally an ox or a sheep) becomes convulsed and insensible, with rapid breathing, and dies after a few hours, or even within half an hour of the first seizure.

2. Other cases are characterised mainly by pyrexia, which is often remarkably remittent in type. Clonic spasms of the limbs are also observed. The fæces may contain a large quantity of blood.

* *Synonyms.*—Splenic Fever—Splenic Apoplexy—Malignant Pustule.—*Old English* Blackbain, *Fr.* Charbon, Maladie de Chabert (in part), *Germ.* Milzbrand.

3. In yet other cases the most marked feature is the formation of brawny inflammatory swellings in and beneath the skin of the neck, chest, abdomen, or any other part, which may lead to extensive sloughing and ulceration.

Obviously only the last of these varieties would naturally be termed anthrax. But it is on every ground desirable that a single name should be given to them all. Indeed, although veterinary pathologists until lately regarded the more rapidly fatal cases as examples of a specific fever without localisation, it is now known that there always is a local lesion in some part of the body. Very often this consists of an infiltration of gelatinous exudation and blood into the connective tissue of the abdomen and of the chest. Or there may be an acute inflammatory swelling of the tongue, or of the submucous tissue of the pharynx, or of the lining membrane of some part of the intestine. We shall presently see that all these forms of the disease are also met with in men. It is to be observed that anthrax is by no means always fatal to animals; the average mortality in horned cattle and in horses is said to be 70 per cent. In cattle a very characteristic lesion is enlargement of the spleen, which is from two to five times its normal size, softened and of a black colour.

The Bacillus.—The proof of the identity of the various forms of anthrax lies in the fact that all are caused by a single microphyte, which is now commonly known as the *Bacillus anthracis.*

This organism was discovered independently by two German observers—by Pollender in 1849, by Bräuell of Dorpat in 1857. But Davaine was the first who, in 1863, maintained that the contagion of the disease lay in these bodies, which on account of their being motionless he distinguished from the common mobile bacteria of putrefaction by the name of *bacteridia.* They consist of straight or slightly-bent rods, measuring 0·007—0·002 mm. in length. Frisch and Dr Cossar Ewart (now of Edinburgh) have under certain circumstances observed movements in them, but as a rule they are motionless. By Cohn they have been classified as a bacillus which he termed *Bacillus anthracis,* though he observes that it is almost exactly like the *Bacillus subtilis* which constitutes the butyric acid ferment, except that the latter is motile.

The rods sometimes cohere together at their extremities, and Koch and Ewart have described them, when cultivated in aqueous humour, as growing into long filaments, in the interior of which bright granules appear. To these granules great importance is attached, for they are spores of the bacillus, far more capable of resisting heat and desiccation than the adult microphyte, and retaining for an indefinite length of time the power of development into it. Such a view was rejected by Nägeli, who maintains that bacilli, like bacteria, multiply only by fission. The *Bacillus anthracis* does multiply by fission under the surface of an infusion, but by spores when exposed to air, and also under certain conditions by assuming a Torula-form. These facts have been ascertained by Klein, so that Koch's original statement is abundantly confirmed. The bacilli are found in the blood, spleen, and many other organs.

Infection.—The origin of anthrax, as it occurs in cattle and horses and sheep, is of great interest, and seems to have important bearings upon the class of human diseases which are known as *miasmatic-contagious* (p. 21), and of which it seems to be a representative.

In the first place, it is readily transmissible from one animal to another. Inoculations are frequently practised by veterinarians and by pathologists for diagnostic purposes. There is reason to believe that the virus is not

infrequently introduced by the stings of insects. Bollinger took gadflies from the body of an ox that had died of anthrax, and found by direct experiment that the contents of the stomach and intestines of these flies were capable of conveying the disease to rabbits. He even thinks that droppings from flies upon the skin of an animal may suffice to communicate it. Another way in which it is sometimes given to sheep is by the bite of a dog that has just been feeding upon infected flesh.

On the other hand, there seems to be no proof that emanations from the bodies of living animals affected with anthrax pass into the air and infect other animals through the lungs. Like enteric fever and cholera in man, it appears not to be directly contagious. Yet the virus of the disease is said by Bollinger sometimes to cling to stable utensils, harness, straw, or hay. He mentions a local outbreak which he carefully investigated at Weriken, and in which for four years the cattle in two sheds of the same proprietor were decimated by anthrax, while it did not affect those in other buildings, some of which actually lay between the two that yielded so many cases. An analogy for this occurrence may be found in those local house-epidemics of enteric fever in which the virus seems to go on multiplying itself in or near a particular water-closet, so as to affect a number of persons in succession, sometimes at long intervals of time.

Anthrax also resembles the miasmatic-contagious diseases of man in being often spread in ways still more indirect. It prevails especially among animals pastured upon damp soils containing much humus, as, for instance, upon peat-bogs, and near the borders of lakes or rivers that have overflowed. And it is most frequent during the hot months of the year, particularly in August and September. There often seems to be a connection between it and ague; but this is only a coincidence, for in other districts the two affections occur quite independently of one another.

Buhl has accordingly included anthrax among the diseases to which he applies his "ground-water" theory; and he states that among the horses belonging to a large stud kept near Donauwörth the disease, which had been raging for a long time, ceased as soon as a system of drainage was carried out. But Bollinger argues that the reason why its prevalence is affected by dampness of soil is, in reality, that ground containing moisture affords conditions favourable for the multiplication of the *Bacillus anthracis*, which he supposes to be capable of maintaining an independent existence under such circumstances. Some observers have even thought that this organism may sometimes appear without having been derived from a previous case of anthrax. Buchner asserted that, by a series of experiments, he converted a bacillus which is found in infusion of hay into an organism capable of producing in animals an infective disease identical with anthrax, and he asserts that to turn the anthrax-bacillus back into a hay-bacillus is comparatively easy. There is, however, good reason to disbelieve this alleged transformation. See Prof. Klein's Report to the Local Government Board, "On the Relation of Pathogenic to Septic Bacteria, as illustrated by Anthrax Cultivations" (Blue-book for 1882).

The malady is never purely *miasmatic*. The specific bacilli of anthrax exist in the soil, or in water, only when they have been introduced from a previous case. They may be either derived from the excreta of diseased animals, or from their dead bodies, buried as they often are in the fields, or even left to decay among the brambles and nettles. Instances are recorded in which "enzootics" of anthrax have ceased, when stringent rules for the

disposal of all dead bodies were enforced. Probably some of the bacilli undergo desiccation, are suspended in the air, and inhaled into the lungs; others pass into drinking-water, and thus reach the stomach.

Conveyance to man.—Shepherds, farm-labourers, and other persons who come into contact with living animals affected with anthrax seem seldom, or never, to take the disease by mere *contagion.* But veterinary surgeons may be infected in performing venæsection, and slaughterers in killing or in skinning; the probability is that a slight abrasion or scratch upon the skin then becomes directly inoculated with the virus.

Another way in which anthrax may arise in man is as the result of *eating the flesh* of an infected animal. Leube, of Jena, has recorded an instance in which the imperfectly cooked liver of a diseased goat conveyed it. Thus the human gastric juice seems not necessarily to destroy the virus, as has been found to be the case with the gastric juice of carnivorous animals such as the dog. Thorough cooking probably removes all danger, but there still remains the risk of injury to the butcher, and to those who are concerned in preparing such meat for the table; and this is sufficient reason why the use of it, as food, should be prohibited. According to Hensinger the disease may even be conveyed by milk or by butter.

But by far the most important cause of anthrax in man is infection *from the dried skins or hair* of diseased animals. In this way it is brought straight from Asia or perhaps from South America to English workmen.

At Guy's Hospital cases are of not infrequent occurrence among the men engaged in the Bermondsey leather trade, and especially among those employed at a particular wharf on the river-side where foreign hides are unshipped. Broca years ago remarked how often anthrax was set up by carrying skins upon the shoulders. It is remarkable that the danger appears to be greater when the hides are first brought ashore than afterwards.

The same thing has been observed at Bradford, where a peculiar *Woolsorters' Disease* has for many years prevailed, and this has recently been shown to be identical with anthrax. The source of infection in that case is chiefly "Van mohair," a material which contains much putrid matter and many "fallen fleeces," torn off the bodies of dead animals. Dr Bell, who gave an excellent account of the affection in the 'Lancet' for 1880, remarked that the men who are attacked are chiefly the "bagmen"—that is, those who open the bags in which the hair is packed, shake it out, and sort it into different qualities. The reason is, no doubt, that the virus is in a dried state, and that as soon as it is disturbed it diffuses itself in the air and is inhaled into the lungs. Perhaps it may actually have multiplied itself during the transmission of the bags from Asia Minor; for, according to Dr Bell, the hairs are not known to communicate disease to persons who handle them in the country where they are grown. Indeed, no form of anthrax so severe as the Woolsorters' Disease had hitherto been described as occurring in man. The work of sorting is said to be free from danger, if the contents of the bags are first washed or even moistened with water.*

Other occupations which sometimes lead to infection with anthrax are the picking of Russian horsehair (as reported by Dr Russell, of Glasgow, in 1879), paper-making, and the manufacture of coarse woollen hats.

Varieties of anthrax.—There are several forms of this disease in man.

* See Reports to the Local Government Board by Mr John Spear: 'On Woolsorters' Disease,' 1880, and 'On Anthrax among persons engaged in the London Hide and Skin Trades,'

1. *Malignant Pustule* or *Charbon* is the name by which the most common form has long been known. This consists in the formation of a vesicle, seated upon a more or less brawny base. Being usually produced by the inoculation of a scratch or slight sore, it occurs chiefly on the face, especially the cheek, and also on the neck, the forearm, or some other exposed part.

There is first a period of incubation, lasting generally several days, often ten, but sometimes only a few hours. A slight pricking or burning sensation is then felt, which often leads the patient to think that he has just been stung by an insect. A papule quickly appears, and soon passes into a transparent vesicle which may reach a considerable size. This ruptures and dries up into a dark-coloured scab. Round it there sometimes arise smaller vesicles arranged in a ring. Meanwhile the base of the vesicle becomes indurated, and a red or purple areola is developed round it. A brawny œdema quickly spreads over the adjacent parts, affecting perhaps the whole of one arm, or of one side of the neck. Sometimes inflammation of lymphatic vessels occurs, and the corresponding glands become swollen.

During the early part of this morbid process the health remains unaffected. The patient may continue at his work, and at the most complains of slight malaise and of febrile disturbance. But after about forty-eight hours severe pyrexia often sets in, with delirium, prostration, diarrhœa, sweating, and acute pains in the limbs; and death, preceded by a state of collapse, may occur between the fifth and the eighth days. In one case which occurred at Guy's Hospital, the original vesicle had been situated an inch and a half behind the left angle of the lower jaw, and respiration became so difficult towards the last that recourse was had to tracheotomy. I made an autopsy and found that brawny infiltration had extended deeply to the fauces, so that the entrance of the larynx and its interior were greatly swollen. In other instances the immediate cause of death is septicæmia, or coexistent anthrax of the intestine or lungs.

Diagnosis.—So far as I know, the only disease liable to be mistaken for anthrax affecting the skin is that which, occurring on the face, has been especially noticed by Sir James Paget under the name of *facial carbuncle.* It presents no definite vesicle or central scab; and in the instances which I have seen, the veins of the face have been found obviously plugged with puriform thrombi, which has not been the case in anthrax.

It must, however, be added that Bollinger and other writers describe, under the name of *anthrax-œdema*, a modification, in which there is neither vesicle nor eschar, but only a pale yellowish swelling of the subcutaneous tissues. The eyelids are the most frequent seat of this variety.

The surest way of arriving at a diagnosis of a suspected case of anthrax is often to inoculate a rabbit or a guinea-pig, or even a mouse, with fluid from the part primarily affected, or with blood. Such small animals commonly die in two or three days, or even still more rapidly, with dyspnœa, dilatation of the pupils, and perhaps convulsions. Their blood may

1883. Also papers by Sanderson ('Journ. R. Agric. Soc.,' vol. xvi, p. 267) and Greenfield (ibid., xvii, 30). That the infection of this disease, which was known to the ancients, is conveyable by the hides is implied by Virgil in the concluding lines of the third Georgic:

"Nec tondere quidem morbo illuvieque peresa
Vellera, nec telas possunt attingere putres.
Verum etiam invisos si quis tentarat amictus,
Ardentes papulæ atque immundus olentia sudor
Membra sequebatur; nec longo deinde moranti
Tempore contactos artus sacer ignis edebat."

then be seen to swarm with bacilli. However, if the experiment of inoculation is followed by a negative result, a negative conclusion is not warranted.

Treatment.—The recognition of the ordinary external form of anthrax is of extreme importance, because surgical treatment at an early stage is capable of arresting its progress. The disease is not, indeed, necessarily fatal, even when left to itself. Sometimes the central part of the swelling sloughs out, the surrounding induration subsides, and an ulcer is left which more or less quickly heals. I do not know that any statistics exist as to the proportion of cases in which such a spontaneous cure has resulted; but it is perhaps worthy of mention that among nine instances recorded in 1863 by Dr William Budd, of Clifton, eight ended fatally. On the Continent it has long been the practice to destroy the local lesion as soon as it is brought under the eye of a surgeon. Bollinger cites the experience of two observers who lost only thirteen cases out of one hundred and forty-two; another had still greater success, since among two hundred and nine patients all but eleven recovered. In 1878, my colleague, Mr Davies-Colley, recorded in the 'British Medical Journal' two cases, in each of which recovery took place after excision of the entire mass of indurated tissue, with the application of chloride of zinc paste to the wound. Contrasting with these is a case which was left alone, and which ended fatally. Several other successful cases have occurred at Guy's Hospital since that time. These have been recorded, together with an analysis of seventeen cases by the same writer, in the 'Med.-Chir. Trans.,' vol. lxv, 1882. Since that date (June, 1882—December, 1885), thirty-one more cases have been admitted to Guy's Hospital; and of the total forty-eight cases of external anthrax, thirty-nine recovered after operation. Two typical cases in butchers during an epidemic of anthrax among cattle were operated on by Dr Pitts, of Chelmsford, and recovered perfectly ('Brit. Med. Journ.,' March, 1887, p. 616).

The rapidity with which the surrounding œdema subsides after the operation is often very striking. It may be effectual even when the case is at an advanced stage, and when severe general symptoms are present. But under such circumstances quinine should be given in large doses, and the patient's strength must be kept up with nourishing food and with stimulants. Carbolic acid should also be administered internally, for Bollinger says its therapeutical value in the lower animals when affected with anthrax has been proved beyond doubt.

2. *Intestinal anthrax*, another form of splenic fever, affects the gastro-intestinal mucous membrane. This has been recognised only within the last few years, and chiefly by German pathologists and physicians. It is sometimes associated with an external malignant pustule; a case in point, in which Dr Goodhart made an autopsy, occurred at Guy's Hospital in 1877. Otherwise its symptoms seem to be obscure, so that a correct diagnosis is not likely to be made during life unless the patient is known to have been exposed in some way to the virus of the disease. However, Bollinger, in 'Ziemssen's Handbuch,' gives the following account of the symptoms: the patient first complains of malaise, loss of appetite, pains in the limbs, giddiness, and headache. Then vomiting may set in, and a more or less severe diarrhœa, the evacuations often containing blood. There may be pain in the abdomen, which becomes somewhat tumid. Dyspnœa and lividity appear, with restlessness, and with excitement or stupor. Epileptiform convulsions may occur, the upper limbs may be affected with tetanic spasms,

there may be opisthotonos, and the pupils may be widely dilated. The pyrexia is but slight, and death is preceded by extreme collapse. The duration of the disease is usually from two to seven days, but sometimes it is scarcely twenty-four hours.

On *post-mortem* examination the abdominal cavity is found to contain a moderate quantity of serous fluid which is often blood-stained. The lining membrane of the stomach and intestines shows patches of swelling, generally of the size of lentils or coffee beans, but sometimes one or two inches in diameter. These on section are seen to consist of a pink fleshy infiltration of the mucous and submucous tissues, so that the valvulæ conniventes and other ridges appear firm and prominent; the surface of the affected parts is more or less excoriated and discoloured or covered with an adherent layer of extravasated blood. There are also spots of ecchymosis on both the serous and the mucous aspect of the gastro-intestinal tract. The mesenteric and the lumbar lymph-glands are often greatly enlarged, and of a dark red colour. The mesentery may form a large brawny swelling, and the connective tissue in front of the spine may be infiltrated in a similar manner. The spleen is softened to a pulp, but it is not generally much enlarged. A nodule in the stomach may slough out, and repair begin (Mahomed, 'Path. Trans.,' 1883).

Fatality.—Hitherto, so far as I am aware, no instance of recovery from intestinal anthrax has been recorded. Leube, however, has related a case in which the lower lip, the inside of the cheek, and the hard palate presented indurated patches of a bluish-red colour: epistaxis and hæmaturia appeared, but the malady ended favourably in about a fortnight. The patient, whose name was Schmidt, took fifteen grains of carbolic acid and thirty grains of quinine each day, and the patches in the mouth were cauterised three times daily with carbolic acid. But I must confess to a doubt whether the diagnosis of anthrax was established beyond question. Schmidt had been admitted five weeks before with pyrexia, which proved to be due to latent tuberculosis; and for one night he was placed upon a mattress on which a patient with anthrax, named Werner, had been lying for twenty-four hours about three days previously. As so long an incubation as five weeks could hardly be supposed to have occurred, Leube suggests that the virus was perhaps taken up by, and remained for a time attached to, some article of property which Schmidt is not unlikely to have hidden in his bed during that first night, and which may thus have come directly into contact with dried stains of the bloody fluid that had been expectorated by Werner. It is said, indeed, that the microscopical examination of the blood in Schmidt's case proved that he really was suffering from anthrax; but to this question I shall allude further on. The bacilli are sometimes not to be found; perhaps destroyed by decomposition. See Mr John Poland's case ('Path. Trans.,' 1886, p. 553).

3. *Pulmonary anthrax.*— A third form of anthrax affects mainly the thoracic viscera. It has hitherto been recognised chiefly among the wool-sorters of Bradford. After Dr Bell, of that town, had drawn attention to it, Mr Spear and Dr Greenfield investigated it for the Local Government Board. Dr Greenfield's description of it is briefly as follows:—The earliest symptoms are great prostration and a sense of oppression of breathing. Shivering seldom occurs. The respiration is not much accelerated, but it is laboured and difficult, with a feeling of pressure or constriction. There may be more or less abundant bloody expectoration, or none at all. Auscul-

tation seldom reveals anything more than slight rhonchus. The face is sometimes congested, sometimes pale, with a slight cyanotic tint. The extremities are cold and bluish; even in the axilla the temperature may be subnormal; but in the rectum the thermometer may rise to 102° or 103°. The pulse is rapid and weak and sometimes irregular. There may be nausea and vomiting, but not generally diarrhœa. In many cases the appetite remains good and digestion seems to be unimpaired. There is sometimes a sort of hysterical condition, or a state of mental anxiety and depression; but other patients have been so unconscious of danger as to refuse to send for medical advice until the last few hours of life, or have even attempted to work. Death may be preceded by delirium, convulsions, or coma, or the mind may be clear to the last, and the end come suddenly and unexpectedly. One of Dr Bell's patients lived only seventeen hours after he was first taken ill. Many cases have terminated fatally in from three to five days. Dr Bell says that those who survive for a week generally recover.

The *post-mortem* appearances vary considerably. Decomposition appears to be somewhat rapid. The lung tissue may either be congested or quite natural. There may be blood-stained secretion in the bronchial tubes, and their mucous membrane may be ecchymosed. Sometimes the pleuræ contain a pint or two of fluid, and the lungs are partially collapsed in consequence. There is often blood-stained serous or gelatinous infiltration of the mediastinal tissues, and the bronchial glands are swollen, softened, and ecchymosed. The pericardium may be marked with hæmorrhages, and may contain a blood-stained fluid; or a large quantity of blood may be poured out between it and the sternum. Sometimes the pharynx and the adjacent tissues are infiltrated or ecchymosed. There may also be ecchymoses in the pia mater, in the kidneys, and elsewhere. It is remarkable that the spleen is hardly, if at all, softened. In prolonged cases hæmorrhagic infarctions may be found in the lungs, attended with slight pleurisy; or there may be patches of broncho-pneumonia.

Obviously there may sometimes be considerable difficulty in distinguishing, by clinical evidence alone, this form of anthrax from a severe attack of ordinary pulmonary inflammation. Indeed, until recently, fatal cases of the Woolsorters' disease at Bradford were commonly registered under the head of Pneumonia, Bronchitis, or Congestion of the Lungs. And on the other hand, it can hardly be doubted, as Dr Bell suggests, that a similar affection will hereafter be found to occur in many towns where carpet and blanket wools, dry hairs or furs, are used for manufacturing purposes.

Bearing on general pathology.—Certain points in regard to the distribution of the bacilli in animals or men affected with anthrax have still to be mentioned. They have an important bearing upon the theory of infective maladies in general, because they illustrate what is perhaps a universal law, namely, that the microzymes of contagion multiply locally before they infect the blood to any considerable extent. In the ordinary form of the disease affecting the skin, the bacilli may be found, according to Davaine, about the second or the third day, in clusters embedded in the rete mucosum at the centre of the vesicle. E. Wagner has since found that they are at this time so closely packed in the papillæ of the cutis as to conceal all the tissue elements. Thence they spread both laterally and towards the deeper structures, enter the vessels, and are carried all over the body with the blood. In the gastro-intestinal variety the bacilli infiltrate all the swollen and œdematous tissues,

so that Buhl and others described this affection under the name of *mycosis intestinalis* before they recognised that it has any relation to anthrax.

At an early stage of the disease the blood may contain no bacilli. Therefore, if in a doubtful case one fails to discover them in this fluid, one must not attach much importance to this result. On the other hand presence of well-marked rods is conclusive; but it does not seem to be sufficient to find round bodies only, as Leube did in the case of Schmidt referred to above (p. 373). In man, even when a fatal termination is approaching, the bacilli seem to be seldom found in such immense numbers, and so generally distributed in the blood, as they are in animals. Davaine calculated that from eight to ten millions are present in a single drop of a diseased beast's blood. In Buhl's first case of intestinal anthrax they are said to have been present in the blood of the portal vein, but not in that of other vessels. They may often be detected in the substance of the spleen and of the solid tissues generally, and also in the fluid poured out into the serous cavities.

Mr Barker, in a case published in the 'Med.-Chir. Trans.,' vol. lxix, p. 127, found the bacilli most abundant in the rete and the papillary layer of the cutis, and they appear to be long confined to that part.

The best account of the histology of the disease in this country is given by Dr F. C. Turner, in the 65th volume of the same 'Transactions.'

In anthrax we have a typical example of a specific contagious malady, which is due to the presence of microphytes. All the conditions stated in the note on p. 22 are fulfilled.

The disease is recognisable from its clinical features, and breeds true.

The organism is well marked in its microscopical characters, size, and shape, in its reproduction and its cultivation.

It exists in the blood and tissues of every case of the disease. It exists nowhere else, for its non-mobility distinguishes it from *Bacillus subtilis*.

When a "pure cultivation" has been obtained, it can be inoculated, and will reproduce the disease with fresh broods of organisms in the blood.

The system of *prophylaxis* by inoculation of the attenuated virus, though inapplicable as a practical measure for human beings, is of too great theoretic importance to be wholly passed over.

Pasteur ascertained that by repeated cultivations of *Bacillus anthracis* in mutton broth, at a temperature above 104°—110° Fahr., he obtained a modified virus which when inoculated into an animal produced a mild attack of anthrax which protected from future attacks. These results were tried on a large scale at Milan and afterwards at Chartres, and proved brilliantly successful (see a paper by Mr Geo. Fleming, in the 'Nineteenth Century' Magazine for March, 1882). Unfortunately the result is not always so complete. For even the attenuated virus is occasionally fatal. The Algerian breed of sheep appear to be insusceptible of the same protection, and in Hungary the results were much less successful than in France. See an excellent abstract by Dr Dawson Williams of Pasteur's and other methods of attenuation, and of Koch's criticism thereon ('Microparasites in Disease,' N. Syd. Soc., p. 560). Dr Roy found in Buenos Ayres that the Viscacha (*Lagostomus trichodactylus*), a rodent allied to the chinchilla, is capable of receiving the disease, and that anthrax thence transferred by inoculation to cattle is both mild and protective, at least for a considerable time—so that the analogy to smallpox and cow-pox is very close.

GLANDERS.*—From an early period in the present century it has been known that those whose occupation brings them into contact with horses sometimes become affected with a disease to which these animals are liable. The disease in question was described as far back as the fourth century B.C. under the names μᾶλις and *malleus*.† In England, writers on veterinary medicine have been accustomed to break it up into two affections, "glanders" and "farcy," the former of which is characterised by a morbid state of the nasal mucous membrane, the latter by the formation of nodules and abscesses in and beneath the skin, and by indolent inflammation of the lymph-glands and lymphatics, attended with swellings (or, as they are termed, "farcy buds") at the valves. But it is now acknowledged that these are only varieties of a single malady, and it is to be hoped they will hereafter receive a single name. The first case in the human subject of which the nature was correctly interpreted in this country occurred in 1821 to Mr. Muscroft, of Pontefract. In 1830, Dr. Elliotson drew the attention of the Royal Medical and Chirurgical Society to the disease, and at a later period he proposed to call it *Equinia*. According to Virchow, the derivation of "glanders" is from *morbus glandulosus*, and the equivalent German word *Druse* is still in use for one of the varied forms of the malady.

Contagion.—As a rule, the virus of glanders is directly introduced into a wound or sore, or fissure in the skin, especially of the face or of one of the hands. A horse-slaughterer who died in Guy's Hospital in 1866 appeared to have poisoned a slight cut on the lip which had been made by a barber in shaving him. Injuries to the hand in skinning dead horses, or in dissecting them, have sometimes been the starting-point of the disease. It has also been transmitted by a bite from a glandered animal, probably through the saliva. Or a diseased horse in sneezing may propel a drop of muco-purulent secretion from its nose directly into the eye, or the nose, or the open mouth of anyone standing near it. The contagion remains active in a dried state for a long time, for horses in a particular stable have been attacked many months after the occurrence of a case. Thus, nothing is more likely than that it should sometimes be deposited upon the straw in a stall, and that a groom, or anyone else sleeping upon the straw afterwards, should disturb the died particles and cause them to float off into the air so as to infect him with the disease. However, the Messrs Gamgee in Dr Reynolds' 'System,' express doubts on this point.

According to Bollinger, who describes glanders in 'Ziemssen's Handbuch,' it may be communicated by eating the flesh of a glandered animal, at least in a raw state; he says that lions in menageries often become diseased in this way. Decroix, indeed, is stated to have repeatedly fed upon such flesh uncooked, without injury. But it must be borne in mind that in all probability comparatively few persons are susceptible of the virus of glanders. At any rate the disease in man has always been exceedingly rare, even in countries in which (as in France during the first half of this century) it has committed the most terrible ravages among horses. When it affects human beings, it is capable of spreading from one individual to

* *Synonyms.*—Equinia—Farcy—μᾶλις, Malleus *v.* Maliasmus.—*Fr.* La Morve, Le Farcin. *Germ.* Rotzkrankheit, Wurmkrankheit.

† Οἱ δὲ ὄνοι μάλιστα νοσοῦσι νόσον μίαν ἣν καλοῦσι μηλίδα· γίνεται δὲ περὶ τὴν κεφαλὴν πρῶτον, καὶ ῥεῖ φλέγμα κατὰ τοὺς μυκτῆρας παχὺ καὶ πυρρόν, Aristotle, 'Hist. Animal.' lib. viii, cap. 25.

another. Dr Elliotson mentions in the 'Lancet' for 1838 that a laundress who washed the clothes of one of his patients contracted it. In at least one case, infection has taken place through a cut received in examining the body of a person who had died of the disease. It is also inoculable from man to the lower animals; goats and rabbits are said by Bollinger to be the most suitable subjects for experiment. Oxen are stated not to be susceptible, but, next to horses, it is especially apt to affect asses and mules. It is seen in hot as well as in cold climates.

A point of some importance is that grooms are sometimes attacked with glanders when they are not aware that any of their horses have been diseased. Bollinger seems to have satisfactorily explained this, by showing that in the horse glanders sometimes fails to present its more characteristic symptoms and attacks the lungs and the air-passages only.

In man the disease occurs in two forms, of which one is termed acute and the other chronic.

1. In *acute glanders* the patient is first attacked with malaise, headache, and pains in the limbs. There is often no initial rigor, and for a time there may be no marked pyrexia. At this stage the disease is generally mistaken for acute rheumatism or for enteric fever. But if a wound or scratch has been infected with the virus, the part commonly becomes red, swollen, and very painful; and an erysipelatoid inflammation may diffuse itself over a wide area, affecting, for example, the whole side of the face, or the hands, and part of the forearm. In such cases the diagnosis generally inclines towards septicæmia or pyæmia from a poisoned wound. In other instances, as in that of a man who died in Guy's Hospital in 1863, the first complaint is of pain in the side with dyspnœa, so that acute pleuro-pneumonia is suspected.

Presently symptoms appear which to an experienced eye reveal the nature of the disease. One of these is an *eruption*. Upon the limbs and the body there arise papules, which rapidly pass into flat vesicles and then into bullæ or pustules, attaining the size of peas or even of sixpenny-pieces. They become depressed in the centre, and rupture, allowing a thin purulent fluid to escape, which is often blood-stained. With regard to their histology, von Wyss has ascertained that the papulation begins as a local inflammatory change in the papillary layer of the cutis. When an incision is made through a pustule after the patient's death, its floor is found to consist of an ashen-grey, infiltrated layer. The eruption sometimes appears within twenty-four or forty-eight hours after the patient is first taken ill, sometimes not for a week, or even longer. It is, I believe, quite characteristic of glanders, although it might be mistaken at an early stage for smallpox or chicken-pox, and perhaps later on for pemphigus. Generally there is mixed with it an affection which may be taken as the representative of "farcy" in horses. This consists in the formation of hard painful lumps or swellings in the subcutaneous tissues and muscles; they more or less rapidly suppurate, and if they are incised they may ulcerate, so as to expose tendons or bones beneath. It is curious, however, that in most cases the lymph-glands fail to become enlarged. The most marked instance to the contrary that I have met with is one recorded by Travers in his work on 'Constitutional Irritation.' Not only was there suppuration of the glands of the arm originally affected, but those at each angle of the lower jaw and in the groin are also said to have been swollen. The joints in glanders not infrequently become inflamed, and pus is effused into them.

The other characteristic symptom is an affection of the mucous membrane of the nose and of adjacent parts (*ozæna*), representing what in horses is termed "glanders" in the narrower meaning of the term. First a thin whitish mucus runs from one or both nostrils; afterwards it becomes purulent, blood-stained, and fœtid. The nose itself now becomes swollen, red, and very painful, and inflammation may spread from it towards the forehead or over the cheeks. If an opportunity arises of examining the parts after death, the lining of the nasal passages is found to be ulcerated, and the septum may even be necrosed. It is to be observed that in the human subject a discharge from the nose is by no means always present in glanders; Hauff observed it in only thirty out of seventy cases which he collected. It is often not an early symptom, but appears in the second or the third week. Thus, during the first days of a doubtful case, the fact that the nose is healthy must never exclude the diagnosis of glanders.

Other mucous membranes are also affected. The conjunctivæ may be severely inflamed, and the eyelids may become greatly swollen. Virchow relates a case which came under von Graefe as one of acute exophthalmos, and its real nature was not suspected until after the autopsy. Sores may form in the mouth, and the gums may become spongy. The pharynx and the palate may ulcerate or become covered with a kind of false membrane. Sometimes ulcers form in the larynx, producing hoarseness, and an œdematous laryngitis may set in, so that tracheotomy may be required. Symptoms of bronchial or of intestinal catarrh may be present. Pustules and sores, which might easily be supposed to be chancrous, are said by Virchow to occur on the glans penis.

The general condition of the patient becomes worse from day to day. The pulse is generally much accelerated, but sometimes it remains slow. The temperature rises irregularly until it may reach 104°. The tongue becomes dry and brown. Albumen appears in the urine, and sometimes lucin and tyrosin. Delirium and sleeplessness pass into stupor and coma. Finally there are symptoms of collapse, with involuntary escape of the urine and fæces. Death usually occurs towards the end of the second, or in the third week. Sometimes the disease has ended fatally within a week, or even in three or four days, sometimes not for four weeks. Bollinger gives one case of recovery among thirty-eight which he collected. All that can be done in the way of treatment is to sustain the patient's strength with quinine, tincture of iron, nourishing food, and stimulants.

2. *Chronic glanders* is much more insidious. It is often characterised by the formation of intractable ulcers with thick livid edges. In the museum of Guy's Hospital we have two models of such ulcers, the part affected being in one case the back of the hand, while in the other case there are separate sores upon the forehead, the lobule of the ear, and the side of the face. In other instances, abscesses form about the joints and give rise to fistulous sores; or inflammatory swellings appear on the limbs, beneath the skin, or in the muscles. Or, again, there may be an eruption of pustules like those which are seen in acute glanders, but more slowly developed. The nose often remains free, but sometimes there is an erysipelatoid redness of the skin or a fœtid purulent discharge from the mucous surface, beginning perhaps after two or three months have passed. Bollinger says that the root of the nose may even become gangrenous. The nostrils are often blocked with dark-coloured crusts. Such an affection is very apt to be regarded as syphilitic, or to be classed under the vague head of ozæna. It is of great

importance to remember that the diagnosis may be cleared up by inoculating a goat or a rabbit with some of the discharge. This practice was adopted in some of the earliest cases of glanders that are known to have been observed in this country, those which were recorded by Travers. Bollinger gives details of two such experiments which he performed with matter taken from horses; the inoculated animals, which lived two or three months, showed characteristic symptoms and lesions. In a case at Guy's Hospital in 1866, the late Mr Poland introduced pus from the patient's face beneath the skin of the shoulder and set up pustules there, but I do not suppose that this result was of any diagnostic significance. Virchow mentions that at Würzburg a man was for six months in the hospital on account of refractory sores upon the limbs. At the autopsy lesions characteristic of glanders were discovered. It was not then known that the disease existed in the neighbourhood, but inquiries were set on foot and led to the discovery of an epizootic prevailing over a wide area among the horses used for towing barges on the River Main.

In some cases the chief symptoms of chronic glanders are cough with sanguineous expectoration, hoarseness, pyrexia, and emaciation, so that phthisis may be suspected.

The average duration of chronic glanders was found by Bollinger to be four months; sometimes it was not more than two or three months. It may end by passing into the acute form of the disease. Among thirty-four cases which he collected from various sources, including many of slight severity, there were seventeen recoveries. As might have been anticipated, the convalescence is generally very slow and is sometimes incomplete. He was himself acquainted with a veterinary surgeon who, at the end of eleven years, was still cachectic, and was troubled with cough and other symptoms due to the presence of cicatrices in the larynx and in the nose. But he says that some patients, after a severe illness lasting for months, get quickly well and regain the flesh that they have lost.

Bollinger thinks that carbolic acid and iodide of potassium are the internal medicines most likely to be useful; the Messrs. Gamgee speak of arsenic in combination with strychnia as having sometimes exerted a remarkable influence. Ulceration of the interior of the nose should be treated with injections of creasote water, or a solution of carbolic acid, and the application of nitrate of silver or tincture of iodine may be of service.

Contagium.—Bollinger failed to find bacteria in very careful examinations of the fresh blood of glandered animals and of fresh nodules from their bodies. Gerlach stated that the virus is not destroyed by putrefaction. Recently Löffler has ascertained the constant presence of minute rod-shaped organisms in the "farcy buds," lungs, and spleen of animals dying of glanders. He has succeeded in cultivating this bacillus, and inoculation with the pure blood has reproduced the disease in horses or asses, and also in guinea-pigs, rabbits, and mice. His results agree with those of Schütz, Bouchard, and other observers, and are now admitted ('Deutsche med. Wochensch.,' 1882 and 1883; 'Revue Médicale Française,' December, 1882; 'Microparasites in Disease,' New Syd. Soc., p. 387, and pl. vi).

The means of diagnosis now adopted in cavalry stables is to inoculate an ass with the mucus from the nostrils of a horse suspected to be glandered.

Morbid anatomy.—Many of the *internal lesions* which are found in cases of glanders in man appear to be the result of pyæmia or septicæmia, which often arises secondarily, just as it does in erysipelas; but nodules and more

or less extensive patches of catarrhal pneumonia, which have no pyæmic characters, are also seen. Virchow placed glanders, in association with syphilis and lupus, among the "granulation tumours." But he himself states that in the human subject the process concerned in the formation of the pustules and abscesses has little to distinguish it from a simple inflammation. In animals both he and Bollinger describe the affection of the nasal mucous membrane as beginning with the development of minute miliary papules, and in the lungs they speak of nodules of various sizes, some almost exactly like tubercles, others as large as peas, or even as walnuts. In man, Virchow says that on careful examination characteristic small yellow bodies may be seen in the mucous membrane of the nose, of the frontal sinuses, and even of the larynx and of the trachea. In von Graefe's case he detected them in the choroid of the eye. Von Wyss has since observed them in the gastric mucous membrane. It is interesting to find in one of Elliotson's cases, recorded in the 'Med.-Chir. Trans.' for 1833, a description of similar appearances. "On cutting into the various tumefactions on the head, trunk, and extremities, they were found to be full of pus, underneath which, in many, a number of small white granules were seen, and others, in several instances, were closely attached to the periosteum and perichondrium. The frontal sinuses contained a jelly-like mucus, and a number of similar granules, and on the septum narium was one ulcer exactly like those I have seen in the nostrils of glandered horses, and upon it lay a cluster of granules. Two of these large white circular elevations were found immediately below the sacculi laryngis; Mr Youatt, who was present, called them true glandrous chancres. About an inch below the valve of the colon, for three inches in extent, upon the whole of the surface, were white granules exactly like those in other parts."

In a case which occurred at the Middlesex Hospital in 1872, and which is recorded in the 'Med. Times and Gaz.' for that year, Dr Coupland discovered an acute inflammation of the spinal cord.

Foot-and-Mouth Disease.*—In striking contrast with the dangerous epizootic diseases hitherto described is one which in cattle seldom causes more than a transitory illness, and which when communicated to man generally produces but trifling effects. It is termed the "Foot-and-Mouth Disease." Some writers have unwisely appropriated to it the mediæval name of *murrain*, which is so familiar from its being used in the English translation of the Bible. On the Continent the complaint in question has been known for centuries, but it is said to have been first introduced into England in 1839. In 1869 it prevailed here extensively, and in 1871 it is said to have affected as many as 700,000 animals. It occurs chiefly in cattle and in sheep; pigs are also liable to it, but probably only as the result of infection by feeding upon the milk of diseased cows. It is very rarely seen in horses.

In cattle, the foot-and-mouth disease is attended with a moderate degree of pyrexia. One of the most marked symptoms is the formation of vesicles and bullæ upon the mucous membrane of the mouth, including the lips and the tongue; these parts also become swollen, and there is a dribbling of saliva. The vesicles soon break, and their floors become covered with a thick grey layer. A somewhat similar eruption appears upon the feet, round the borders of the hoofs, and in their clefts. The vesicles then pass

* *Synonyms.*—Aphthous Fever—Aphtha Epizootica—Glossanthrax—Eczema Epizooticum.

into pustules and dry up into crusts. They give the animal pain in walking or standing, so that it generally remains lying down. Another seat for the vesicles is upon the udder and the teats, which may in consequence become raw and excoriated. But within twelve or fourteen days all the effects of the complaint pass off. It almost always ends in recovery, except in calves. Of these from 50 to 75 per cent. die, probably because the milk yielded by their mothers (when they also are affected) has an irritant action upon the alimentary canal, beside conveying the specific virus.

One may well be surprised that, if foot-and-mouth disease is capable of transmission to man at all, it should not be frequently transmitted. Yet in the 'Brit. Med. Journal' for 1867 will be found a list of only twenty-two notices of such an occurrence, which were all that Prof. McBride could find recorded during the previous thirty years; and most of these were taken, not from medical journals, but from the 'Veterinary Review.' It might therefore be supposed that human beings are but little susceptible of it. But if so, it is curious that two foreign observers, Hertwig and Jacob, should each have readily succeeded in infecting himself experimentally by drinking the milk of diseased animals.

The contagium appears to reside in the fluid contained in the vesicles. By cultivating in agar-agar, in broth, and in milk (which it turns acid, but does not curdle), Dr Klein has succeeded in isolating an apparently distinct micrococcus.

Conveyance.—There are two ways in which the complaint may be communicated to man. One is by the direct *inoculation* of the specific virus into a crack or sore place, generally on the hand or on a finger. This is especially apt to happen to a person engaged in milking a cow with the eruption upon the teats, but sometimes saliva or fluid from the mouth conveys the contagion to a veterinary surgeon who is attending to the sores there; and a butcher is said to have taken it by holding his knife between his lips while dressing an infected carcass.

The other way is by *drinking the milk* of a diseased cow. This is far more important, not only because it concerns children, who suffer much more than adults, but also because it is very likely to be overlooked, at least by persons who are not conversant with the symptoms observed under such circumstances. As to the characters of the milk which is yielded by cows suffering from the foot-and-mouth disease, there have been some discrepancies of statement. Sometimes it is offensive to the smell and the taste, and it may even be mixed with blood or pus in considerable quantity; but this seems to be only when the teats are severely excoriated, or when the mammary glands themselves have become inflamed as the result of an undue retention of their secretion. In other cases it is less obviously altered; and then one of its most marked peculiarities is a tendency to coagulate when boiled, or even at a much lower temperature, forming innumerable little flocculent masses which float in a bluish whey. Sometimes it seems to have a yellowish tint, and when examined microscopically displays granular cells like those of the colostrum. But sometimes it is said to have a perfectly natural appearance. It seems generally to be diminished in quantity by about one half. There is no doubt that it is very often drunk with impunity, not only by adults, but even by children. In any case boiling may be assumed to render it innocuous.

Course.—The *incubation* of the "foot-and-mouth" disease in man is said to be from three to five days. At the end of this time slight pyrexia arises

with loss of appetite. A sensation of dryness and heat in the mouth is then noticed, and vesicles quickly appear upon the inside of the lips, on the tongue, and sometimes upon the fauces and hard palate. They reach the size of peas, their contents become opaque and yellowish, and in from one to three days they break, forming shallow, dark-red ulcers. There is some pain, which is increased by mastication, by swallowing, and by talking. The lips become swollen, and mucus and saliva are poured out in excess. In a case related by Mr Briscoe, of Chippenham, in the 'Brit. Med. Jour.' for 1872, the tongue swelled until it protruded for two inches and a half outside the mouth, and it was so firmly wedged between the teeth that for thirty-six days the patient took no food except milk; there was great dyspnœa, and suffocation appeared at one time imminent; sloughs peeled off the tongue, and the discharge became very fœtid. Such a condition, however, is altogether exceptional. There is often slight disorder of the digestive organs, indicated by diarrhœa and abdominal pain.

In some patients an eruption appears upon the fingers, especially round the nails. It consists of small clear vesicles which pass into pustules, and sometimes run together. It very rarely happens that the inflammation is sufficiently intense to cause shedding of the nails. The feet are seldom affected, but sometimes vesicles have been observed between the toes. Mr Amyot ('Med. Times and Gaz.,' 1871) mentions the case of a woman in whom the feet "became hot and covered with painful tubercles." It is said that the female breasts have occasionally presented tubercles, and even the face and other parts of the body. In Mr Hislop's case ('Ed. Med. Jour.,' 1863), in which there was a red scaly rash upon a farmer's forehead, and upon the body and lower limbs of his wife, cured by bichloride of mercury and iodide of potassium, I must confess that it seems to me doubtful whether foot-and-mouth disease was really present, or syphilis.

Diagnosis.—The eruptions, however, for which the foot-and-mouth disease seems most likely to be mistaken are varicella and slight smallpox. The affection of the lips and tongue might be set down as a simple aphthous stomatitis. Indeed, it is not impossible that a slight catarrhal inflammation of the mouth, with a little soreness of throat, may be a far more common result of infection by diseased milk in infants than has hitherto been suspected. A doubtful case might always be cleared up by the inoculation of a sheep which has not had the disorder already.

Prognosis.—The duration of the foot-and-mouth disease in man is usually from ten to fourteen days, but sometimes not more than a week. A case fatal by septicæmia has been recorded, but with that exception death has occurred only in very young children.

The sole *treatment* required is the application of a solution of borax to the mouth, or of the solid nitrate of silver to any painful ulcers; the eruption on the fingers may be dealt with like a mild eczema.

Actinomycosis.—Of late years a fourth contagious disease of the lower animals has been recognised in man.

The disease was long known among cattle as a suppurative inflammation, usually affecting the tongue or jaw, occasionally the skin or the lungs (*e. g.* Rivolta, of Pisa, in 'Virchow's Archiv,' 1875, vol. 88, p. 309). It was called wooden-tongue by the herdsmen, scrofula and osteosarcoma by veterinary surgeons. The vegetable parasite which gives rise to it was first described in cattle by Bollinger in 1877, and named Actinomyces (*i. e.* ray-fungus).

In man it has probably been often mistaken for tubercular nodules and caseous inflammation, or for sarcomata, in the liver, lungs, and other organs.

The first two undoubted cases published were by Dr James Israel, of Berlin, in 1878,* one a man of thirty-nine, with multiple abscesses and serous inflammations, and the other a man of thirty-six, with a submaxillary abscess, who recovered. In both the characteristic nodules and the "club-shaped or pear-shaped bodies" are unmistakeable (pl. i, fig. 2, iii, fig. 5). In his article he publishes notes of a third case observed by Professor von Langenbeck at Kiel in 1845 of vertebral caries in a man; and the drawings then made and reproduced by Israel (pl. iii, fig. 9, *a* and *c*), prove that it was Actinomycosis. Ponfick first recognised these *Mycosen* to be the Actinomyces discovered in cattle by Bollinger. He has since published a monograph on the subject.†

A remarkable case has just been brought before the Hunterian Society by Dr F. Charlewood Turner (March, 1887). Here it was combined with true tubercular disease, probably of independent origin.

Numerous cases in the jaws and tongue of cattle are in the Museum of the Royal College of Surgeons (Nos. 2254, *b*, *c*, &c., and 2274, *b*, &c.).

The remarkable peculiarity of the disease is that each inflammatory nodule or abscess has in its centre a parasitic fungous growth, which varies from the 100th to the 10th of an inch in diameter. In one of the earlier cases observed in this country, a section from the morbid tissues of the tongue of an ox was shown the editor by Professor Roy, at the Brown Institute. The large size, glittering aspect, globular shape, and surrounding mass of radiating club-shaped structures distinguished the organism at once from any known morbid product, and suggested the presence of lime salts or some other crystalline material. This, however, is not the case.‡

Actinomyces, as the fungus was named by the botanist Hartz in allusion to its radiating gonidia, is not one of the group of Schizomycetes, to which the micrococci, sarcinæ, bacteria, bacilli and vibriones of anthrax, glanders, and other specific contagious febrile diseases belong. It is apparently one of the Hyphomycetes, the group to which moulds like *Penicilium glaucum* and *Mucor* belong. The centre of each minute yellowish nodule consists of immense numbers of interlacing threads, apparently mycelium.

How it gains an entrance to the organism is not known, but when seated in a living tissue each mass reproduces by gemmation, and so spreads in its neighbourhood. As it grows, a zone forms around it of large nucleated cells not unlike giant-cells in appearance. These are surrounded by leucocytes (inflammatory or exudation corpuscles), and these again, in the larger and older specimens, by a capsule of spindle-cells or fibrous tissue. The inflammatory nodules thus formed coalesce and gradually form large tumours. While still scattered and small in the lungs they much resemble tubercles. The characteristic rosettes of club-shaped glistening cylinders stain strongly with eosin, or magenta and picric acid.

As we should expect from the botanical character of the parasite, it does

* 'Virchow's Archiv,' 1878, vol. lxxiv, p. 15, "Neue Beobachtungen auf dem Gebiete der Mykosen des Menschen"—'Klin. Beitr. zur Kenntniss der Actinomycose des Menschen,' Berlin, 1885. Translated in abstract for the New Sydenham Soc. in their vol. on 'Microparasites in Disease,' by Dr Hime, of Bradford, 1886.

† 'Die Actinomycose des Menschen,' 1882.

‡ See Israel's original paper above quoted, where it is stated that Dr Ferd. Cohn, who recognised the fungus, at first thought them to be fat crystals; also the discussion on Dr John Harley's case of so-called "Actinomycosis of the Liver," 'Med.-Chir. Trans.,' 1886, p. 135, and 'Proc.,' N.S. vol. ii, p. 20.

not infect the blood, lymph, and tissues like bacteria and their allies, but acts as a local irritant, spreading slowly, and limited in its action to the tissues and organs successively affected. Pathologically, therefore, actinomycosis does not belong to the same group of acute febrile infective diseases as anthrax and glanders. It belongs to a group well represented in the pathology of the lower animals, of which the *pébrine* of silkworms is a well-known example. The *Saprolegnia*, which causes salmon disease, is another. Grobe, Loch, and other pathologists have produced artificial "mycosis" of the kidneys and other viscera in the rabbit by injection of spores of *Aspergillus*. In Favus and Ringworm, as in Thrush, the parasite is a fungus (probably in all three cases a species of *Oidium*), but owing to its not penetrating the skin, or mucous membrane, its effects are only superficial. It is possible, however, that cases may hereafter be recognised in man of other internal diseases due to invasion of parasitic fungi, and therefore analogous to actinomycosis.*

There are about forty cases (some perhaps not certainly genuine †) now recorded of this disease in man. Most, as in cattle, affect the mouth and jaw, many affect the bronchi and lungs or the intestines and peritoneum, and several affect the liver only. Two of the last group were described by Mr Shattock in an excellent paper in the 'Pathological Transactions' (1885, p. 254), one of them an old specimen from the museum of St Thomas's Hospital; and a third is Dr John Harley's case referred to above.

The fungus differs considerably in some recorded cases in man from the characters described in cattle. Whether these are specific differences or depend on a stage of development or some other modifying course remains to be seen. (Compare Dr Theodore Acland's figures 'Path. Trans.,' 1886, pl. xxv, with those in Dr Harley's case, and both with Israel's, Ponfick's, and Crookshank's drawings.)

Where the tumours or abscesses are accessible to surgical treatment, actinomycosis may be cured both in animals and in man. Hence the best prognosis is when the disease affects the tongue, jaws, or neck, and the worst when it affects the lungs or the liver.

The mode of origin of Actinomycosis is unknown. It is remarkable that it has never been traced to direct contagion from cattle, and it is possible that both cattle and man derive it from some vegetable source, and that it is introduced through carious teeth, or directly from the mouth, intestines, or bronchi.

The fungus is difficult to cultivate out of the body. Recently, however, Boström is said to have succeeded, and his results throw doubt on its position among Hyphomycetes.‡ Inoculation has generally failed, but Ponfick has succeeded in reproducing the disease in calves.

* In Dr Paltauf's fatal case of *Mycosis mucorina* in man, the fungus invaded the body through the intestines, and led to abscesses in the lungs, brain, and other organs ('Virchow's Archiv,' vol. cii, with references to fungi in the air-passages, &c., p. 543).

† One, for instance, tabulated by Israel, is Mr Treves's "Case of Supposed Actinomycosis" (as he rightly called it), which proved to be large-celled alveolar sarcoma, 'Path. Trans.,' 1884, p. 356.

‡ 'Trans. German Med. Congress of 1885' (Crookshank's 'Bacteriology," p. 217).

DISEASES OF THE NERVOUS SYSTEM

Local or regional diseases compared with general and specific diseases—Principles of their classification and nomenclature—Order to be followed in this work. Nervous Diseases—Peculiarities of this group—Arrangement adopted.

THE group of specific fevers of which we have now concluded the survey is the only one which can be called "natural." They agree in ætiology, and in the nature of the disturbance set up by the exciting cause, *i. e.* in their pathology; they agree in the important character of being infectious; in their general symptoms and course, and particularly in their relations to time; in their protective power; and, lastly, they are usefully studied together from the practical points of view of diagnosis, prognosis, and treatment.

Even in this group, however, we find that some members are more closely related than others—the eruptive fevers, for instance; and some are placed together from such accidental characters as being derived from the lower animals, or being confined to tropical climates. Moreover, as in all natural classifications, certain members of the group, as Syphilis, Ague, Actinomycosis, are wanting in some of its characters, and might, not without reason, have been excluded, while other specific febrile diseases have been postponed to later chapters, as Mumps and Whooping-cough, chiefly for practical convenience, or, as Rheumatic Fever, partly in order to group them with other diseases, probably very different in origin and pathology, but closely connected in clinical history. Lastly, some of the general diseases already treated of are in important respects local—witness Enterica and Diphtheria—while others, counted among local disorders, undoubtedly affect the whole organism, as Gout and Purpura, Leprosy and Pneumonia.

It is desirable to dwell on these considerations in order that the student may clearly understand the futility of attempts at a scientific nosology. Diseases are not natural objects. They are structural or functional disturbances, viewed from the human standpoint of life and death, comfort and pain. They are often dissimilar in origin and nature, and incapable of uniform classification.

No one system of Nosology can produce a useful, a scientific, or even a symmetrical result.

If we study the natural history of diseases in their *origin* we should form certain large classes which would express true relations as far as they go, but would be impracticable for adoption in a systematic treatise. For instance, we should collect together :—contagious disorders; malarial diseases; those directly the result of mechanical injury, of chemical irritants, or of heat, "traumatic" as they are sometimes called; those produced by exposure of the surface to chills, the "catarrhal," or, as some German writers most

unfortunately call them, "rheumatic" diseases; those produced by chemical poisons ingested, as lead colic, lead palsy, and saturnine gout, the zinc-ague of brassfounders, mercurial tremors and stomatitis; those produced by drugs, as urticaria from copaiba; those produced by unwholesome food, as alcoholic delirium and cirrhosis, gout, and the long list of dietetic disorders.

Then would come the diseases of imperfect development, in intra-uterine life or in infancy, at dentition or at puberty, and the large and important series of morbid changes which are the result of premature or normally supervening senile changes—atheroma, fatty and fibrous degeneration, involution of the uterus or the ovaries or the breast; atrophy of certain organs and hypertrophy of others. These, when delayed to the full term, are the natural diseases which lead to a natural and kindly death.

If we attempted a uniform classification by *ætiology* we should place near to the specific febrile diseases produced by the access and propagation within the body of a bacterial microphyte, those disorders which are produced by a vegetable parasite propagating on the surface—ringworm and aphthæ.

If we attempted a *pathological* classification we should range with the specific fevers those maladies in which the same physiological process of pyrexia is excited by local inflammation, and make a class including contagious and inflammatory fevers, as was done by Cullen and by Hildenbrand.

A *histological* classification would be impracticable as the basis of a complete system, yet it is instructive to remember that serous membranes, lymphatic organs, mucous surfaces, bones, and secreting glands have points in common under morbid as well as under healthy conditions. Indeed, we do find it convenient to group together primary diseases of the blood, diseases of the skin, and diseases of the joints.

A nosology based on *morbid anatomy* would follow the lines traced by Rokitansky and his successors, but we have only to turn to the best systematic works on the subject to see how unsuitable such a plan would be for a treatise of medicine. Of many diseases we know neither the seat nor the cause; some whose cause we know have no anatomy, and many anatomical changes have no clinical history.

The only course open to one who plans a systematic treatise like the present is, after setting aside general diseases, to describe those together which affect the most important organs—the brain and spinal cord, the larynx and lungs, the heart, the digestive organs, and so on; and this plan is followed here, as it has been by Dr Bristowe and by other recent writers. Functional diseases are classed with those of the structures which subserve the disordered functions—neuralgia with neuritis, epilepsy with cerebral diseases, asthma with bronchitis. Diseases of uncertain seat are arranged according to their most important symptom, or according to custom and convenience. Thus, exophthalmic goitre goes with disorders of the circulation, and diabetes with those of the urine. We have no satisfactory grounds for putting them anywhere else, and at least readers know where to find them.

Nomenclature.—Closely allied to classification is terminology. In the case of specific diseases with a constant origin and a definite course the best names are those which, first, are meaningless; secondly, which consist of a single word, and that short, distinctive, and euphonious; and, thirdly, can form adjectives. Syphilis and Typhus (not typhus fever) are two of the

best names we have. When, however, the disease is less definite and certain, names otherwise good become ambiguous, as Erysipelas, Diphtheria, and Pneumonia.

In choosing names we must in the end be guided by usage—

Quem penes arbitrium est et jus et norma loquendi;

but we should follow the best usage, and in this country it is in every way desirable to keep, as nearly as may be, to the official nomenclature of the Royal College of Physicians and the Registrar-General.

Some names are vernacular, like Measles, Mumps, Smallpox, Shingles, Dengue, Glanders, Ague, Gout, and these are excellent.

Others are Greek or Latin names, more or less arbitrarily defined, as Morbilli, Variola, Zona, Psoriasis, Lupus, Scabies, and these are as good or better. Or Latin names may be more or less successfully coined, as Diphtheria, Pertussis, Enterica, Equinia, Purpura, Leuchæmia.

Personal names are undesirable. They are seldom generally accepted; they are cumbersome, they are often ambiguous, and they are seldom historically accurate. Such are Pott's disease, Morbus Maculosus Werlhofii, Bell's palsy, Maladie de Raynaud, Graves' or Basedow's disease, Cruveilhier's paralysis, Parkinson's disease, Hodgkin's disease, Maladie de Charcot.

When the anatomical lesion which causes the symptoms is known, it furnishes the best designation of the whole train of structural and functional disturbance, as abscess of the liver, annular cylindrical carcinoma of the descending colon, uric-acid renal calculus, cirrhosis of the liver, stenosis of the mitral valve, glioma of the middle lobe of the cerebellum, and so on. These, however, are rather diagnoses than names.

Better, perhaps, are the names which express the pathological processes which lead to the structural results, *e. g.* suppurative interstitial hepatitis, anterior polio- or nephro-myelitis, tubercular peritonitis, acute yellow atrophy of the liver.

Often, when neither the structural change nor the morbid process is certainly known, we must be content with clinical names, denoting a recurrent group of physiological events—a "complex of symptoms" the German writers call it—as Epilepsy, Chorea, Asthma, Chlorosis, Scurvy. Many such terms—Dropsy, Jaundice, Hemiplegia—have now become symptoms rather than "diseases," steps to a further diagnosis. But often no further step is at present possible; the symptom is the disease (see p. 5).

The order in which to take the various local diseases is of little consequence. From their complexity, interest, and importance, those of the nervous system are often placed first; and this will be done in the present volume. Chapters on the diseases of the Chest will follow those on disease of the Brain; then will come diseases of the Abdomen and Digestive organs generally; then those of the Liver, the Kidneys, and the Spleen, with its lymphatic allies; then diseases of the Bones and Joints, diseases of the Blood, and, lastly, those of the Skin.

In entering upon the study of Nervous Diseases, we find ourselves face to face with the most difficult department of pathology. Unlike the lungs and heart, the brain works silently, so that no auscultatory phenomena accompany its disorders. From the abdominal viscera it differs in being beyond the reach of palpation and percussion, and in pouring forth no secretions to be submitted to physical or chemical examination. Hence *the*

symptoms of nervous disease are, almost without exception, those of perverted nervous function.

There is here the same broad line of demarcation, as in the case of affections of other organs, between those which appear to be wholly functional, and those which depend upon organic changes; for the former we have a special name, that of *Neuroses.*

It will not, however, be convenient to take the various diseases in a regular ascending order, beginning with those in which no lesion of structure exists, and passing gradually to those which depend upon lesions of greater severity. For some of the most destructive affections of the nervous system present a very simple series of disorders of function, while some of the most trifling neuroses are attended with very complicated symptoms. We will therefore deal first with the former, and in such an order as may appear most conducive to their easy comprehension.

One cannot absolutely separate the diseases of the nervous system into those which affect respectively the brain, the spinal cord, and the nerves. For first, the precise seat of many of these disorders is still obscure or doubtful; and secondly, there are affections which, although they begin in one of these great divisions of the nervous apparatus, afterwards involve the rest. Still, the arrangement adopted here will, as far as possible, be to take first the diseases of the Peripheral Nerves, next those of the Spinal Cord, and lastly those of the Cerebral Centres.

AFFECTIONS OF THE NERVE-TRUNKS

PERIPHERAL PARALYSIS.—*Causes—Motor palsies of vertebral nerves—The reaction of degeneration—Sensory paralysis—Pain—Causalgia—Glossy skin and other trophic results—Diagnosis, prognosis, treatment—Paralysis of cranial nerves—Motor, of the portio dura, hypoglossal, and oculo-motor—Sensory of the trifacial and olfactory.*

NEURALGIA.—*Pathology and general characters—Trifacial neuralgia, &c.—Sciatica—Treatment of neuralgia.*

PERIPHERAL NEURITIS.—*Its history and symptoms—Histology—Causes—Prognosis and treatment.*

THE affections of the nerve-trunks may be broadly divided into two clinical groups. In one the chief symptoms are *loss of muscular power and of cutaneous sensibility;* these are commonly called the *Peripheral Paralyses*, to distinguish them from similar conditions due to diseases of the brain or spinal cord. In the other group, *pain* is the principal symptom; these affections constitute the *Neuralgiæ*. A third section is that in which paralysis and pain are both assignable to a definite and constant lesion of the peripheral nerves.

PERIPHERAL PARALYSIS.—Many points in regard to the causes and symptoms of the paralyses due to lesions of the nerve-trunks are the same, whatever parts of the body may be their seat. It is convenient to take the nerves of one of the limbs by way of illustration, and I will choose those of the arm, because the movements performed by its several muscles are more distinct and definite than those of the individual muscles of the leg, and their functional integrity is accordingly more easy of determination.

Injuries of the *brachial plexus* may be followed by loss of power and of sensation in the whole upper limb. The surgeon sees cases in which this occurs as the result of severe accidents in which the shoulder-joint is dislocated, or the humerus or scapula fractured. But similar symptoms not infrequently follow pressure upon the nerves, or an injury so slight that its occurrence, especially in a child, may be altogether overlooked. Thus Paget ('Med. Times and Gaz.,' 1864) mentions the case of a boy who, when an infant, had had his left arm violently pulled by a little brother; the limb appeared altogether powerless for a long time afterwards, and it remained permanently weak and much smaller than the other one. The employment of force to reduce a dislocated shoulder has sometimes been attended with like consequences. Other causes which have been noted are the pressure of a crutch which is too long or insufficiently padded; having the arm forcibly compressed by a cord (in some countries prisoners are confined in this manner); and, among the water-carriers of Rennes, carrying certain large vessels with handles through which the arm is passed. "Crutch paralysis" is not

uncommonly seen in hospital practice in London; and the patient is often unaware of its cause. A similar effect may also be produced by lying on one side, with the weight of the body resting upon the arm; or, more often, by sleeping with the arm across the back of a chair. Bnt in most instances of this kind, the sensibility of the patient has been blunted by some narcotic poison, or at least by alcohol; and they are sometimes spoken of as cases of "Sunday morning" paralysis, on account of the frequency with which Saturday night is chosen by the lower classes for indulgence in drink. When the cause is doubtful, the axilla and neck must of course be carefully examined, lest an aneurysm, exostosis, or some other tumour should be present. Some writers believe that the brachial plexus is sometimes affected by external cold, as we shall presently see to be the case with the facial nerve; but this appears to be doubtful. Mr Salter has related ('Guy's Hosp. Rep.,' 1868) two cases, in each of which caries of a wisdom tooth, besides causing severe pain in the arm, led also to partial paralysis of the muscles (of the nature of a "reflex" paralysis, as I shall hereafter have to define that term), so that the patients could not grasp objects with the hand, nor raise the hand to the head, nor employ it in dressing, nor hold a fork at dinner. In one instance all the symptoms vanished as soon as the tooth had been extracted and the pain of the operation had passed off; in the other instance a similar result occurred a few hours after the same treatment.

It is comparatively exceptional for the whole of the brachial plexus to be paralysed at once by any of the causes that I have been mentioning; and the loss of power may often be limited to the muscles supplied by a single nerve. In such cases the interpretation of the symptoms may be so obvious that no one acquainted with the rudiments of anatomy could possibly misunderstand them. But sometimes they are of a more recondite nature, and require a somewhat careful study.

Special motor paralyses.—Particular interest attaches to paralysis of the serratus magnus muscle supplied by the *posterior or long thoracic nerve.* That nerve, indeed, leaves the plexus at so high a point that it is never involved when the cause is the pressure of a crutch, or sleeping upon one arm. But it may be affected by a blow upon the shoulder, or any other severe injury to that part; by carrying a heavy weight upon the shoulder; or by over-exertion of the shoulder-muscles, as in the case of mowers, puddlers, cobblers, or rope-makers. Sleeping on damp ground is also said to have given rise to it, and even exposure to draughts. The principal symptom is an alteration in the position of the scapula. Even when the shoulder is at rest, with the arm hanging down, the scapula, as a whole, is drawn slightly upwards and inwards, and has its inferior angle tilted, so as to be nearer to the vertebral column than the rest of its posterior border. But what is especially characteristic is that this posterior border stands off a little from the part of the back on which it should rest, forming a "wing-like" (pterygoid) projection. These peculiarities are greatly exaggerated when the patient is told to perform certain movements. Thus if he is directed to lift his arm straight over his head, he finds himself unable to raise it above the level of the shoulder, because the necessary rotation of the scapula by the serratus cannot be effected; but when once another person has moved the bone to the proper position for him and fixed it there, he acquires the power of lifting the limb to whatever height may be desired. During the attempt to raise the arm, the scapula, if left to itself, moves further than ever towards

the spine; and where both serrati happen to be paralysed together, the two bones may actually come into contact at their inferior angles. If the raised arm is brought forwards, the "wing-like projection" becomes greatly augmented, the posterior border of the scapula standing off so as to leave a deep hollow, in which one can place one's hand. Another symptom, according to Dr Poore ('Clin. Soc. Trans.,' viii), is an alteration in the shape of the chest on the affected side, as shown by the cyrtometer. He finds that in a healthy person, when the arms are thrown horizontally forwards, the chest becomes flattened in front, and its lateral diameter is increased. When there is paralysis of one serratus the widening fails to occur on that side.

An affection of more frequent occurrence is paralysis of the muscles supplied by the *circumflex* nerve, among which the *deltoid* is the chief. This, too, may result from direct injury to the shoulder. According to Erb it is sometimes caused by "rheumatic" affections of the articulation. One must, however, bear in mind that osteo-arthritis (to which disease Erb probably refers) is commonly attended with destruction of the long tendon of the biceps, and that unless this structure is perfect the arm cannot be raised; and inability to lift the arm from the side is also the principal symptom of paralysis of the deltoid. It may be thought that a sufficient proof of the existence of paralysis would be found in the presence of atrophy of the muscle, which, indeed, is often so marked that a deep groove is formed between the acromion and the head of the humerus. But a considerable degree of wasting may be the result of mere disuse when the joint is diseased.

Paralysis of the *musculo-cutaneous* nerve is of course indicated by inability to flex the elbow-joint, the biceps and brachialis anticus muscles being paralysed. It must be borne in mind that the supinator longus (which receives its supply from another source) is also a flexor, and the patient must therefore be told to keep his hand supinated at the time when the power of the other muscles is being tested.

Among the nerves which send filaments to the hand and fingers, the *musculo-spiral* is much more apt than the other two to be separately affected. In a considerable proportion of the cases of paralysis due to the pressure of a crutch, or to lying on one side during the deep sleep of intoxication, the musculo-spinal nerve suffers alone; and as in the latter case the triceps often escapes, it is probable that the nerve undergoes compression where it is winding round the humerus, and after it has given off the branches to that muscle. The extensors of the wrist, of the two joints of the thumb, and of the metacarpo-phalangeal joints of the fingers are the chief other muscles supplied by this nerve; and accordingly one symptom of paralysis of it is a flexed condition of these joints, with inability to straighten them; the two remaining joints of the fingers being nevertheless capable of extension by the interossei and lumbricales, provided that the metacarpo-phalangeal joints are held up by another person's hand, or in some other way. Another consequence of the loss of power in the extensor muscles of the wrist-joint is impairment in the extent to which the fingers can be *flexed* upon the palm. Thus the paralysis of no other nerve interferes so much with the motions of the hand as does that of the musculo-spiral.

An isolated paralysis of the *median* (or of the ulnar) nerve is more commonly due to a wound or injury affecting it at the elbow, in the forearm, or at the wrist than to any morbid state of the brachial plexus above. The

symptoms, in the case of the *median* nerve, are inability to use the flexors of the wrist and fingers, the metacarpo-phalangeal joints being alone capable of flexion by means of the interossei. Indeed, the excessive action of these muscles is apt to cause an over-extension of the two phalangeal joints of the fingers, and especially of the index finger.

On the other hand, when the *ulnar* nerve is paralysed, the muscles of the little finger and the interossei are more markedly affected than any others. The fingers cannot be abducted nor adducted, and the patient is unable to flex the metacarpo-phalangeal, or to extend any of the phalangeal joints of the fingers. There being nothing to antagonise the traction excited by the common extensor and flexor muscles, the joints in question become distorted, so that the two last phalanges are over-flexed, while the first phalanges, on the other hand, are over-extended. Accordingly, the prominences of the knuckles are replaced by hollows on the back of the hand, while they project towards the palm, and are the more noticeable there because the interossei and two of the lumbricales muscles are atrophied. The fingers of a hand so affected are often compared to the claws of a griffin; French writers speak of it as the "*main en griffe.*"

The production of deformities, in affections of the nerve-trunks, by contraction of the muscles which are physiologically the antagonists of those that are paralysed, has not before been alluded to; but it may occur in every form of these affections, and often complicates the symptoms very considerably. On the other hand, at very advanced periods of such cases, the paralysed muscles themselves may waste and shorten, so as to produce distortions of precisely opposite characters.

It by no means necessarily follows, in all cases of peripheral paralysis, that the anatomical distribution of the nerves is strictly adhered to. Erb speaks of having seen several instances in which the deltoid, biceps, brachialis anticus, and supinator longus were chiefly, if not exclusively, attacked; and he throws out the suggestion that this depends upon an affection of the sixth cervical nerve where it is emerging from between the scaleni. In such patients the arm hangs by the side, and the elbow cannot be flexed. It is remarkable that a precisely similar affection was described by Duchenne in newly-born children, as the result of traction or pressure upon the shoulder during birth. Erb says that he has himself seen two cases of this kind.

Lastly, one must bear in mind that in exceptional cases a morbid process beginning in a single nerve at a particular spot is very apt to pass upwards along that nerve, and may afterwards extend to other nerves with which it is connected. Thus Dr Weir Mitchell relates the case of a man who received an injury to the nerve which supplies the pectoralis major; on the third day he was attacked with pain in the course of the median and musculo-cutaneous nerves, and the muscles to which they are distributed subsequently became paralysed and wasted. Another patient bruised his ulnar nerve at the bend of the elbow, and about five months afterwards the flexor muscles of the wrist became affected with paralysis, those supplied by the median nerve as well as those supplied by the ulnar. These, however, were probably examples of peripheral neuritis. In the chapter on Epilepsy I shall have to describe some still more remote effects of injuries to nerves.

The reaction of degeneration.—Cases of local paralysis due to lesions of

the nerve-trunks are all examples of "peripheral paralysis," and the electrical reactions of the affected nerves and muscles present characteristic modifications. These have been minutely studied by Erb, Ziemssen, and others in Germany, and more recently by several observers in this country.

Let us suppose, for example, that a nerve-trunk is cut across, or that a small portion of it is crushed in an injury to the upper part of a limb. The result is that throughout the whole length of the nerve and its branches below the affected spot (so far as one can test them without at the same time stimulating the muscles), its excitability undergoes a progressive diminution, both to the make and break of galvanic currents, and to the rapidly alternating faradic currents from an induction coil. In the course of the second week it is entirely extinguished, so that not the slightest contraction can be produced by the application of either form of electricity to the nerves. But the reactions of the paralysed muscles are very different. They, too, fail to respond when a faradic current is applied to them. But when a galvanic current is employed they contract much more readily than under normal circumstances to the stimulus of opening or closing. The method of determining this fact is very simple. We first ascertain what number of cells of the battery used are required to excite contractions in the muscles of the healthy limb which correspond to those paralysed; and then perhaps find that only half that number is needed to excite contractions in the muscles supplied by the injured nerve. Indeed, they can often be thrown into vigorous action by a current which is too feeble to give rise to any sensation.

Moreover, there is what is termed a "qualitative" change, *i. e.* the order in which the two poles respectively excite contractions is altered. According to Pflüger's law, stimulation depends upon rise of catelectrotonus or fall of anelectrotonus (*i. e.* in either case the passage of a segment of nerves from a lower to a higher degree of excitability); the descending current has greater exciting power than the ascending, and closing or making than opening or breaking the galvanic current. Accordingly, the order in which currents of successively increased strength produce contraction in the muscle supplied by the motor nerve stimulated is as follows:

	CC	AO	AC	CO
Weak current causes . .	c			
Strong current causes . .	C	c	c	
Very strong current causes	C′ or T	C′	C	c

Here C.C.c. means that a small Cathodal Closing contraction is produced by a weak current. The current is descending, and the stimulus is the rise (in this case from zero) of catelectrotonus on closing (making) the galvanic circuit. A.O.C. means that an ascending galvanic current produces an Anodal Opening (breaking) contraction, the stimulus being the fall of anelectrotonus (to zero). A.C.c. or A.C.C. means that making contact again with the ascending current will, if it is as strong or stronger, produce a slight or a marked Anodal Closing contraction. Lastly, C.O.c. means that breaking contact with the descending current will, if it is strong enough, act as a slight stimulus and produce a Cathodal Opening small contraction.

The rule may be shortly remembered thus: With increasing strength of galvanic current the order in which contractions appear is C.C.C., A.O.C., A.C.C., C.O.C.*

When, however, a muscle is paralysed by severance of its motor nerve in any part of its course, or by destruction of its anterior root, or of its corresponding large ganglion-cells in the anterior cornu, and when this has lasted for a week, not only is the muscle less excitable when the "motor point" of its nerve (*i. e.* the place where it becomes superficial and is most favourably stimulated) is excited by the induced (faradic) current, not only is it *more* excitable when the same point of its nerve is stimulated by the make or break of the direct (galvanic) battery current, but the above physiological order is altered.

So far from contraction at the moment of closure with the cathode (C.C.C.) being now the most marked effect, there is often as much or even greater contraction when the circuit is completed with the anode; while instead of cathodal opening contractions (C.O.C.) being the last of all to appear, they may precede the anodal opening contractions (A.O.C.). Accordingly the normal order for contractions with currents of increasing strength given in the above table is altered, and instead of C.C.C., A.O.C., A.C.C., C.O.C., we have A.C.C. before C.C.C., and C.O.C. before A.O.C., *i. e.* the anodal closing contraction is for equal currents stronger than the cathodal, and the cathodal opening contraction is for equal currents stronger than the anodal.

This is called the Reaction of Degeneration.

Lastly, the contractions themselves are slow and protracted, and resemble tonic spasms rather than the short clonic movements which occur in healthy muscles when stimulated by galvanic currents.

Very little progress has hitherto been made towards the explanation of these remarkable facts. The seeming paradox that muscles insensible to powerful faradic currents should nevertheless respond to very weak galvanic currents has, indeed, been accounted for by Neumann, who has shown that it depends upon their requiring a current to pass for some length of time before they can react to it. Momentary galvanic currents, even when of considerable strength, are found to be as inoperative as faradic currents themselves. But why the muscles should be incapable of responding to currents which are only of brief duration, and why they should be sensitive to weaker galvanic currents than under normal conditions, we cannot at present say.

It seems, however, to be established that the peculiar reaction in question, as well as the loss of electrical irritability in the trunk of the nerve below the seat of injury, are coincident with and indicative of a very remarkable series of degenerative changes which have been shown by several observers to affect the structure of the parts paralysed. Erb accordingly proposed to designate as the "reaction of degeneration" the

* The German symbols used by Erb are Ka. for Cathodal, An. for Anodal, S. (Schleissung) for closure, O. (Oeffnung) for opening, and Z. (Zuckung) for contraction. These are wholly or partly adopted by some English writers. If it were important to avoid using the same letter for different words, it might be done by spelling Cathode with a K, and using Break and Make instead of close and open. The normal sequence would then stand: K.M.C., A.B.C., A.M.C., K.B.C. But in such matters whatever is most generally used and best understood is best.

It may be added that c. means a weak contraction, C′. a strong one, and T. a tetanic contraction, that > and < mean respectively *greater than* and *less than*, and that R.D. means the Reaction of Degeneration.

presence of increased galvanic excitability in a muscle, when associated with diminished faradic excitability. Within a few days after the occurrence of any severe lesion of a nerve-trunk, the whole length of the nerve below is found to have its fibres greatly altered. Their medullary sheaths break up into fatty granules, and it is even doubtful whether the axis-cylinders remained undestroyed. At the same time the neurilemma everywhere undergoes a greatly increased development; first it becomes crowded with corpuscles, and these presently develop into spindle-cells and fibres, so that the nerve-trunk itself becomes much denser and harder than before. In the muscles, too, similar changes take place, and these have been investigated by Erb. The fibres themselves gradually diminish in size, and their striation becomes indistinct; but their nuclei multiply, and the connective material between them is crowded with cells, which after a time undergo development into fresh fibrous tissue. If the injury to the nerve should remain unrepaired, the muscular fibres waste still further and at length disappear. Thus the whole substance of the muscles is at last converted into a whitish yellow, flattened mass of dense fibrous tissue, in which no striated fibres can be discovered. It often includes in its interstices a large number of adipose cells.

The cause of these curious changes is at present unknown. The easiest way of explaining them would be to attribute them to the extension of an inflammatory process along the nerve from the seat of injury. But, as Erb remarks, they ought not then to be confined to that part of the trunk which is to the peripheral side of the lesion, nor should they be traceable through a plexus or be accurately limited to certain fibres; nor should they occur simultaneously in distant muscles, or in those which are placed in the upper part of a limb. He believes that in some way they depend directly upon the separation of the affected structures from the nervous centres.

It is important to remember that this remarkable reaction of degeneration is not confined to the paralysis from injury to peripheral nerves now under consideration. We shall hereafter see that it accompanies paralysis dependent on lesions of the motor ganglion-cells of the anterior cornua of the cord and of their homologues in the bulb and mesencephalon. If, therefore, we speak of the D.R. as characteristic of "peripheral paralysis," we must extend the meaning of the latter phrase. The D.R. and the histological changes in the nerves which follow it are indicative of *severance of the nerve-fibres affected from their trophic centres, either by destruction of these ganglia, or by severance of the nerve in its course.*

It must not be supposed that the commencement of degenerative changes in the peripheral nerves and muscles is necessarily a proof that the trunk above has undergone permanent and irremediable injury. On the contrary, these structures possess extraordinary powers of regeneration. Even after the muscles have for several weeks exhibited the "reaction of degeneration" it often happens that the connection between their nerves and the spinal cord is at length gradually restored. Erb has noticed in such cases very curious anomalies in the reactions of the nerves and muscles respectively to electrical and other stimuli. Nerves that have completely recovered the power of transmitting volitional impulses may still remain devoid of all excitability by faradic or galvanic currents; or if their regeneration should take place early, they may regain their sensitiveness to both kinds of current before the muscles have ceased to present the "reaction of degeneration." That reaction, indeed, is itself never persistent for any great length of time after

the original injury. If the nerve fails to undergo repair, and the muscle-fibres begin to disappear, their excitability to galvanic currents quickly begins to diminish, and at length it becomes extinguished; and even when regeneration of the nerve does occur, the muscles commonly display a lessened rather than an increased galvanic irritability while it is going on, and for some time after the normal conditions have in all other respects been completely restored.

On the other hand, it is only when the original lesion of the nerve-trunk is of a certain degree of severity that the "reaction of degeneration" presents itself. In the slighter forms of paralysis, such as that due to the presence of a crutch, or to compression of the brachial plexus or musculo-spiral nerve during sleep, both the muscles and nerves often retain their normal irritability both to faradic and to galvanic currents.

When a motor nerve is severed (to take the simplest form of peripheral paralysis), voluntary motion is, of course, at once and completely lost. For about the first week both muscle and nerve are less and less susceptible to all stimuli, but after a few days, while the galvanic and faradic excitability of the muscle and the faradic excitability of the nerve continue to diminish, until after two or three weeks they are entirely lost, the galvanic excitability of the motor nerve rapidly increases with the other phenomena which accompany degeneration of the nerve-fibres and atrophy with degeneration of the muscle. In cases which recover, the faradic excitability of muscle and nerve returns as voluntary motor power is re-established, and the abnormal galvanic reactions of the nerve more gradually disappear. In cases which do not recover, the reaction of degeneration continues, though in slowly diminishing degree, for many weeks or months, until at last it also is lost, and the atrophied muscle responds to no kind of stimuli.

Sensory paralysis.—Nerve-trunks possess sensory beside motor functions, and these too may be impaired by the same lesions which cause paralysis. Thus, if we still take the brachial plexus by way of illustration, we find that affections of its nerves may completely extinguish the transmission of tactile impressions from all parts of the limb to which those nerves are distributed, and may likewise render the patient unable to feel a prick or a cut, heat or cold, or the passage of a galvanic current. The resulting loss of common sensation is termed Anæsthesia, while the incapacity to feel pain is distinguished as Analgesia. They do not necessarily accompany one another. Mitchell relates two cases in which, without the slightest impairment of the sense of touch, there was great diminution in the susceptibility to pain, one patient experiencing only a slight pricking sensation when a large needle was run into the palm, and the other being scarcely sensitive to the electric wire-brush. In ordinary cases, however, this instrument is a most delicate test for the presence of minute degrees of susceptibility to sensory impressions. The part to which it is applied must be perfectly dry, so as to oppose the current passing through to the underlying muscles and confine its effects to the skin. The numerous minute points of the dry brush help to the same result. Mitchell says that if no sensation can be produced by it one may generally conclude that the loss is absolute; but that on several occasions the use of it has saved him from despairing of cases which had seemed altogether hopeless.

On the other hand, there are all possible degrees of anæsthesia—to a point at which very careful investigation is necessary to show that the

transmission of impression is not perfect. The best ways of detecting a very slight anæsthesia are to bring the tip of a feather gently upon the surface of the skin, or to touch a single hair with a needle or the edge of a knife. If the patient can feel this on one side but not on the other, sensation is to some degree impaired on the latter.

A pair of compasses may be used, as taught by E. H. Weber, the object being to determine at what distance from one another the points are felt to be distinct. This "limit of separate perception" varies greatly in different parts of the skin under normal conditions, from 2·25 mm. on the extremities of the fingers to 77 mm. on the arm. But for each region there is an average, and any very considerable departure from it may be taken as proof that some perversion of sensibility exists. Or a direct comparison may be made between the opposite sides of the body in the individual whose case is being investigated. An improved form of the instrument is sometimes employed, in which the two limbs of the compasses slide upon a graduated bar; this is called the "æsthesiometer." Mitchell teaches that, to ensure the most correct results that are attainable, one must cover the patient's eyes, keep the part which is to be tested perfectly at rest, and apply the compass-points (which should be rounded) lightly upon the surface of the skin, but with equal force, at exactly the same time, and in a line bearing a definite relation to the axis of the limb. But experience has shown that it is useless to base precise numerical statements upon Weber's method. It often happens that widely different results are obtained from the same spot at an interval of a few minutes. All we can obtain are relative results, and these are after all what we most want.

It would be a great mistake to suppose that in affections of a mixed nerve-trunk the loss of cutaneous sensation is necessarily equal to the muscular paralysis. Very often, particularly when the disease or injury is of but slight severity, there is little or no anæsthesia, although the loss of power is complete; or sensation may be quickly regained while the motor power is being slowly recovered. Probably the sensory centres are readily affected by very feeble impulses, whereas the muscles are unable to respond unless their stimulus is conveyed to them in large amounts. As Mitchell points out, this supposition accords with the well-known fact that by irritation of the ulnar nerve at the elbow one can excite sensation far more readily than motion.

I am not aware that lesion of a mixed nerve-trunk anæsthesia, unaccompanied by motor, ever produces paralysis. But a certain degree of intensity of lesion, as measured by the presence and duration of the "reaction of degeneration," will be found to be constantly attended with impairment of sensation.

Beside degrees of anæsthesia there are certain perverted sensations, conveniently grouped together as *paræsthesiæ*. These, however, are less frequent and less characteristic in cases of injury or disease of nerve-trunks than in spinal and cerebral diseases, and will therefore best be treated hereafter.

Pain.—Another symptom of affections of the nerve-trunks—and one which likewise has its seat in their sensory fibres—is pain. It is by no means constantly present, and, indeed, is wanting in most cases of ordinary peripheral paralysis. It commonly exhibits in a more or less marked degree the features which will be hereafter described as those of neuralgia, and is

referred mainly to those parts of the skin to which the diseased or injured nerve is distributed. It is sometimes due to peripheral neuritis; and the trunk of the nerve may then, in thin persons, be felt to be swollen and exceedingly tender. In some cases it is periodic, returning every day at about the same hour, generally towards the latter part of the day. There is a special variety of it, to which Mitchell has given the name of Causalgia (καῦσις, cautery), on account of its burning character. This may vary greatly in severity, up to the most unendurable agony, which the patient compares to a "red-hot file rasping the skin." In cases due to wounds, this kind of pain seldom, if ever, comes on until the process of cicatrisation is beginning. Its most frequent seat is—in the upper limb, the palm of the hand; in the lower limb, the instep. Heat aggravates it, and allowing the limb to hang down has the same effect. It is relieved by moistening the skin, and two of Mitchell's patients always carried a bottle of water and a sponge, and would not allow the affected part to become dry, even for an instant. Others found some ease in walking, by pouring water into their boots. In course of time, the severe suffering caused by this condition often leads to a general irritability of the nervous system, allied to hysteria. The patient may then complain of hyperæsthesia of the whole body, particularly when the surface is dry.

This peculiar kind of pain seems never to occur in parts which have been rendered altogether anæsthetic by the complete division of their nerves; nor does it accompany the subjective sensations that are so often referred to the distal ends of limbs which have been amputated. From these facts Mitchell argues that its immediate cause is some change in the peripheral extremities of the nerves, rather than in the trunks above.

Trophic changes.—That causalgia is due to a peripheral lesion is the more likely because the integument of the parts in which the pain seems to have its seat is liable to a special change, to which Sir James Paget first gave a name—that of "Glossy Skin" ('Med. Times and Gaz.,' 1864). The surface then shines, as though it had been varnished. Or, in Paget's words, "the fingers"—for they are most often affected—"become tapering, smooth, hairless, almost void of wrinkles, glossy, pink, or ruddy, or blotched as if with permanent chilblains." An eruption of small, pointed vesicles (somewhat like those of eczema) is also of common occurrence; or bullæ may form, which afterwards ulcerate; or painless whitlows, leading to shedding of the nails. Or, again, the nails themselves may become arched and incurved, and the skin may shrink away from their roots, leaving the sensitive matrix partly exposed.

Parts which are the seat of causalgia are slightly *warmer* than those on the opposite side of the body; whereas the general effect of injuries of the nerve-trunks is to lower the temperature; sometimes, as in some cases of Hutchinson's, by as many as eight or ten degrees Fahrenheit.

Zona or Herpes Zoster is the name given to a remarkable affection of the skin which is no doubt analogous to these results of lesions of trophic nerves. It will be considered in the last section of this work on Diseases of the Skin.

Another occasional result of morbid conditions of the nerves of a limb is a painful *swelling of its articulations* which may go on to anchylosis, and greatly increase the deformity and the disablement experienced by the patient. The arthritic effects of injuries to nerves have been well illustrated by Mitchell; they are analogous to the affections of joints which occur in certain

diseases of the spinal cord, and will be afterwards described as the arthropathies of Charcot. They are in the highest degree rebellious to treatment.

Erb also mentions as sometimes following paralysis of the musculo-spiral nerve, a thickening of the *tendons* at the back of the wrist—a circumscribed painless swelling, of the size of a hazel nut, evidently due to their having been exposed to mechanical strain, in consequence of the flexed state of the joint.

These various conditions appear to be sure indications of the existence of some disease affecting the nerves of the parts concerned. But they do not necessarily depend upon a definite lesion confined to the nerve-trunks. Paget mentions two cases in which glossiness of the fingers developed itself as the result of neuralgia after shingles. Mitchell relates one in which it was due to a chronic affection of the spinal cord. And some years ago the late Dr Moxon had in Guy's Hospital a remarkable case in which a precisely similar state of the fingers was caused by a mediastinal tumour.

Diagnosis.—With regard to the diagnosis of paralysis from lesions of the nerve-trunks I need say but little, as there is seldom much difficulty about it when once one is familiar with their various symptoms. Sometimes, indeed, progressive muscular atrophy in an early stage might be mistaken for the paralysis resulting from lesion of a nerve; particularly if the deltoid muscle should be alone affected, or the interossei and the muscles of the little finger. The application of galvanic and faradic currents to the paralysed muscles would clear up the doubt in some cases, but not in all.

Having ascertained that the paralysis is of peripheral origin, one must not omit to search for all possible causes of compression of the nerves. Some years ago a woman came to Guy's Hospital complaining of numbness and loss of power in the right arm, which symptoms (she said) had already been of six months' duration; she was found to have a cancerous growth in the breast, which had extended to the axilla and involved the nerves of the brachial plexus. One must also bear in mind the possibility that the paralysis may be "reflex," due perhaps to irritation from diseased teeth (see p. 391).

In the lower limbs the mistake which one is most likely to make is that of attributing to an affection of the spinal cord a paralysis really due to a pelvic tumour compressing the sacral plexus, or to a mass involving branches of the lumbar nerves. In 1876 a woman died in Guy's Hospital who for ten years had suffered from paralysis of the sphincters of the bladder and rectum, and for seven years from loss of power and sensation in the legs below the knee-joints, so that she was unable to walk, and could only crawl about on her hands and knees. The cause was found to be a large abscess in the pelvis, dependent upon disease of the bones, through the middle of which the cords of the sacral plexus ran.

The *prognosis* of the different paralytic affections that may result from lesions of the nerve-trunks of a limb depends upon their nature and severity. Most of the cases that come under the care of the physician get well sooner or later, and one can do much by treatment to hasten their recovery. Thus crutch-paralysis commonly passes off in a week or a fortnight; that which follows compression of the nerves during sleep more often lasts from four to six weeks, and it may be prolonged for several months; that which is caused by violent injury, or even by severe stretching or bruising of the nerves, generally lasts more than a year, and may never be entirely removed. Even

after very long periods improvement sometimes takes place. Mitchell says that only the careful notes previously made could have convinced him of the extent of restoration of function which had taken place, in some of the worst cases, at the end of three or four years.

As may be inferred from what has been stated above, most valuable aid in the prognosis of this form of paralysis is afforded by an examination of the electrical reactions of the affected parts. In cases which are to terminate rapidly in recovery, the muscles and nerves retain their normal irritability; in those which are to run a protracted course the muscles present the "reaction of degeneration." In cases which are incurable, susceptibility to every form of galvanic stimulus is altogether extinguished.

Treatment.—Electricity is useful, not only as a guide to prognosis, but also in treatment. There is, indeed, still much uncertainty as to how it acts, but all writers are agreed that it is beneficial. Sometimes its effects are extraordinary: after a single application of faradic or galvanic currents the patient may suddenly find that he can perform movements which he had for weeks or months been unable to accomplish. But in such cases it may generally be assumed that the regeneration of the affected parts was already far advanced—that they were, in fact, on the brink of recovery—independently of any treatment. At earlier periods the action of electricity is generally less striking, and any apparent success is still less capable of bearing a critical scrutiny. Mitchell, indeed, speaks strongly of the importance of the use of faradic or galvanic currents at the earliest possible moment. As regards the choice of one form of electricity rather than the other, he adheres to a well-known rule which has been laid down by several other writers, namely, that whichever current is found to produce muscular contractions most readily should be used. The idea has been that by stimulating the muscles to contract one helps them in keeping themselves well nourished, besides possibly doing something towards opening up the path for the entrance of volitional impulses. But Erb maintains that if the "reaction of degeneration" is present one cannot by galvanisation hasten the recovery of motor power, although he admits that when recovery has once begun its progress may be accelerated by such treatment.

In employing galvanisation in the treatment of paralysis from an affection of a nerve-trunk one should use the "labile" method. The anode is placed over the plexus above, or on some convenient "indifferent" spot, and the cathode is then slowly moved over the skin, covering each of the affected muscles and nerves in turn. The strength of the current should be such as to excite distinct muscular contractions, but not to cause more than a slight sensation of heat, and a little redness of the skin. Its application should be continued for about ten minutes, and at intervals of twenty-four or forty-eight hours.

If the faradic apparatus be chosen, its two poles, both transient, should be held in one hand between different pairs of fingers, and they should be placed in succession over the various muscles at a little distance from one another.

Another method of treatment on which Mitchell lays great stress is that of shampooing the affected parts, or (as it is termed) "massage." Persons have to be specially trained for the performance of this manipulation, which requires strength and endurance as well as gentleness, for it ought to be continued for an hour at a time. The limb should first be

immersed in a hot bath. Afterwards every part of the skin, so far as the paralysis extends, should be lightly pinched and tapped, and moved to and fro on the tissues beneath. Then the joints are to be moved in turn, and, lastly, the muscles must be gently kneaded and rolled, the power employed being gradually increased. Mitchell says that at the close of the sitting the temperature of the limb is raised by 1° or 2° Fahr., that the muscles show a marked increase of tone, and respond more readily to faradisation, and that the skin is sometimes intensely reddened. The patient feels refreshed and sleeps better. It should be repeated every day. A further proof of the efficacy of "massage" is that when it is carried on for too long a time it sometimes causes lumbar pain, headache, nausea, and even great exhaustion.

For persistent anæsthesia Mitchell recommends the application of a rather strong faradic current by means of the electric brush. After two or three sittings he has commonly found sensation beginning to return. He has also employed as counter-irritants rags dipped in hot spirits of turpentine and covered with oiled silk. This affects different persons very differently, causing unbearable pain in some individuals, while others scarcely feel it. In some cases he says that it is advantageous to apply irritants with due caution over an extensive surface, and he quotes the case of a man who had lost sensation in the whole arm as the result of a fall on the shoulder, and who completely recovered after his arm and back had been severely blistered by the sun.

When the affected nerve is painful and tender a bag of ice should be kept constantly applied, both by day and by night.

Causalgia may to some extent be relieved by dressings with cold water constantly renewed. The injection of morphia into the tissues of the affected part was also found useful by Mitchell, but he seems to rely mainly upon the repeated application of blisters. Sooner or later this form of pain almost invariably subsides.

Cranial Nerves.—The account which I have given of the affections of spinal nerves is applicable, *mutatis mutandis*, to those of the cranial nerves likewise. But the latter possess functions, which, when disordered, give rise to special symptoms. I shall accordingly discuss first the diseases of the *motor* cranial nerves (the facial, and the nerves of the ocular muscles, and the hypoglossal), then those of the *sensory* nerve of the face (the fifth), and lastly, those of the two *nerves of special sense* (the olfactory and gustatory), which are still left within the province of the physician.

Facial Paralysis.—The motor division of the seventh nerve,—the *portio dura*, or facial nerve,—is very liable to affections which destroy its conducting power, and are consequently attended with paralysis of the muscles to which it is distributed. The patient is then said to suffer from facial paralysis, or *Bell's paralysis*, as the disease is named, from Sir Charles Bell having discovered its seat in the portio dura (1838).

Ætiology.—Of the causes that may give rise to this form of disease the most frequent is the direct action of cold upon the side of the face. Sitting at the open window, whether of a room or of a railway carriage, sleeping near a damp, cold wall, or with the face exposed to a draught of air,—even the general exposure of the body to cold, as in a patient of Sir Thomas Watson's, who walked about the streets for some days without shoes or

stockings during a thaw,—may each of them be followed by facial paralysis. Such cases are spoken of by some German writers as "rheumatic," according to that wide use of the term which really deprives it of meaning.

It is supposed that the peripheral part of the nerve becomes affected with a slight inflammatory swelling, which (perhaps by extending into the stylo-mastoid foramen) leads to compression of its fibres. The comparative rarity of similar paralyses of the spinal nerves from cold may depend on the larger size of the intervertebral foramina, and on their greater distance from the exposed portions of the nerves. Parotitis, tumours growing in the substance of the parotid gland, swellings of the cervical glands, wounds of the cheek, and pressure of the forceps in instrumental delivery, are the chief other causes that may affect the nerve beyond the aqueductus Fallopii.

In its course within that canal the portio dura is liable to be involved in the severe forms of disease of the petrous bone: caries and necrosis; or, more rarely, tumours of various kinds. In fracture of the base of the skull, the nerve may be torn across. Dr Moxon has recorded ('Path. Trans.,' vol. xx) a case in which a clot of blood was found compressing and destroying this part of the nerve. It seems probable that a similar condition, resulting from the rupture of some small blood-vessel, was present in an oft-quoted case of Sir Charles Bell's, in which facial paralysis followed a box on the ear; and also in one, related by Sir Thomas Watson, of a man who was attacked by facial palsy within three hours of having a fall in which he struck his hip and his elbow, but not his head.

On the cerebral side of the internal auditory meatus, the facial nerve may be implicated in various tumours and other diseases of the base of the skull; or its origin in the pons may be affected by morbid processes of different kinds. As a rule, such cases are distinguished by the circumstance that other nerves are involved at the same time, or that further symptoms of encephalic disturbance are present. But I have myself met with an instance in which simple paralysis of the right facial nerve, in a man who died of granular disease of the kidneys, was found to be caused by a minute spot of softening which existed in the pons, rather to the left of its centre, with a cyst of the size of a pea. And Dr Gowers has "seen two cases of seizure, evidently apoplectic, in which the only paralysis that followed the seizure was seated in the muscles supplied by the portio dura."

Lastly, it sometimes happens that well-marked facial paralysis occurs in the course of some cerebral disease which cannot be shown to affect the nerve in any special manner. I am not now referring to the partial implication of the facial muscles in ordinary hemiplegia; still less to that which is a constant feature of bulbar paralysis; but to a case to which I shall hereafter refer, in which facial paralysis was the earliest symptom of tubercular meningitis. Again, I have myself had a patient who was attacked with Bell's paralysis twenty-four hours after the development of an eruption of herpes zoster on the same side of the face. He came to me about seven weeks afterwards; the loss of power to move the facial muscles was then almost complete, and the "reaction of degeneration" was found to be present when the electrical test was applied. The purple scars caused by the eruption were still visible. No indication of any disease affecting the seventh nerve could be discovered; and I was inclined to regard the paralysis as reflex. He slowly recovered, after the lapse of several months.

The symptoms of Bell's paralysis are very striking and present many points of interest. When the affection is developed to the fullest possible

extent, the patient is altogether unable to move the muscles supplied by one facial nerve. Whether he frowns, or smiles, or laughs, one side remains expressionless; the forehead is unwrinkled, no creases appear round the eye, the cheek and chin are marked by no dimples, the angle of the mouth remains in the same position as before. When he tries to close the eyelids forcibly, those on the paralysed side are motionless, a condition pedantically called lagophthalmos (hare's-eye); but since there is a physiological association between forced closure of the eye and elevation of the globe (for protection of the cornea from injury), the latter action, which is affected through the third nerve, still goes on; and the eyeball is involuntarily turned upwards, or upwards and inwards, until the pupil is hidden beneath the upper lid. The actions of spitting, whistling, or blowing are alike incapable of being performed; the sides of the mouth cannot be pursed up; the cheek is not held close to the teeth by the buccinator muscle, and bulges out when the breath is propelled against it. For the same reason, when the patient masticates, the food cannot be retained within the space between the jaws, and collects outside the teeth on the affected side, so that he has to dislodge it with his finger, unless, indeed, he keeps his hand pressed against the cheek as long as he goes on eating. In speaking, he pronounces the labial consonants indistinctly,—*p*, *b*, *f*, *v*, and *m*.

The degree to which the face is distorted when in repose varies considerably in different cases, even among those in which the loss of voluntary power is complete. In some patients the mouth is drawn far over to the opposite side, the eye stares fixedly, and the countenance has at all times a most comical aspect. This seems to be due to a contraction of the antagonist muscles analogous to that which causes distortion of the fingers when the interossei are paralysed. But perhaps one element in it is an absolute loss of tone in the affected muscles, which occurs only when the nerve is completely destroyed, as in cases of necrosis of the temporal bone. For I think that in most of the so-called "rheumatic" cases the patient's features are but little disturbed while they are at rest, so that at a cursory glance one may scarcely notice that anything is amiss with him. Even when he is altogether unable to close the eye, the palpebral aperture may appear but little wider than on the unaffected side, and the *tensor tarsi* muscle may still be able to keep the lachrymal puncta in contact with the globe, and to prevent the tears from running down over the cheek, as they do in the most extreme forms of the disease. It seems not improbable, although I do not know that the fact has ever been observed, that during sleep, when the levator palpebræ is relaxed, the eyelids may approach one another more nearly than would be expected from the patient's inability to close them when he is awake, and when the levator is acting in opposition to his efforts. In this way one may perhaps find an explanation of the not infrequent absence of inflammation, which one would expect to be a necessary result of particles of dust settling upon the exposed eyeball. As is well known, the conjunctiva is very apt to become inflamed under such circumstances, and the cornea may lose its transparency or ulcerate; but Valleix had a case of facial paralysis of twenty years' duration in which no such results took place. In the daytime, as Trousseau remarks, the patient is able to compensate for the failure of movement in the eyelids by bringing the globe into various positions so as to wipe it upon different parts of their inner surfaces; or he may with his finger push down the upper lid, and rub it gently over the eyeball.

Among the indirect effects of facial paralysis, impairment of the sense of

smell is sometimes mentioned; and it is attributed to a dry condition of the corresponding nostril, which results from the escape of the tears over the cheek. A much more frequent symptom is a perversion of the sense of *taste*. Trousseau, for example, speaks of a man who said that his food tasted like "salt plaster;" and other patients have experienced a subjective metallic or sour taste in the fore part of the tongue on the affected side; or a loss of power to detect acid, sweet, or saline flavours; this appears to be directly due to interruption of the conducting power of a branch of the seventh nerve, the chorda tympani. It is also said that the secretion of saliva may be diminished from the same cause. On the other hand, the sense of *hearing* is often abnormally acute; there is an augmented sensibility for all musical notes and sounds, the patient hearing a watch at a greater distance from his ear on the affected than on the healthy side; there is also an increased capacity for the detection of sounds of very low pitch; and there may be a subjective sensation of hearing an acute sound. This state of the auditory sense has been (barbarously) named "oxyakoia." Brown-Séquard propounded the theory that it is due to hyperæmia of the auditory nerve consequent on paralysis of its vaso-motor nerves. But Lucæ has since shown that it is the result of paralysis of the stapedius muscle, which receives a branch of the facial nerve; its antagonist, the tensor tympani, being no longer opposed, keeps the membrane too much on the stretch. ('Berl. klin. Woch.,' 1874, nos. 14, 16.) Attention of hearing therefore should never occur in facial palsy of peripheral origin.

Another branch of the facial nerve, which is often affected in Bell's paralysis, is that which goes to the soft palate. Dr Sanders has shown ('Ed. Med. Jour.,' 1865) that there is then "a vertical relaxation or lowering of the corresponding half of the velum, with diminished height and curvature of the posterior palatine arch," a condition which he believes to be due to loss of power in the levator palati muscle. In certain cases the uvula is turned to the paralysed side; but this seems not to be constant, and it possesses little significance, inasmuch as lateral deviation of the uvula sometimes occurs in healthy persons. If the levator palati is paralysed, it would follow that the lesion of the portio dura must be on the cerebral side of the geniculate swelling of the nerve in the petrosal bone. But the symptom is more often looked for than seen.

Diplegia facialis.—The right facial is as liable to paralysis as the left. It sometimes happens that *both nerves* are paralysed in the same patient; the symptoms are then to a certain extent peculiar, so that such cases have been dignified with the special name of "diplegia facialis." The chief point is that there is no distortion of the features under emotion of any kind. The face remains without expression, as Romberg puts it, and the patient laughs or cries behind a mask. Double facial paralysis may be caused by a new growth or a syphilitic gumma, involving both nerves at the base of the skull; but more frequently it is due to disease affecting the two petrous bones separately, or to the action of cold upon both sides of the face at once or in succession.

Diagnosis.—This is seldom difficult when facial palsy is fully developed. One must not omit to notice whether any other nerves beside the seventh are paralysed, for if such is the case there is reason to suspect the presence of some new growth or syphilitic disease at the base of the skull. Even when the loss of power is very slight and incomplete one can for the most part decide its peripheral origin; by a little failure of expression

limited to one side of the face, and by a narrow chink remaining between the closed eyelids, for these symptoms do not accompany a partial paralysis of the facial nerve from disease of the brain.

Course.—The onset of Bell's paralysis is sometimes gradual, sometimes sudden. Among hospital patients it often happens that what first attracts the patient's notice is his finding himself unable to spit or to whistle ; or he may find his face "all on one side" when he gets up in the morning, having gone to bed well.

The subsequent course of facial palsy varies greatly. When due to destruction of a considerable portion of the nerve, as in cases of necrosis of the petrous bone, it is of course permanent and incurable. And although recovery is possible after an injury, such as the division of the nerve in a surgical operation, yet this seldom occurs before the lapse of several months, and it is apt to remain incomplete. Even where the affection is the direct result of exposure to cold, it may last from four to six months ; but sometimes it gets well much more rapidly, the patient beginning to regain power over the muscles at the end of ten or twelve days, and recovering completely within a few weeks.

Prognosis.—It might be expected that one of the best indications of the probable duration of the affection would be the presence or the absence of paralysis of the higher branches of the facial nerve, the chorda tympani, and those to the stapedius and palate muscles. Sanders, however, maintained that the prognosis is not more unfavourable in cases in which the last-named branch is implicated. But although it is certain that the palate may be affected when the paralysis is the result of exposure to cold, and will ultimately disappear, yet I do not know that this symptom has hitherto been observed in cases which have recovered rapidly. On theoretical grounds, one cannot but suppose that it must indicate an extension of the morbid action along the nerve for a considerable distance. The point is not, however, of much practical importance, because we seem to have in the application of electricity a ready method of determining the gravity of an attack of facial paralysis from its very commencement. This point has been carefully worked out in Germany by Brenner and by Erb. From their observations it appears that there are two forms of the affection, one of which may be called *mild*, and the other *severe*. In the first form, which recovers in two or three weeks, the muscles and nerves of the paralysed side of the face react both to galvanic and to faradic currents exactly in the same way as on the healthy side. In only a single instance, in which a rapid recovery took place, Brenner detected a slight and transient diminution of excitability to both kinds of current. But in the *severe* form, including a large proportion of those cases which are due to the direct action of cold, the currents give rise to those peculiar effects which have been described at p. 394, under the name of the "reaction of degeneration." Indeed, it was in a case of facial paralysis that such a reaction was first noticed, namely, by Baierlacher in 1859. The prognosis is then always so far unfavourable, that no improvement is to be looked for until two or three months have passed, that a complete recovery seldom takes place before the lapse of another period of equal duration, and that more or less weakness or stiffness of the affected muscles sometimes remains during the rest of the patient's life. Erb, however, describes an intermediate form of the affection in which the excitability of the nerves is not completely lost, although the muscles present the characteristic sensitiveness to feeble galvanic currents

with absence of faradic contractility; such cases, he says, recover in from four to six weeks.

In severe cases of facial paralysis, at an advanced stage, *spasmodic affections* are observed, which present many points of interest. When the patient is just beginning to recover voluntary power over some of the muscles, it often happens that the effort to induce contraction in one of them causes simultaneous movements in muscles of a different part of the face. He may wish to move his eyebrow or his eye, and he involuntarily draws his mouth to that side; he may intend to close his lips, and he finds that he also shuts his eye. In a patient who was under my care in 1877, the distortion of the mouth was so great when an attempt was made to close the right eye, that my clinical clerk actually supposed the left side of the orbicularis oris to be paralysed. Hitzig, who has especially studied these curious phenomena ('Arch. f. Psych.,' 1872), notes that they sometimes occur while there is absolute paralysis (for all volitional efforts) of the muscles which are concerned in the associated movements. It seems difficult to avoid the inference that the motor impulse must find its way through the obstructed part of the nerve along the fibres which have regained their conducting power, and so reach the peripheral branches. But it would seem that the facial nucleus in the medulla oblongata is also in a condition of exalted irritability in many cases, for Hitzig has found that touching the affected side of the face sometimes gives rise to spasms which may even involve the muscles of the opposite side. In one case of facial paralysis, contractions actually extended to the muscles of mastication, and to those of the limbs. The occurrence of associated movements is commonly a precursor of the return of voluntary power over the paralysed muscles. They generally quickly disappear, but sometimes they seem to delay recovery, and (according to Erb) they have been known to last for as many as thirteen years.

On the other hand, when the paralysis is to be permanent, *tonic contractions* of some or all of the muscles often show themselves, which seem to have been described by Duchenne before they had attracted the notice of other observers. The result may be an elevation of the angle of the mouth, a deepening of the naso-labial groove, a narrowing of the palpebral aperture, or a general exaggeration of all the markings on the affected half of the face. The features are sometimes dragged over to such an extent that one might fall into the error of supposing that the healthy side was the one paralysed. Duchenne asserted that whenever in the course of facial paralysis some particular muscle recovers its tone earlier than one would have expected, one should be prepared for the supervention of tonic spasm in it. But this statement seems inconsistent with the fact that such spasms depend upon the occurrence of degenerative changes in the muscles themselves,—a fact established by a case of Hitzig's, in which a patient who already had contraction of the orbicularis palpebrarum from a former seizure of Bell's paralysis was attacked a second time by the same disease, without the muscle in question undergoing relaxation.

Treatment.—This is still a doubtful matter. When the discovery was first made that the muscles in many cases exhibit an augmented susceptibility to galvanic currents, most observers assumed that the application of that form of electricity would cure the disease more quickly than the faradisation which had before been employed. But in 1869, Erb, after analysing the cases which had then been recorded, expressed the opinion

('Deutsches Archiv,' vi) that the "severe" form, in which alone the reaction of degeneration is present, cannot be materially shortened in its course by galvanism. According to him, the constant current is of little value until the conductivity of the nerves is re-established, although he believes that it then accelerates the recovery of voluntary power over the muscles. He recommends that the anode should be placed behind the ear, and the cathode moved over the paralysed half of the face; or else that the two poles should be placed one over each mastoid process, the affected side receiving the anode. In mild cases he says that each application of the current is usually followed by increase of voluntary power over the muscles. It must not be forgotten that Duchenne believed that he attained still better results from faradisation; he speaks of cases which had lasted for years, and which were, without exception, cured or greatly relieved by this procedure, although all other treatment had failed. So impressed was he with its potency, that he regarded it as capable of inducing secondary spasms, and consequently advised that when the muscles began to regain their tone, the intermissions of the battery should be reduced in frequency to not more than four in the second. He did not indeed recommend that faradisation should be laid aside altogether, even when contractions of the affected muscles had set in; but he suggested that they should also be stretched with the fingers at regular intervals, or that a wooden ball should be carried in the cheek.

On the whole, however, it would appear that as much is to be anticipated from medicinal treatment as from electricity. All recent writers recommend leeches behind the ear, blisters, and mercury or iodide of potassium, as the best remedies for facial paralysis during the first two weeks. Erb, in particular, says that the iodide in full doses has appeared to shorten the duration of the disease even in some "severe" cases.

Paralysis of the Muscles of the Eyeball.—Of the movements of the eyeballs some are direct results of the action of single muscles; while the remainder, for which several muscles are brought into play, are capable of a physiological analysis far more exact than that to which the movements of any other parts of the body can be submitted. And, since three distinct cranial nerves are devoted to them, their study has very important bearings upon medical diagnosis. It might therefore be expected that the affections of these nerves, or of the muscles to which they are severally distributed, should be taken separately. But this is impracticable, because under normal conditions the two eyes are always moved simultaneously, and because the muscles of opposite sides which are associated together are often not the two of the same name, but one of a certain name and the antagonist of its fellow, receiving nerves from different sources. For example, the external rectus of one side, supplied by the sixth nerve, works, not with the *external* rectus, but with the *internal* rectus of the other side, a muscle which receives its branch from the third nerve. Now, one of the most important symptoms of loss of power in the external rectus is a deviation from the natural movements of the internal rectus of the opposite eye, and *viee versâ*, so that the affections of these muscles have to be discussed together.

At the same time it must be borne in mind that two of the ocular muscles receive each the entire distribution of a cranial nerve,—the external rectus that of the sixth, the superior oblique that of the fourth. The consequence is that either of these muscles is more likely to be separately paralysed than any single one of the rest, which are all supplied by the third nerve,

and that the affections of the former muscles are far the most important from a practical point of view. Moreover, these affections present differences in their symptoms which, *mutatis mutandis*, are typical of those which characterise the paralysis of all the other muscles; so that it is in every way convenient that I should describe them first.

Paralysis of the sixth nerve.—Each external rectus muscle is associated in its principal movement with the internal rectus of the opposite side. Their combined function is of the most simple kind; they merely carry the eyes to the right, or to the left, without any change in the direction of the vertical axes of the globes. Thus, if an external rectus be paralysed, the corresponding eye cannot be moved outwards when the other eye is moved inwards. Let us, for example, suppose that a patient has his *left* sixth nerve paralysed. The result is that he is unable to look with his left eye at any object to his left. If we hold a candle before him, and move it to his right side, his two eyes follow it until the right one has the edge of its cornea touching the external canthus, and the left has a small part of its cornea buried beneath the caruncle. But if we now carry the candle to his left side the right eye alone follows it; the left remains motionless and looks straight forwards, or may perhaps perform a slight zigzagging movement to the left under the combined influence of the two obliqui. The further the object is carried to the left the greater is the difference in the directions of the two eyes; and this *primary deviation* or *squint* at once clears up the nature of the case, when the paralysis is complete. But if the loss of power is partial the affected eye may lag behind its fellow until it has already moved some distance to the left; and no obvious squint may declare itself. In such a case, one can generally make out the nature of the defect by covering the right or healthy eye; the patient then discovers that he has not got his left eye fixed upon the object, and he accordingly moves that eye further to the left. This change in its position may itself be readily detected by a close observer; but what is far more striking is a movement which is simultaneously made by the right eye. For, the left external rectus being partially paralysed, any voluntary movement of that muscle requires the exertion of far more force than that which would have sufficed under normal conditions to produce the same degree of contraction in it. But whatever amount of force is used, it is at the same time thrown upon the associated muscle,—the right internal rectus,—which is in possession of its full vigour; and thus the right eye moves two or three times as far as the left one.

This movement of the healthy eye is called *secondary deviation;* and it is of the more importance because it affords a sure means of distinguishing a paralysis of the external rectus from a mere contraction or shortening of one or both of the internal recti, such as exists in cases of ordinary strabismus. When the loss of power is considerable, this distinction is indeed made by the fact (already mentioned) that the primary deviation increases as the object looked at is carried further over towards the left side; for if the affection were a mere convergent strabismus, the axes of the eyes would remain at exactly the same angle, the one lagging at a fixed distance behind the other, but nevertheless travelling with it, so that such an affection is sometimes called "concomitant." But in slight cases this criterion fails. Its place is then taken by the secondary deviation, the *augmented amplitude* of which is (as we have seen) dependent on the existence of paralysis. In cases of ordinary strabismus the secondary is exactly equal to the primary deviation, since the muscles on both sides possess equal power.

The other symptoms of paralysis of the sixth nerve consist of subjective sensations experienced by the patient himself, and of movements to which they lead. One is that when his eyes are turned in a certain direction he sees double images of the objects at which he looks; and this "diplopia" is not infrequently the first thing which draws his attention to the fact that something is amiss with him. We will assume, as before, that the *left* external rectus is the muscle paralysed, and we will call the image which is formed upon the retina of the affected (or left) eye "the *false* image;" the one which is formed on the unaffected (or right) eye "the *true* image." It is obvious that double vision occurs only when the eyes are directed to the left, for it is then that they fail to converge properly upon the object. And a moment's consideration will show that since the left eye has its axis turned inwards (or to the right), the false image must be formed upon the inner side of its retina, and not upon its centre; and consequently, that it appears to be *outside the true image*, or *further to the left*. Both images are upon the same level, and both are upright. By placing a piece of coloured glass before one of the patient's eyes, we can enable him to distinguish very easily which image is formed by that eye, and which by the other; reddish violet is said to be the best colour for this purpose.

It must be borne in mind that diplopia is not necessarily a proof that paralysis of any of the ocular muscles is present. Double images are sometimes formed upon the retina of a single eye; and in other cases they are due to a mere "concomitant strabismus." Making the patient look at the object with each eye separately will of course exclude the former condition; but the latter can only be dismissed from further consideration if it is found that the two images get wider apart as the object is moved further to the left. One must not assume that a diplopia which is of recent origin and which began suddenly cannot be due to a concomitant strabismus; for it sometimes happens that a patient whose ocular muscles are imperfectly antagonised is able to keep up the balance between them until he overfatigues his eyes, or until he is weakened by some illness, when he may abruptly begin to squint.

On the other hand, it would almost be a mistake to suppose that whenever there is a loss of power in the external rectus muscle the patient must necessarily notice diplopia. There is always the possibility that he may not previously have been accustomed to use the two eyes together, or that he may have been in the habit of concentrating his attention upon a single retina, in which case all that he is likely to notice is a blurring of the objects at which he looks, interfering with the distinctness of his vision. One can generally make such a person aware that he really sees double by directing him to fix his eyes upon some bright small object, especially if a coloured glass be held in front of one eye.

Another subjective symptom is that which is known as the "erroneous projection" of the visual field. We have seen that a patient who has paralysis of the left external rectus muscle refers the false image of any object to the left of him, which he sees with both eyes open, to a position outside its true position, or more to the left. And the same thing occurs if he is told to look at an object with the right eye closed; only that his judgment is then based upon an appreciation of the degree of effort required to bring his left eye to bear upon it, which effort is augmented in proportion to the failure in the power of the muscle. One consequence is that such a patient feels giddy, and may even stagger, if he attempts to use his left eye alone,

a point of some importance, because a careless observer might mistake it for a sign of serious cerebral mischief. Another result of "erroneous projection" is commonly employed as an aid in the diagnosis of paralysis of the ocular muscles. The patient is told to close the eye supposed to be unaffected, and to strike suddenly at an object placed towards the outer side of the other eye. If the external rectus muscle is paralysed he is sure to miss the object by going to the outer side of it. Yet another point is that some patients free themselves from the uncomfortable sensations to which such an affection gives rise, by keeping the head fixed over towards the paralysed side, so that the images of the objects at which they look may fall upon the outer side of the retina, and be referred to their right positions.

When paralysis of the left external rectus muscle has lasted for a considerable length of time the position of the eyeball often undergoes a further change. It is now drawn inwards by the uncontrolled action of its internal rectus, so that a convergent squint is constantly present, whatever may be the direction in which the patient looks, unless perhaps it be very far indeed to the right. Such a condition is designated by von Graefe the "secondary contraction of the antagonistic muscle;" and he has pointed out the curious fact that it is sometimes altogether disproportionate in degree to the loss of power in the muscle originally affected.

Paralysis of the fourth nerve.—The action of the *obliquus superior* is far from being as simple as that of the rectus externus; and its paralysis is proportionately difficult of detection. Modern investigations have shown that the upward and downward movements of the eyes, instead of being respectively the direct results of the contractions of the superior and inferior recti, require that the obliqui should be associated with these muscles. Each of the two recti, when acting alone, rotates the globe slightly inwards, beside raising or lowering it; but as one of the obliqui tends to rotate it in an opposite direction, the resultant of their combined action is a straight movement in a vertical plane. The left inferior rectus, by itself, would lower the cornea and carry it towards the right; the left superior obliquus, by itself, would lower it, and carry it round to the left. Acting together, they move it straight downwards. It is further evident that the rotatory action of the two recti upon the (left) cornea must be more apparent when the eyeball has been previously directed outwards, to the left; that of the obliqui when it has been previously directed inwards, to the right.

Now let us suppose that the left superior oblique muscle is paralysed. The result must necessarily be that when the patient looks downwards the cornea will be carried to the right by the inferior rectus, which has now no antagonist capable of counteracting its tendency to rotate the globe in that direction. In other words, he will squint to the right and slightly upwards with the affected eye, when it is directed upon an object towards his feet. The "secondary deviation" of the sound eye will be downwards and to the left. These symptoms, however, are comparatively little marked, so that the subjective phenomena of diplopia acquire a far greater relative importance in the diagnosis of the paralysis of the fourth than in that of the sixth nerve. The double images are seen chiefly when the patient looks downwards, and their position in regard to one another at once indicates what muscle has lost its power. Thus, firstly, the false image lies *below* the true one; secondly, it is placed to its *left;* and, thirdly, it appears to be *tilted* in a particular manner. This last circumstance depends upon the way in which the affected eye is rotated, for since the upper end of an object necessarily

has its image formed upon a part of the lower half of the retina outside the normal vertical meridian of the retina, the object itself seems to that eye to be placed obliquely *with its upper end tilted to the right.* Another point noticed by von Graefe is that the false image seems to be on a plane nearer to the eye than the true one; this, he says, depends upon the circumstance that they are both referred to positions upon a horizontal surface spread at the patient's feet, for the true image, being the upper of the two, is of course supposed to be the more distant. The writer also says that the patient, in order to avoid a sensation of giddiness when he looks with both eyes at objects before him, keeps his head turned downwards and to the right, a position which is very characteristic, because it is not one which a person is likely to adopt under other circumstances.

Paralysis of the third nerve.—Since this nerve, unlike the others, is distributed to four of the ocular muscles, the symptoms produced by paralysis of it must of course be different, according as the affection is limited to particular branches or involves all of them alike. Thus I must first describe the effects of paralysis of each muscle singly, but I need not enter much into details, for in every instance they are analogous to one or other of the two conditions already so fully described.

Thus in paralysis of the *internal rectus* the symptoms are the converse of those which belong to paralysis of the external rectus. If we take, as before, the muscle of the left side, the "primary deviation" occurs when the eyes are directed on an object towards the right; the affected eye cannot then be moved inwards while the other one is being moved outwards, and a *divergent* squint is produced. The "secondary deviation" of the right eye is towards the right. Double images are perceived by the patient when he looks to the right, and the distance between them increases the further the object is moved in that direction. They are both upon the same level and both upright. They are said to be "crossed," that is, the false image (which is seen by the left eye) lies to the *right* of the true image (which is seen by the right eye). The "false projection" of the image takes place toward the right; the patient, if he aims suddenly at an object with the right eye closed, misses it by going too far to the right. To avoid giddiness the patient keeps his head over to the right.

In paralysis of the left *inferior rectus* most of the symptoms are the converse of those which are produced by paralysis of the superior oblique muscle. As in that affection, the affected eye squints a little upwards when the patient looks downwards, but the rotation of the globe is now to the left. Diplopia occurs when the eyes are directed upon an object below them; the false image is below the true one, lies to its right, and has its upper end tilted to the left.

In paralysis of the left *inferior oblique* the affected eye squints a little downwards when the patient looks upwards; the globe is rotated to the right. The false image lies above and to the left of the true one, and has its upper end tilted to the left.

In paralysis of the left *superior rectus* the affected eye squints a little downwards when the patient looks upwards; the globe is rotated to the left. The false image lies above and to the right of the true one, and has its upper end tilted to the right.

Now, if the *whole of the third nerve* on the left side is paralysed, the symptoms are necessarily in great measure identical with those which indicate affections of the muscles severally supplied by its branches. There is com-

plete loss of power to move the eye inwards or upwards. It cannot be moved straight downwards, but it can be lowered slightly if at the same time it is carried a little outwards. In the direction horizontally outwards its play is perfectly free. These last two movements are effected respectively by the muscles supplied by the fourth and sixth nerves, and in the former of these the globe is of course made to revolve on its antero-posterior axis by the uncompensated action of the superior oblique muscle. Indeed, the rotation is so marked under such circumstances—especially when the eye is allowed to move as little outwards as possible—that it affords a striking confirmation of the modern views with regard to the action of the ocular muscles.

The visual field of the affected eye is reduced, according to von Graefe, to about one twelfth of its normal extent; it is limited by a straight horizontal line above, but below by a curved line which sweeps downwards and then outwards.

The direction of the "primary deviation" of the left eye, and that of the "secondary deviation" of the right eye, necessarily vary according to the position of the object towards which the patient directs his sight; the relations of the false to the true image of course undergo corresponding variations, and so also does the "erroneous projection" of the different objects that meet his eye on different sides of him. The consequence of this is that giddiness is a far more marked symptom when several of the ocular muscles are paralysed than when one alone is affected, and it cannot be obviated by adopting any particular posture for the head, nor, indeed, by any method except that of keeping the affected eye closed. It is only when the patient looks at an object in the extreme left of the visual field, that he ceases to squint and sees a single image with the two eyes.

But in paralysis of the entire third nerve, in addition to the affection of the four ocular muscles, there is loss of power in certain other muscles which are also supplied by that nerve. One of these is the levator palpebræ, and the result is that the upper eyelid is dropped, a condition which is termed *ptosis*. The patient is altogether unable to open the eye in the ordinary way; at most he can only slightly separate the lids by wrinkling the forehead—by means of the occipito-frontalis. Ptosis may exist without paralysis of any other muscle; indeed it is much more common than an isolated affection of any one of the other branches of the third nerve. Again, the sphincter muscle of the pupil receives filaments from this nerve, and paralysis of them leads to dilatation of the pupil, or (as it is termed) *mydriasis*. Ophthalmic surgeons state that the aperture of the iris never becomes so wide in this affection as it does under the influence of atropine; but it is often large enough to interfere very much with the accuracy of vision, owing to the formation of "circles of diffusion" upon the retina. Lastly, the ciliary muscle is also supplied by the third nerve, and *paralysis of accommodation* may be one of the symptoms of affections of that nerve. Except in very shortsighted persons, this interferes greatly with the distinctness of vision for small print and other near objects. To detect it, one must of course test the range of accommodation in the usual manner with a convex lens. The head is usually turned obliquely towards the sound side.

When all the muscles of the eyeball are paralysed together, there is immobility of the globe with ptosis, but with no squint, no double vision for distant objects. This has been called *Ophthalmoplegia externa* (von Graefe). It is usually double and progressive. More than half the cases are syphilitic and curable. The term *Ophthalmoplegia intima vel interna* is applied to para-

lysis of the iris and ciliary muscle (Hutchinson, 'Med.-Chir. Trans.,' 1878, p. 215, and 1879, p. 307).

Ætiology of ocular paralysis.—With regard to the causes of paralysis of the third, fourth, and sixth nerves respectively, our knowledge is at present imperfect. One point of very great practical importance is that almost all those cases in which the affection remains limited to a single nerve or to a single branch of the third nerve, terminate sooner or later in recovery. Their pathology is consequently unknown, but most writers think that they are either "rheumatic" (*i. e.* that they are like the common form of facial paralysis) or syphilitic. In 1876 a man died in Guy's Hospital of aortic aneurysm who had about nine months before been in another ward suffering from ptosis of the left eye and pain in the left side of the head; an old meningeal apoplexy was found. The termination of the left internal carotid artery was dilated, and its coats much thickened, and the third nerve on that side was adherent to the side of the artery and stained of a deep brown colour. Indeed, Sir William Gull used to say of the third nerve that it ran a "dangerous course," on account of its liability to compression in passing between the posterior cerebral and the superior cerebellar arteries, if these vessels should happen to become diseased; and, although I am not aware that such a hypothesis has ever been verified, it certainly accords well with the fact that ptosis is very apt to occur in old people whose arteries are dilated and tortuous. Whenever two or more nerves are simultaneously affected one must always suspect the presence of some malignant growth, or aneurysm, or gumma at the base of the skull. But of course any one of these may cause paralysis of a single nerve, and indeed, an intracranial carotid aneurysm is very likely to compress the sixth for some time before it reaches the rest. It appears to be doubtful whether syphilitic affections of isolated nerves are necessarily dependent upon an actual gumma. I have seen one case in which there was ptosis of each eye, the ocular muscles escaping entirely, and it was difficult to suppose that the corresponding parts of the third nerves were the seats of such a growth on both sides. Von Graefe is said to have traced to syphilis about one third of all the paralyses of the muscles of the eyes. We shall hereafter find that such affections may accompany various diseases of the base of the brain, and that they are often early phenomena in locomotor ataxy. But when an organic lesion of the brain or spinal cord is present one can almost always discover other symptoms, which exclude the supposition of "peripheral" paralysis of a nerve-trunk.

Prognosis.—I have already remarked that most cases of ptosis or of paralysis of the various ocular muscles get well, unless, indeed, they are due to serious organic mischief, such as aneurysm or malignant growth, compressing the affected nerve. But it must be added that recovery is sometimes slow, taking many months; and that it may be imperfect, one or more of the affected muscles remaining permanently weak.

Treatment.—When the palsy can be traced to syphilis, treatment by mercury is indicated and is usually successful. Most observers think that iodide of potassium is capable of hastening the cure, even in cases which are not of a syphilitic nature, and the application of blisters behind the ear is also believed to be serviceable. Benedikt and Erb have found galvanism very useful, a current from six to fourteen cells being applied for two or three minutes at a time, with the anode over the temple or the back of the neck, and with the cathode upon the closed eyelids. The improvement is said to be often instantaneously manifested, and Benedikt thinks that if no

good is effected within the first fortnight there is no prospect of benefit from the continuance of this plan of treatment; but according to Erb many cases require to be galvanised for several months before any result is discoverable. Faradic currents also are sometimes useful.

Paralysis of the ninth or hypoglossal nerve is a common symptom of cerebral, pontine, and bulbar paralysis, but as a lesion of the nerve-trunk it is a comparatively rare affection. It is attended with one very remarkable effect, —an extreme degree of wasting on the corresponding side of the tongue. I have seen two cases of this kind: one was in an old woman, in whom a cancerous nodule (secondary to a cancer of the breast) involved the nerve where it passes through the base of the skull; the other in a boy, who had extensive caries and necrosis of the atlas and neighbouring bones. In both instances the paralysed half of the tongue was remarkably flaccid, and wrinkled; and its tip was curved round towards the affected side. Similar cases have been recorded by Dupuytren, Sir James Paget, and Mr Fairlie Clarke. In Paget's case ('Clin. Trans.,' vol. iii) the affection was caused by necrosis of a part of the occipital bone, from an injury. Several pieces of dead bone were removed by operation; a few days afterwards the wasted part of the tongue began to grow larger, and within a month it had nearly regained its former size and muscular power. Cases of double hypoglossal paralysis are on record apart from those due to narcotic poisoning, as in the administration of chloroform, and in cyanosis, and to cerebral hæmorrhage. The tongue lies motionless and low between the rami of the mandible, and articulation, deglutition, and even respiration are impeded.

Paralysis of the fifth nerve.—Of this affection the principal symptom is, of course, anæsthesia of the face. The loss of sensation or the corresponding subjective sensations of numbness and painful tingling may in some cases be traced with greater accuracy to the median line of the forehead, nose, and mouth. A circumstance which commonly first attracts the patient's notice is that when he puts a cup to his lips he feels only half of it; it seems to him exactly as though it were broken. The eyelashes and conjunctiva are perfectly insensible; so also is the nostril, and liquor ammoniæ may be applied to it without causing sneezing; but there is no impairment of the sense of smell, except from diminished secretion of tears and consequent dry state of the mucous membrane. On the other hand, the sense of taste has in several cases been found absent in the anterior portion of the tongue. Three instances of this are given by Romberg in which quinine was used to test the gustatory powers of the patient. It is the same part of the tongue which is deprived of the sense of taste in cases of facial paralysis; and the only possible explanation seems to be that the chorda tympani is a true gustatory nerve, and that in different parts of its course it runs both with the fifth and the seventh nerves, the great petrosal nerve being probably the connecting branch.

Another effect of lesion of the fifth nerve is paralysis of the masticating muscles on the affected side. This only deprives the patient of one movement completely, that in which the lower jaw is carried forwards and laterally towards the healthy side by the external pterygoid muscle. His power of chewing food is much less impaired than might have been expected, but he really masticates only with the muscles of the unaffected side. If he is thin, one can see that the temporal and masseter fail to swell out when

the mouth is forcibly closed; even if he is well nourished one can easily feel that they do not harden.

In certain cases of facial anæsthesia the circulation in the affected parts is disordered, or their nutrition is to some extent impaired. Romberg mentions one instance in which the cheek became livid when exposed to cold, in marked contrast with the healthy hue of the opposite side of the face. Sponginess of the gums, ulceration of the mucous membrane of the nose and mouth, and hæmorrhage from their surfaces have been observed. But such changes are rare and comparatively unimportant, whereas there is another affection which is of frequent occurrence, namely, inflammation of the eye, leading to sloughing of the cornea. Physiologists have made this the subject of elaborate investigations on account of its bearing upon the question of trophic nerves. A point which seems to be established is that the inner portion of the trunk of the fifth nerve contains all those fibres which are specially concerned in the destruction of the eyeball; but there is still a difference of opinion as to whether the result is due to a mere interruption of their conducting power, or (as Charcot thinks) to some directly irritant influence. The view of Snellen that the anæsthesia is its immediate cause, the eye being exposed to mechanical injuries of various kinds in consequence of the loss of the protection afforded by the sensitive nerves, is now generally abandoned. Indeed, Charcot cites several instances in which the cornea sloughed as the result of affections of the fifth nerve, unattended with loss of sensation.

We have seen that in affections of the great nerve-trunks of the limbs, when the continuity of their fibres is not completely destroyed, anæsthesia, if present at all, soon passes off. We should therefore expect that a nerve running so protected a course as the fifth would be little liable to have its conducting power for sensory impressions interfered with except by lesions of a serious kind. And I believe that this is the case. Romberg, indeed, mentions that a watchman who had to spend his nights in a hall where the left side of his face was constantly exposed to a draught experienced violent pains in that part, and subsequently anæsthesia of the second division of the trigeminal nerve; within three weeks, under treatment with iodide of potassium, he recovered. But such instances seem to be extremely rare. The same writer relates two or three other instances in which loss of sensation in the face was due to morbid changes in the Gasserian ganglion, that structure having been found after death to be swollen, indurated, or discoloured. The nature of the change in question seems, however, to be doubtful; and we are not told why a fatal termination occurred. In the immense majority of cases of paralysis of the fifth nerve the cause is a complete destruction of the trunk or of its ganglion resulting from caries and necrosis of the bones, syphilitic disease of the pons or meninges, cancerous or sarcomatous growths, or, lastly, aneurysm of the internal carotid artery.

Paralysis of the olfactory nerve.—On account of the important contrast which exists between certain symptoms that accompany diseases of the trifacial and of the olfactory nerves, I now pass on to consider loss of smell, or, as it is termed, *anosmia*. I shall not have a more convenient opportunity, for the corresponding affections of the auditory and the optic nerves will not require notice in this work.

A man may be deprived of the sense of smell in one side of the nose, or in both. When the affection is unilateral, he is very likely to remain

ignorant of his loss, unless one is particular to test the power of the olfactory nerves separately, carefully closing each nostril in turn. We shall hereafter see that this form of anosmia frequently accompanies hemiplegia. When both nerves are affected, the patient is sure to notice the defect. His inability to perceive odours is then complete ; he is altogether insensible to the most delicious perfumes, and to the strongest stinks. But he retains his susceptibility to pungent vapours, such as those of ammonia and of acetic acid, and snuff is still capable of making him sneeze. The impressions from all these substances are conveyed to the brain by the branches of the fifth nerve which are distributed to the nasal mucous membrane ; and it is when that nerve is paralysed that they fail to excite sensations.

But, after all, the symptom which chiefly forces itself upon the notice of a patient affected with complete anosmia is not the loss of the sense of smell (as he understands it), but that of a large part of what he considers to be the gustatory sense. He can, indeed, still recognise bitter and sweet, sour and salt tastes ; and he can distinguish the rough or smooth character of the solids and liquids that he takes into his mouth. But in all other respects his sense of taste appears to be extinguished. He cannot tell one kind of meat from another ; boiled apples, boiled onions, boiled turnips, all appear the same to him ; all kinds of wine seem to have lost their flavour, tasting merely like sour or sweetish water, except that they are more or less rough to the palate. If it were not from habit and prejudice, he would probably be altogether indifferent as to the nature of his food. Evidently, therefore, physiologists are right in maintaining that the true gustatory sense is limited to four tastes, sweet and bitter, acid and saline ; and that the other perceptions which are commonly ascribed to it belong to the sense of smell.

The pathological significance of anosmia was first fully worked out by Dr Wm. Ogle (' Med.-Chir. Trans.,' 1870). He refers, in the first place, to five cases in which this condition resulted from blows upon the head, the part struck being generally, if not always, the occiput. He believes that in such cases the olfactory nerves are torn across as they pass through the holes in the ethmoid bone. But it seems difficult to suppose that all the filaments of both nerves should be simultaneously ruptured ; and I should have been rather disposed to think that the olfactory bulbs themselves had shared in the bruising of the anterior lobes of the brain, which is so common a result of injuries to the back of the head.

The remaining causes of loss of smell are independent of any affection of the olfactory nerves, but it may be well to enumerate them here. One is, perhaps, an absence of the pigment in the mucous membrane of the upper part of the nose ; at least, a case has been recorded in which a negro boy lost the power of smell more or less completely at the same time that his skin became white. Dr Ogle supposes that the nasal pigment also underwent absorption, and he cites some other evidence to show that there is a relation between the presence of this pigment and the olfactory sense. Another cause of anosmia is closure of the passage into the posterior nares, by adhesion of the palate to the pharynx ; this prevents the patient from drawing air through the nose, and so deprives him of susceptibility to odours, and also to flavours. But there are other conditions in which the one kind of impressions is lost without the other, and which therefore would correspond exactly with the popular conception of an affection of smell, independently of taste. Such cases always depend upon an obstruction to the passage of air upwards to the olfactory region through the anterior nares, while the

passage through the posterior nares is open. They generally result from thickening of the Schneiderian membrane by chronic catarrh, so that one part of the lateral wall of the nose is apparently brought into contact with the septem. Dr Ogle mentions the case of a woman who for several years had had entire loss of smell from this cause. Some years ago a lady came to me who said that for ten months she had lost both smell and taste as the result of a severe cold. I have little doubt that what she was really devoid of was the power of appreciating flavours. I prescribed some liquor arsenici hydrochloricus, and in less than a fortnight she perfectly recovered.

With respect to the loss of the sense of taste, the cases on record, which are certainly of nervous origin, are almost entirely referable to paralysis of the chorda tympani (cf. p. 404). Taste is sometimes affected on one side in hemiplegia. It is unaffected in glosso-pharyngeal paralysis.

NEURALGIA.—In the second of the two groups into which (at p. 389) affections of the nerve-trunks were divided, the principal, and sometimes the only symptom, is *pain*. This, however, is the chief sign of many other diseases likewise, and therefore it is of the first importance that one should lay down as accurately as possible the characters which distinguish neuralgic pains from those which are not neuralgic.

Now, pain itself, although it is so familiar to everyone, seems, nevertheless, to be an ultimate fact of sensation, incapable of definition or of explanation. Erb surely throws but little fresh light on its nature when he describes it as "the reaction of the sensorium to a certain degree of excitation beyond that which would cause common sensation."

Under ordinary circumstances, the impressions which give rise to pain are made upon the peripheral filaments of a nerve, and are conveyed by that nerve to the cerebral centres; and the sufferer recognises as the seat of the pain the part to which the filaments in question are distributed. Sometimes the irritation which causes pain is applied to the trunk of the nerve and not to its terminations; as, for instance, when the ulnar nerve is struck or compressed, where it passes behind the internal condyle of the humerus; but such conditions are exceptional.

In neuralgia, on the other hand, the pain is never excited by direct irritation of the distal extremities of the nerve to which it is referred.

I think that two distinct affections are included under "neuralgia." (*a*) The one is really due to peripheral irritation, but this is not applied to the painful nerve, so that the patient is mistaken in his interpretation of the local sign. This is sometimes called "reflex neuralgia." As an instance of it I may cite the trifacial neuralgia which is so often excited by disease of a tooth. Such an affection can most readily be explained by the hypothesis that the irritation is directly transferred from one nerve-nucleus to another within the cerebro-spinal centres. (*b*) In the other form of neuralgia, of which Sciatica may be taken as an example, there is every reason to believe that the morbid process begins in the trunk of the nerve which seems to be the seat of the pain. It is a form of local "neuritis" or perineuritis.

We are altogether ignorant as to the nature of the change in the nerve-trunks, or in their nuclei, which constitutes neuralgia. When sciatica has lasted for any length of time, the muscles of the affected limb are always flabby, and their strength is impaired, so that they may be said to be partially paralysed. This would seem to suggest that the state of the nerve

differs rather in degree than in kind from that which is present in the slighter forms of peripheral paralysis; and such a view harmonises well with the fact (already noticed at p. 397) that a slight injury to a nerve-trunk causes tingling and pain along its course, whereas severe pressure causes loss of muscular power, without necessarily incapacitating it for the transmission of sensory impressions. But, according to Erb, partial anæsthesia of the skin supplied by the affected nerves is present in all cases of neuralgia.

It may be thought that the two varieties of neuralgia to which I have referred ought to be entirely separated, and to receive distinct names. But this would be inconvenient, for they are really alike in many respects.

(1) In both of them the pain, instead of being referred to the peripheral extremities of the nerves alone, is felt to shoot or dart along their course.

(2) In both of them, after a time, there are constantly developed certain "tender points," pressure upon which causes great increase of the patient's sufferings, while they are often, though not always, the seat of spontaneous shooting pains. It was Valleix who first drew attention to these *points douloureux*, as he termed them; and he maintained that they are of great importance in the diagnosis of neuralgia. He further showed that they are constant in position for each of the principal cutaneous nerves; corresponding generally with the spots at which they emerge from bony canals or from fasciæ. Trousseau has since laid stress upon the presence of an additional tender "point" at the spinous process of the vertebra, beneath which the affected nerve escapes from the spinal canal; this he terms the apophysial point. With regard to the frequency of the occurrence of "tender points" in cases of neuralgia there has been much diversity of opinion. I think that this depends upon the varied scope of application of the term neuralgia by different writers. Thus Eulenburg, who includes migraine, found tender points in only about half the cases which he examined. Anstie insisted on the fact that it is only when the pain reaches a certain degree of severity and of persistence, that they develop themselves. To me it seems probable that they are after all merely spots at which nervous filaments happen to be so placed that they can be readily compressed. I have always found that in cases of sciatica the external popliteal nerve is much more sensitive to pressure on the affected than on the healthy side, in that part of its course in which one can without difficulty feel it and roll it beneath the finger,—namely, when it is running under cover of the biceps tendon.

(3) Another character of neuralgia, which is remarkably constant, and which is often useful for diagnostic purposes, is its limitation to the nerves of one half of the body. Even when it attacks a large number of nerve-trunks on the same side it comparatively seldom extends across to those of the opposite side; and I believe that it is never absolutely symmetrical, presenting the same degree of severity, and reaching the same extent of distribution, both to the right and to the left of the spinal column.

(4) A fourth characteristic of neuralgia is, that after its subsidence it leaves the affected parts tender to the touch.

According to what has been stated, neuralgia may be defined as including every pain that is not excited by irritation of the peripheral ends of the nerve to which it is referred. This obviously opens a very wide field of application for the term. The pain in the shoulder which attends upon disorder of the liver, the pain in the arm which is experienced in aortic aneurysm, the pain in the testicle which is caused by a renal calculus, are

each typically neuralgic; and so also are gastrodynia and angina pectoris, when they are not due directly to a morbid stimulation of the terminal filaments of the gastric and cardiac nerves respectively. Even the pain in the side which accompanies a hepatic abscess or pleurisy is in part neuralgic, for it often extends along the cutaneous branches of the affected nerves far beyond the area of inflammation, and the skin itself may be tender.

Nevertheless, one may fairly refuse to apply the name of neuralgia to cases in which the pain is altogether subordinate to a definite organic lesion; and also, as I think, to those in which it is transitory and (so to speak) accidental in its origin. I shall hereafter have to insist on the fact that the occurrence of a single epileptiform fit does not constitute a case of epilepsy; and so it may be said that the neuralgic pain which accompanies the passage of a renal calculus is not actually neuralgia. But it must be confessed that there is great difficulty in drawing the line as regards some other affections. For instance, *migraine*, which is regarded as a form of neuralgia by all the older writers, and even by Eulenburg and Anstie, is now described as a separate neurosis. It is, indeed, certain, for reasons that will be given hereafter that the seat of migraine is in the nervous centres themselves. But it is equally certain that the pain is referred to the cutaneous nerves of the scalp, and shoots and darts along in the course of their fibres. Moreover, in persons who have suffered from this disease for some time, true "tender points" are developed, exactly as in an ordinary neuralgia; and other trophic changes occur, which show that a morbid influence has been reflected outwards upon the hair and other superficial tissues. Thus it does not appear easy to exclude migraine from the definition of neuralgia, while the common "reflex" forms are allowed to retain their places in it. The latter, however, seem to be affections of the lowest nerve-nuclei, whereas the former probably has its seat in one of the higher centres for sensation such as the thalamus. And I think that the difficulty may be removed by the consideration that the nerves of the scalp may fairly be supposed to bear the same relation to the brain beneath, which has been shown by Hilton and by van der Kolk to exist between the superficial nerves of all parts of the body, and the organs or other structures which are covered by them. Just as almost any disease of the lung may be accompanied by pains referred to the nerves which are distributed to the skin over the chest, so we shall find that various affections of the brain and its membranes may be attended with pains running in the course of the frontal, the temporal, and the occipital nerves. The pains themselves are neuralgic, but the presence of other symptoms shows that the disease, as a whole, is something more than neuralgia.

However this may be, it is certain that neuralgia bears a very close ætiological relation to the neuroses in general: I shall more than once have occasion to refer to this when speaking of the different local varieties of the disease, some of the chief among which I must now pass on to describe.

Neuralgia affecting the fifth nerve. Trifacial neuralgia. Prosopalgia.—I have already remarked that most cases in which pain is referred to the branches of the first division of the fifth nerve belong to a separate neurosis, called migraine. But there remain numerous instances in which the lower middle branches, or even all three of them, are affected; and among them is included the most severe of all neuralgiæ,—a terrible malady for which the most convenient name is perhaps *tic douloureux*. By Trousseau it is called "epileptiform neuralgia" apparently because it occurs in paroxysms with

sudden onset, which so far resemble the attacks of epilepsy. But, as I shall hereafter explain, I prefer to use the word "epileptiform" in a different sense. It was described by Fothergill, in 1773, as "a painful affection of the face."

The absolute suddenness with which the pain of tic douloureux comes on is, indeed, one of its most remarkable characters. The patient is perhaps sitting quietly reading when he jumps up from his seat, and rushes up and down the room, with his hand forcibly pressed against his cheek. Or he may rock himself backwards and forwards in the chair, crying out or uttering deep groans. In ten or twenty seconds, or a minute at the longest, the paroxysm is over. It ceases as abruptly as it began. The pain does not always affect all the branches of the fifth nerve; sometimes only those of the second or of the third division are attacked. In some cases the muscles of the side of the face are at the same time thrown into violent spasms, so that the patient makes horrible grimaces and contortions. The paroxysms may return every few minutes. Trousseau mentions one case in which there were sometimes twenty in an hour; they sometimes do not intermit even during the night. They are often brought on by movements of the jaws, as in speaking, or in eating or drinking; and sometimes pressure upon one of the teeth will instantly excite an attack. Sometimes remissions occur, the patient remaining free from the disease for several days together, or even for months. But presently it returns, and is as severe as ever. The "tender points" are well marked in all cases of tic douloureux which have lasted for any length of time; they are situated at some or all of the numerous spots at which branches of the fifth nerve emerge from bony channels, or perforate fasciæ. Pressure upon any of them is instantaneously followed by agonising pain, and a breath of cold air upon the face may have a similar effect. Another result of the disease, when it is of long standing, is the disappearance of the whiskers or beard from the affected side of the face, the hair being worn off by the friction to which it is exposed. According to Trousseau, even the configuration of the bones may become altered, the malar eminence and the prominent part of the lower jaw being flattened down. Herpes zoster has been observed as the consequence not the cause of neuralgia and was often of the infra-orbital nerve. Spasm of the facial muscles is a rare concomitant affection. But flushing of the face and eyes with lacrymation, usually following decided pallor, is very frequently present, and shows that vasomotor and secretory fibres accompany the fifth nerve. The hair of the scalp in supra-orbital neuralgia and of the beard in infra-orbital is sometimes turned white in the region and on the side of the afflicted nerve.

A patient who labours under tic douloureux acquires an expression of intense distress and suffering; his countenance is worn and wrinkled, and looks like that of a much older person. But the disease scarcely ever occurs in those who are under forty years of age, and very often begins at a still later period. Anstie says that the worse case he ever saw was in a woman who was eighty years old when she was first attacked. According to this writer, a special feature in the ætiology of this form of neuralgia is that the sufferers from it almost invariably come of a stock which is tainted with insanity; indeed, they are themselves often the subjects of a suicidal melancholia, and their mental condition is almost always one of moody depression. This is no doubt partly caused by the severe pain which they have to endure, and partly by their inability to eat solid food; moreover, they are very apt to seek a temporary relief in drink.

True tic douloureux is happily very rare. I believe that its pathology is as yet unknown. Sir Thomas Watson gives an account of a post-mortem examination which was made in the case of Dr Pemberton, a London physician of great repute in his day, whose career was ruined by the disease. The os frontis was unusually thick, and a bony mass lay within the falx cerebri. It was supposed that these changes had set up an irritation which caused the neuralgia, but there is no evidence that any branch of the fifth nerve was involved in them, and outgrowths of bone of a precisely similar kind are often found in those who have had no pain in the head or face, nor any cerebral symptoms. Even in a celebrated case related by Romberg, in which the carotid artery was dilated to twice its usual size where it passed through the cavernous sinus, it seems to me exceedingly doubtful whether the so-called aneurysmal condition of the vessel had anything to do with the tic douloureux from which the patient had suffered on the corresponding side of the face; for that disease had existed for eighteen years at the time of his death, and one cannot suppose that the artery had been dilated during the whole of that time. I have repeatedly seen a similar affection of the carotid at the same spot in the bodies of those who had made no complaint of pain. It is also difficult to believe that the fifth nerve could have been pressed upon, without the sixth and the third nerves having been first paralysed. Moreover, Romberg found other changes in the affected parts, the nature of which one cannot determine from his account of them, namely, a hard yellow nodule in the crus cerebri, and a softened state, with reddening, of the roots of the fifth nerve.

It does not appear that this form of neuralgia is ever of "reflex" origin or caused by peripheral irritation of any branch of the fifth or of other nerves. There is, indeed, Mr Jeffries' oft-quoted case, in which a triangular piece of china from a broken cup remained lodged in the cheek of a girl and gave rise for fourteen years to violent pains, which ceased a few weeks after its removal by excision. But that patient was too young to have suffered from tic douloureux. Mr Tomes says that this complaint is never, so far as he knows, caused by irritation from diseased teeth; he quotes, as a warning, a case of Trousseau's, in which the pain came on when the patient touched with the tip of his finger his few remaining teeth, but in which they were extracted without the slightest benefit resulting. Some years ago a patient, who had in vain had a large number of his teeth removed, used to come to me as an out-patient.

There is a less intense but much more common form of trifacial neuralgia, to which even young persons are liable, and which is often dependent on morbid conditions of the teeth. The pain shoots and darts along the branches of the fifth nerve, but it is generally accompanied with a dull aching or gnawing sensation. It is often distinctly paroxysmal, and (like all other forms of neuralgia) it is especially apt to come on when the patient is over-fatigued or exhausted from want of food. It is seldom severe enough to prevent him from attending to duties or pleasures, although it may cause him great discomfort. In many cases it subsides at once if a glass of wine be taken, or a dose of quinine. According to Mr Tomes, the most frequent cause of this form of neuralgia is chronic inflammation of a tooth-pulp. But other conditions, which are mentioned by this writer and by Mr Salter as being capable of giving rise to it, are the difficult eruption of a wisdom tooth (or its impaction in its socket), the presence of secondary dentine in a pulp cavity, exostosis, hypertrophy of the

crusta petrosa, alveolar periostitis, decomposition of a dead pulp in a confined space, and even the exposure of sensitive dentine, or the crowding of the teeth together from insufficiency of room. In many instances the affected tooth is tender, so that sudden pressure on it, or the contact of substances much hotter or colder than itself, greatly augments the pain. But sometimes there is no local indication of any local disease. Mr Tomes points out that this may be the case even where an inflamed pulp is exposed, if the cavity is so situated as to be out of the way of irritation by particles of food. Mr Salter has related one instance in which, although the offending teeth were painful when touched, doubt for a time prevailed as to the real cause of the neuralgia from which the patient suffered, because this returned again and again, there being only a temporary interval of ease after each tooth in succession had been extracted, until the last two were removed at one operation. The pain then entirely ceased. All the teeth had nodules of exostosis on their fangs. It might be expected that any affection of a tooth in the lower jaw should give rise to neuralgia in the course of the branches of the third division of the fifth nerve, and that a diseased tooth in the upper jaw should set up pain in the distribution of the second division. But this is not necessarily the case. Indeed, Mr Salter mentions, as very frequent seats of "dental neuralgia," the supra-orbital nerve, the globe of the eye, the temple, and particularly a spot a little to one side of the vertex. I shall have again to advert to this when speaking of migraine. The practical rule would seem to be that the only way of avoiding the risk of overlooking affections of the teeth as causes of the milder forms of trifacial neuralgia, is to have the jaws thoroughly examined by the dentist in every case. Mr Salter has recorded some very curious instances in which the nutrition of parts affected with reflex neuralgia from caries of the teeth, underwent perversion; one in particular, in which the iris of the affected eye, from being of a deep hazel colour, became of a dull grey.

The late Dr Anstie believed ('Lancet,' 1866) that in certain cases he traced a neuralgia of the fifth nerve to the "reflex" disturbance caused by injuries to distant nerves; once to a knife-wound, by which the occipital nerve was divided, and twice to sickle-wounds of the wrist involving the ulnar nerve. In each instance the pain first came on when the injury was already repaired, and when the cicatrix had become firm.

It must also be borne in mind that pains undistinguishable from those of neuralgia, often constitute the earliest symptom of various diseases and tumours of the maxillary and other facial bones, and that such cases may come under the observation of the physician at a time when there is no obvious deformity or swelling to lead the patient to seek surgical advice. There is, in the museum of Guy's Hospital, a specimen of Bright's, in which a morbid growth which has started from the sphenoidal sinuses made its way into the middle fossa of the base of the skull, and doubtless pressed upon the fifth nerve; the patient, a woman of forty, whose countenance showed strong indications of suffering, had complained chiefly of an extremely severe pain on the corresponding side of the face, which became more violent in paroxysms.

Neuralgia affecting the neck, trunk, and arm.—Cervico-occipital neuralgia needs but brief consideration. Among the ascending branches of the cervical plexus, the great occipital, or posterior division of the second spinal nerve, is the one to which neuralgic pain is most often referred. Such cases are often due to the direct action of cold. Anstie speaks of a patient who was several

times attacked after sitting in a draught which blew strongly on the back of the neck. One must of course not overlook the existence of disease of the cervical vertebræ.

Cervico-branchial neuralgia generally affects several of the branches of the brachial plexus. Numerous "tender points" are developed, most of them at spots where the nerves pierce the fascia, but some (as, for instance, one of the inferior angle of the scapula) where their occurrence is not so readily explicable. Glossy skin and causalgia is sometimes present, particularly in cases of traumatic origin. One special feature of this affection is its liability to be aggravated by muscular movements of the arm. Anstie alludes to a case in which, at a time when convalescence seemed to be almost established, the act of playing on the piano for half an hour at once brought the pains back. Salter ('Guy's Hosp. Rep.,' 1867) has shown that neuralgia affecting the nerves of the upper limb is sometimes due to diseases of the teeth. He says that pains in the shoulder and acromion, over the insertion of the deltoid, or at the bend of the elbow, not rarely depend on this cause, (cf. p. 390); and he cites the case of a lady who, whenever any of the teeth in the left side of the lower jaw became tender from caries, was immediately attacked with severe neuralgia at a circumscribed spot in front of the forearm. Among the rarer courses of pains in the arms that might be mistaken for neuralgia, I may mention disease of the articular processes of the cervical vertebræ. In 1876 a woman was admitted into Guy's Hospital under Dr Wilks, who had for four months been suffering from pains in the right shoulder, round the side, and down the arm to the tips of the fingers. No cause for her complaints could be discovered, but after a few weeks she was attacked with pneumonia and died of that disease. Dr Goodhart made a post-mortem examination, and found that there was a quantity of inflammatory exudation outside the notch between the seventh cervical and the first dorsal vertebræ. This had its origin in disease of the joint between these two vertebræ; the lowest root of the brachial plexus was surrounded by it. Eruptions of zona and other cutaneous lesions are sometimes observed, but as with supra-orbital neuralgia, it is often difficult to determine which is the cause and which the effect.

The neuralgiæ of the *dorsal* and *lumbar nerves* do not need to be described in detail. They are less frequent than those of the face or limbs, but agree closely with cervical neuralgia. It is probable that the affection described by Sir Astley Cooper as irritable testis, is really a neuralgia of the spermatic plexus.

Sciatica.—One of the most important of all the varieties of neuralgia is that which attacks the great sciatic branch of the sacral plexus, and has long been known by the special name of Sciatica. The part of the nerve which most commonly suffers is that which lies behind the upper half of the femur, but sometimes it includes the whole length of all the main branches; in some instances it is even limited to the back of the knee, or to the terminal twigs in the calf or the foot. Its characters differ widely from those of some other forms of neuralgia, such as tic douloureux. Instead of consisting mainly of paroxysms of acute pain, darting like lightning through the nerve, and separated by intervals in which the patient is almost free, it is a constant, heavy, gnawing sensation; if it runs along the affected nerve at all, it does so slowly; its intensity is not indeed ever absolutely fixed and uniform, but the variations are comparatively slight and uncertain. It often appears to be worse at night, but this is ascribed by the late Dr Henry Lawson (who

was himself a sufferer from sciatica) to the fact that the patient then has less to distract his thoughts than in the daytime. Few diseases induce more peevishness, restlessness, and misery, spoiling the appetite, perverting sleep, and interfering with every kind of occupation, as well as with most amusements and all athletic sports.

In advanced and severe cases, the pain sometimes radiates into the sciatic nerve of the opposite side, or to branches of the lumbar plexus, or to nerves which are still more remote.

The pain is often greatly increased by pressure on the sacral plexus within the pelvis, or at its exit from the sciatic notch, and thus the act of defæcation may be attended with much suffering, and the patient may be fearful to sneeze or cough. He is also very susceptible to a draught of cold air.

When sciatica has lasted for any length of time, several "tender points" are generally to be found; one is said to be situated where the nerve emerges from the pelvis, others where its branches pierce the fascia lata, and others over the sacrum, the crista ilii, &c. The whole length of the nerve also becomes unduly sensitive to pressure, and especially (as I have already remarked) that part of the external popliteal nerve which lies under cover of the biceps tendon. I have never met with a case in which the patient did not complain of much more pain and tingling when I rolled this nerve beneath my finger on the affected side than when I did the same thing on the healthy side.

Wasting of the muscles is another symptom, which is present in all cases of sciatica of which the duration has been sufficiently long. The buttock becomes flattened and flabby, the back of the thigh and calf are reduced in size and feel soft and flaccid. In one case Anstie found the faradic excitability of the gastrocnemius much diminished. It is therefore a mistake to suppose, with some writers, that the loss of power depends merely upon disuse of the muscles. Even within the first few days, the patient often begins to limp in walking, so that he puts only his toes to the ground and is glad to make use of a stick. Afterwards he lies on the sofa all day long, with every joint of the limb bent, dreading to make the slightest movement. At last the hip- and knee-joints may become rigidly flexed, so that any attempt to straighten them causes severe pain. Painful cramps in the flexor muscles of the toes may come on, especially when the patient is just falling off to sleep. Sometimes fibrillary tremors are observed, or even shaking movements of the limb.

A certain degree of anæsthesia or impairment of tactile sensibility appears to be of constant occurrence in the more severe cases of sciatica, and subjective sensations of numbness and tingling are often present. The affected limb is sometimes paler and commonly colder than the opposite one; the patient occasionally complains of a sensation of chilliness in it, and it is said that the difference of temperature may be detected by surface-thermometers.

The fact that the symptoms of sciatica are to a certain extent unlike those which are commonly regarded as characteristic of the neuralgiæ, has led to some diversity of opinion with regard to its nature. The writers of a few years ago regarded it, not as a definite complaint, but as a group of affections due in different cases to widely different causes. Thus Sir Thomas Watson spoke of it as being sometimes inflammatory, sometimes plainly a part of rheumatism, sometimes the result of irritation within the pelvis, or

connected with a disordered state of the kidney, and, lastly, as sometimes purely nervous and neuralgic.

It is most important that one should not set down to "sciatica" the effects of pressure upon the sacral plexus and sciatic nerve from organic disease of whatever kind. I have myself published in the 'Guy's Hospital Reports' (vol. x, 1864) a case that was regarded as an example of this complaint, until the patient unexpectedly died; whereupon the gluteal region was found to contain a large aneurysm formed upon an abnormal artery, which passed down through the sciatic notch and along the back of the thigh, and constituted the main source of supply to the lower limb. That case is, I believe, unique; such a course of the vessel (which is normal in birds) being one of the rarest of all abnormalities in the human subject. But a precisely similar pain might probably be caused by an aneurysm upon one of the regular arteries of the buttock, or by a bony or sarcomatous outgrowth from the sacrum or os innominatum, pressing upon the nerve.

But, after all, such affections are comparatively seldom met with, and it appears to me that in all ordinary cases sciatica has as much claim to be regarded as a substantive disease as most other members of the nosology.

I quite agree with Anstie that the relation of sciatica to rheumatism is altogether imaginary, if any definite meaning be attached to the word "rheumatism;" and I do not think that the points which have been supposed to distinguish it from other forms of neuralgia are really fundamental. Many of them are perhaps attributable to the large size and the superficial course of the affected nerve, and to the way in which it is exposed to irritation when the patient walks about, or even sits.

The first case of sciatica that came under my treatment in private practice occurred in a gentleman who has since been to me for neuralgia affecting the nerves of the brachial plexus; and Anstie cites in his work three or four cases in which there appeared to be a tendency to neurotic diseases in other members of their respective families, or the patients themselves had had other forms of neuralgia. One of them occurred in a lady whose paternal grandfather had suffered from sciatica.

Other writers, however, deny that inheritance or predisposition plays any mportant part in the ætiology of the disease, and there appears to be no doubt that it is far more commonly traceable than are other neuralgic affections to accidental circumstances, especially to the action of cold. Lawson states that nine tenths of all cases of sciatica are referable to this cause, and Erb says that it is pre-eminent above other causes; exposure to a draught of air when the body is heated, wetting of the clothes, sleeping on damp ground or against a damp wall, have so frequently been followed by sciatica that no doubt can be entertained of their power to excite the disease.

Another frequent antecedent appears to be over-fatigue of the lower limbs. In Lawson's own case the complaint came on after a long walk; and some writers have described it as common in those who work hard at the sewing-machine.

The fact that certain classes of persons are exposed more than others to the exciting causes of sciatica probably explains the circumstance that it is more common in men than in women (according to Erb, in the proportion of four to one), and in adults between the ages of twenty and sixty, persons

from twenty to forty years old being still more subject to it than those who are older. Lawson, however, says that he has seen one well-marked case in a boy aged fourteen, who was addicted to masturbation.

The *diagnosis* of sciatica is seldom difficult to the practitioner who is alive to the numerous other diseases that may cause pain in the thigh and leg. Where the calf is the part mainly affected one must think of thrombosis of the femoral vein, but this can be excluded by digital examination of the vessel at the groin, even if the complaint should be too recent to have caused œdema of the ankle. Disease of the hip-joint can be put out of consideration by the fact that neither pressure on the trochanter, nor forcing the head of the femur against the acetabulum, gives rise to pain; and disease of the sacro-iliac synchondrosis by the fact that no tenderness is elicited by a blow over the articulation. Advanced cases, in which the patient limps in walking and has wasting of the muscles, are very likely to be mistaken for spinal disease; this occurred some years ago in the case of a medical man who was under my care, and whose brother practitioners in his own neighbourhood all felt sure that there was something the matter with his vertebral column. Such disease is, indeed, very unlikely to have its effects confined for a length of time to one limb; but, on the other hand, I have already remarked that, when sciatica is at its height, the pain often radiates to the sciatic nerve of the opposite side. Even if one is satisfied as to the seat of the pain, one must still search carefully for local causes of irritation. I remember to have heard of one very instructive case in which a surgeon, feeling carefully along the course of the nerve, was fortunate enough to detect the presence of a bit of broken needle, the removal of which led at once to the cure of the patient.

Prognosis and treatment.—The duration of neuralgia in general and its amenability to treatment vary greatly in different forms of the disease, as well as in different cases of the same kind. The most protracted and obstinate form of all is Tic douloureux; most writers speak of it as incurable. Even this, however, is a very long time in wearing out the patient's life. Trousseau mentions a case in which it went on for thirty years. In the other varieties of neuralgia it is altogether exceptional for the disease to last more than a few months; but Anstie alludes to some instances of sciatica as defying all medical skill. In forming a prognosis, one is to a great extent guided by the age of the sufferer; the older he is the more likely is the course of the disease to be prolonged. Recovery is generally gradual, and it is apt to be interrupted by relapses. Even after the pain is gone the affected part sometimes feels stiff for a long time, and its muscles are quickly fatigued by exertion.

The *treatment* of neuralgia is a matter which often taxes to the utmost one's patience and skill. Hygienic conditions must be very carefully attended to; fresh air, regular bodily exercise, freedom from worry and overstrain of mind, plenty of sleep, an abundant supply of wholesome nutriment, are each essential. Anstie laid special stress on the importance of a liberal diet, and particularly on the value of fat as an article of food. Whenever he could he made his patients take cod-liver oil; if the stomach revolted at that, he insisted on their eating butter, cream, Devonshire cream, or sometimes olive oil, or even cocoa-nut oil. He strongly objected to allowing sufferers from such affections to have wine or brandy, excepting in very moderate quantities and with the meals. I entirely

concur with him in the protests which he again and again uttered against the practice of prescribing stimulants for the mere relief of pain.

Quinine in large doses is often of striking benefit, especially in trifacial neuralgia, and in cases which show periodic exacerbation.

When neuralgia particularly affects the lower jaw, and whenever it is connected with dental irritation, *Butyl-chloraldehyde hydrate* ("croton-chloral") has sometimes striking curative effect, and is always worthy of trial. I have seen it unmistakably succeed and as unmistakably fail.

Ammonium chloride in full doses is believed, on excellent authority, to be often an efficient remedy.

Of all drugs, however, *arsenic* appears to hold the first place. I prefer the liquor sodæ arseniatis, because I have several times found that patients could take it without inconvenience who were attacked with diarrhœa and vomiting if they were placed on Fowler's solution. Or the liquor arsenici hydrochloricus may be used, in combination with the tinctura ferri perchloridi, itself a remedy to which Anstie attached a special value.

Another agent in the treatment of neuralgia is *phosphorus;* its employment has been especially advocated by Dr Ashburton Thompson. The formula to which he gives the preference is a solution of the drug in cod-liver oil; of this a fluid drachm, containing one twelfth of a grain, is administered every four hours. Other vehicles are alcohol and ether. Or the phosphide of zinc may be used in doses of one third of a grain every two hours; the great objection to this compound is its tendency to cause vomiting. Or, again, phosphorus may be made into pills, and the Pharmacopœia contains directions for their manufacture; but it has been shown by Dr Rees that phosphorus pills are apt to pass through the intestinal canal without being dissolved. Capsules containing phosphorus have also been employed. Dr Thompson gives a table of fifty cases of neuralgia treated by phosphorus, in several of which the effects of the remedy seem to have been very remarkable; but he says that if marked results are not attained within three days it is useless to persevere longer.

Galvanism is often very valuable in the treatment of neuralgia. One of the poles of a constant-current battery may be placed close to the spine of the affected side, or near the roots of the painful nerves, the other being applied upon the various "tender points" in succession, or being perhaps immersed in a vessel of water into which the hand is also dropped. A "descending" current is usually employed, the positive pole being put nearer to the centres than the negative one, but reversal of the poles seems to make no difference in the result. A good example of the effect of this form of electricity is afforded by a case related by Anstie in the fourth volume of the Clinical Society's 'Transactions.' A woman had for nearly five months suffered from cervico-brachial neuralgia, which lasted for several hours each day, and had defied all kinds of medicinal treatment. The very first application of a battery of from ten to fifteen cells arrested the pain; the attack which followed was much less severe than usual; and after thirteen days the complaint ceased to recur. The treatment was continued altogether for six weeks. Another case in which great relief was afforded was read to the Clinical Society at the same time by Dr Buzzard.

But it sometimes happens that this continuous galvanism fails. Faradisation of the nerve is rather injurious than beneficial, but the application of a powerful current to the skin by means of the electric brush is said to be sometimes very serviceable as a means of severe *counter-irritation.* Its

action is similar to that of *blisters*, which are undoubtedly of great value in many cases. Anstie recommended that they should be placed not upon the painful part itself, but close to the spine at the level of the affected nerves. Even in the desperate tic douloureux of old age he sometimes found that a blister applied to the nape of the neck removed the pain entirely for a long time. But in sciatica he obtained better results by placing flying blisters over the nerve-trunk in the gluteal region. Mustard plasters, chloroform liniment, and other rubefacient applications may also be prescribed in mild cases with advantage.

Acupuncture over the sciatic nerve, and *deep injections* of morphia in that region are powerful and frequently successful measures. A more recently introduced practice is *stretching* the affected nerve, either by cutting down upon it and forcibly pulling the trunk out of its bed, or by forcible flexion of the thigh upon the pelvis with the knee extended. Both methods have proved successful, but neither is without risk of injury.

Sciatica of the left side is said to be not unfrequently caused by retained scybala in the dependent loop of the sigmoid flexure or in the rectum, and to be cured by purges and enemata. These cases, however, are rather symptomatic pain than true neuralgia (p. 425).

Apart, however, from the pressure of gummata or other tumours on the nerve in the pelvis, it is probable that syphilitic neuritis may cause sciatica, for where other proofs of this are present full doses of potassic iodide and local mercurial inunction may effect a cure.

Moreover, while true rheumatic sciatica is extremely rare, if it occur at all, there is no doubt that some cases occur in persons who have suffered from gout, and it is probable that these are due to gouty neuritis. Such patients are benefited by purging and by colchicum. A few days before writing these lines, a patient of mine, with long-standing sciatica, who has repeatedly suffered from gout, was cured in a fortnight by colchicum alone.

When, however, all such attempts at "rational" treatment by local treatment or by drugs specially indicated have failed, we must fall back on the remedy which would naturally first suggest itself for so painful a disease as neuralgia—*opium*. Yet one should be very cautious in administering it, at least when there is any hope of the ultimate subsidence of the complaint. In the incurable tic douloureux the objection does not apply; and Trousseau employed opium in that affection with not a little success, increasing the dose boldly until sometimes as much as a quarter of an ounce or even half an ounce of laudanum was taken daily. In a few instances the result was more than a palliation of the patient's misery. The frequency and severity of the attacks of pain were lessened, so that after a time the drug could be discontinued. And although a relapse occurred a few weeks later, the interval of ease was a great gain; for food could be taken, which had before been almost impossible, so great was the suffering caused by the act of mastication.

In many cases the administration of opium by the mouth disorders the digestive organs. Hence the subcutaneous injection of morphia is greatly to be preferred, for it is attended with no such ill-effects; and of late years this method of treating neuralgia has been extensively employed. The quantity should be very carefully regulated; not more than a fifth of a grain (two minims of the pharmacopœial solution) should be used on the first occasion; in mild cases it is even well to begin with one tenth of a grain. If relief is not afforded by such doses, larger ones may

be tried; a quarter of a grain, half a grain, even a grain, is commonly injected under these circumstances; and sometimes much greater quantities still. One seldom has to use the syringe oftener than twice in the twenty-four hours; but Anstie laid stress on the importance of performing the operation as early as possible after the commencement of an exacerbation of the pain. It seems to be almost a matter of indifference whether the morphia is injected at the seat of pain or at any other part of the body, except so far as concerns the effect on the patient's imagination.

The effect of subcutaneous injection of morphia is often marvellous. Suffering which had been almost intolerable is removed, as by magic, within a few minutes. And this is not infrequently the first step towards the complete cure of the disease. It was so, for instance, in the severe sciatica of which Dr Lawson was himself the victim, and in which other plans of treatment had been tried without the least good result.

This method is not, however, free from evils of its own. Dr Oliver has related in the 'Practitioner' a case in which immediately upon the injection the patient cried out in great alarm, the pulse became very small, the face red, and the eyeballs prominent. Brandy was given, and in about half an hour these symptoms passed off. It is thought that the alkaloid may have been thrown directly into a small vein. But such an accident is excessively rare. A much more serious risk is that of setting up a "morphia-habit," a peculiar state of depression and irritability, which can be relieved by nothing but a fresh dose. There is also reason to fear that the remedy itself sometimes renders the pain more intense than before, or at least diminishes the intervals between the exacerbations. Even when the original disease is cured, patients sometimes experience great difficulty in discontinuing injections of morphia to which they have been accustomed; a vigorous effort may be needed, and the endurance of much discomfort and even misery for several days. For this reason, as well as for others, the syringe should not be entrusted to the hands of the sufferer himself, nor even to those of a relative or servant, unless perhaps in incurable cases. Anstie, however, laid stress on the fact that the evils connected with the establishment of a morphia habit never arise when the dose is kept below a certain point. In a valuable paper in the 'Practitioner,' he insisted strongly on the importance of "economy" in the use of the alkaloid; and he related the case of a lady, who for three years had enjoyed a complete immunity from severe sciatica, under the daily use of a dose of morphia which was at first one twelfth of a grain and was never raised above one fourth.

Peripheral Neuritis.—It has long been known that nerve-trunks are liable to inflammation; and some cases of neuralgia, of trophic changes, and even of motor paralysis are, as above stated, correctly ascribed to this lesion. Leyden explains reflex paralysis generally by ascending peripheral neuritis, and his views have been confirmed by histological evidence. Indeed, the Wallerian degeneration of a nerve severed from its trophic ganglia, which is associated with R. D., is itself a parenchymatous neuritis. But it is only lately that progressive neuritis of extensive tracts of peripheral nerves has been clearly ascertained to occur, not only as a concomitant or secondary condition, but also as an idiopathic affection involving many nerves, usually in a symmetrical manner, and having its definite course, associated symptoms, and results; in fact, the characters which constitute a "disease" as distinct from a pathological process.

Chomel appears to have first described the clinical features of this affection, which occurred epidemically in Paris in the spring of 1828, and his account was corroborated by Graves, who saw it there in the summer following. The great Irish physician describes it as beginning with pricking and severe pain in the hands and feet, followed by excessive tenderness to the touch. This was succeeded by anæsthesia, and finally by loss of motor power. It thence spread up the arms and legs, but after the patients had lain helpless for weeks they in most cases gradually recovered.

Duménil described a case of the disease in 1864. The clinical symptoms leave little doubt of its nature, and the microscopical examinations of the nerves (but not of the cord) by Pouchet confirms that opinion ('Gazette Hebdom.,' p. 203).*

Dr Buzzard described in the 'Clinical Transactions' for 1874 a case of bilateral facial and general paralysis, which recovered under anti-syphilitic treatment, and which he has since regarded as due to multiple peripheral neuritis; but there was no increased excitability of the paralysed muscles to interrupted galvanism, and of course no anatomical proof of the nature of the lesion. There is no mention of the disease as now recognised in Erb's article in 'Ziemssen's Handbuch' (Bd. xii, 2te Auflage, 1876). But in 'Virchow's Archiv' for 1877 (vol. lxix, p. 265), Eichhorst described, under the title "Neuritis acuta progressiva," a remarkable case occurring in a woman of sixty-six, with successive invasion of the nerves of the legs and arms, accompanied by pyrexia. It was a case of chronic Bright's disease. The lesions of the nerves are figured in pl. viii.

Professor Grainger Stewart published three cases of the same affection in the 'Edinburgh Medical Journal' for April, 1881, under the heading "Paralysis of the Hands and Feet from Disease of the Nerves." There is an excellent account of the disease under the title "Progressive Multiple Neuritis" in Dr Ross's treatise (vol. i, p. 354), and Dr Buzzard has published an interesting monograph in his lectures on "Paralysis from Peripheral Neuritis" (1886). He points out the probability that many cases of alcoholic paralysis which recover belong to this pathological group, especially those described by Dr Wilks in his well-known 'Lectures on Diseases of the Nervous System' (p. 272).†

Course and symptoms.—Multiple symmetrical neuritis (called peripheral to exclude affections of the nervous fibres which run in the brain and cord, and of the roots of the vertebral nerves) usually begins somewhat abruptly, and runs a rapidly ingravescent course, though it is less rapid than infantile paralysis. Pyrexia is sometimes present. Pain in the course of the nerves affected is quickly followed by more or less complete loss of power in the muscles they supply, and this paresis is accompanied by tingling, numbness and loss of tactile sensibility, and is frequently followed by trophic disorders of the skin, particularly vesicles and bullæ.

The pain is that of neuralgia, often the peculiar burning pain described as causalgia (p. 398). The loss of sensation occurs over broad patches of skin, often independently of a particular nerve-trunk, and these are frequently bordered by a zone of hyperæsthesia. The loss of power is variable in degree, from the slightest paresis to complete akinesis.

* The same writer contributed a monograph on the subject, with several other cases, to the 'Gazette Hebdomadaire' in 1866 (pp. 51—84).

† Other cases of paralysis due to alcohol, *e.g.* those recorded by Dr Broadbent ('Med.-Chir. Trans.,' vol. lxvii, 1884), are clearly different in clinical, and probably also in pathological character.

The distribution is symmetrical, and affects the extremities or the face, rarely the trunk; and the hands or feet, or both, rather than the proximal parts. The legs are most often affected of all parts of the body, and the extensors of the foot suffer most, so that the patient has a characteristic gait, in which the toe drops and catches the ground.

Faradic contractility is lost, as was noted by Duménil in his first case, and most often, as we should expect, the galvanic contractility is increased, with the other characters of R. D. (p. 324). But Dr Buzzard has found this not to be a constant feature, and it was wanting in a case (as I believe) of peripheral neuritis, which recovered under my own care.

The *prognosis* is generally good; the most typical cases appear to become rapidly worse, then to remain at a standstill, and then slowly to recover. But in practice our prognosis depends greatly on the clinical features of the case, and especially on its origin.

Diagnosis.—The distinction from neuralgia is not a real one, *i. e.* the pains *are* neuralgic; but anæsthesia (little marked, though often present, in neuralgia) is here nearly absolute, and there is motor paralysis as well. The course and clinical features are different from those of mere neuralgia, and probably the histological condition of the affected nerves is different also. Its symmetry distinguishes it from ordinary hemiplegia and from crossed hemiplegia; while lesions of the pons which affect both sides of the trunk and both sides of the face, without producing rapid death, have rather a theoretical than a practical existence. More often there is difficulty in distinguishing multiple neuritis from symmetrical affections of the cord and its membranes, particularly spinal meningitis. Cervical pachymeningitis in particular, closely resembles peripheral neuritis. So also do certain central lesions which run an acute course, like Landry's ascending paralysis, and some forms of essential spinal paralysis in adults. The absence of implication of the bladder and rectum, and of bedsores, distinguish it from destructive transverse lesions of the cord. Hysterical paraplegia does not affect the hands, and is not limited to the feet; while hysterical anæsthesia is unilateral and without akinesis.

Apart from the remarkable distribution, the most distinctive symptom is deep-seated tenderness on pressure, and particularly tenderness of the affected nerve-trunks. The usually favourable course, and the absence of atrophy or rigidity in the affected muscles, are additional helps.

The severe pains, along with disturbance of sensation and voluntary movement, are the symptoms which most resemble spinal meningitis. Loss of power in the extensors of the hands ("dropped wrist"), with deep-seated tenderness, resemble the ordinary symptoms of lead-palsy.

In some cases there are striking exceptions to the usual symptoms. Thus the R. D. may be absent or imperfect; there may be loss of control over the bladder and rectum, as in a case of my own, above noticed; and there may be, as Leyden has recorded, paraplegic bedsores. Atrophy, commonly slight, may be well marked, or rigidity of the affected muscles may supervene. Probably in these cases the lesion is not confined to the peripheral nerves. Loss of knee-jerk is very common, particularly in alcoholic cases, and with the pains and unsteady gait has led to the diagnosis of tabes. The gait, however, differs from that of locomotor ataxia, as well as from that of cerebellar disease, and is very characteristic of one of the forms of alcoholic paraplegia. If we call the tabetic gait "unsteady" or "stagger-

ing," and the cerebellar "reeling," we might apply the term "halting" to the dragging of the feet described above.

Histology.—This depends on the existence of inflammation affecting the nerve-fibres, and leads to "breaking up of the axis-cylinder" (Stewart), to "sclerotic atrophy" of the nerves with loss of myelin and thickened perineurium (Leyden). To the naked eye the affected nerves appear normal, and in the typical cases no secondary changes are found in the nerve-roots or in the cord. In guinea-pigs, by administration of lead, Gombault (quoted by Ross) produced similar changes, not continuously but in segments of the nerve.

Ætiology.—It seems clear that while some of the best-marked cases are idiopathic, others are certainly due to alcohol, others are decidedly gouty, others are due to poisoning by lead, and others again are sequelæ of enteric fever and other acute diseases. The cases which recover cannot, of course, be tested by anatomical investigation; but judging by clinical features most cases of lead palsy, certain cases of alcoholic, and certain cases even of syphilitic paralysis may be grouped with those described by Chomel and Graves, Eichhorst, Leyden, Stewart and Buzzard, and it seems likely that diphtheritic paralysis will prove to be of the same nature.

At present, however, it is well to leave the best marked of these in their ætiological relations, and still to speak of alcoholic, syphilitic, plumbic, and diphtheritic paralysis.

Of the cases clinically conforming to the character above stated, which have fallen under my own observation, one was apparently idiopathic, and occurred in an elderly man with great acuteness and severity. He gradually and slowly recovered. Another, of decided alcoholic origin, in a powerful man about thirty, proved fatal after a short course, and the brain and cord were free from disease. A third, more certainly genuine, occurred soon after an attack of gout, and subsided without local treatment. It was confined to the hands and feet, and showed all the features above described.

Treatment.—In gouty, plumbic, and syphilitic cases, the appropriate treatment is that which is directed to their respective causes. For patients of intemperate habits, entire abstinence from liquor and assiduous feeding will often succeed against expectation. In "idiopathic" cases no therapeutical measures are at present generally accepted, and it is most rational to treat them, not "on general principles," but according to the "indications of the disease" as successive symptoms demand relief. One plan, however, seems to be almost always beneficial, namely, the application of slowly interrupted galvanic currents.

It is worthy of mention, before leaving this subject, that some of the symptoms of a disease endemic in Japan, and known as *Beri-beri* or *Kakki*, are remarkably like those of symmetrical peripheral neuritis. It is sometimes epidemic, like the cases described by Chomel, and it is said to be contagious. It is also associated with dropsy. See Mr Wm. Anderson's "Lectures on Japanese Beri-beri," 1879; Morehead ('Br. and For. Med.-Chir. Rev.,' 1855); Fayrer ('Med. Times and Gaz.,' 1880), and Vilette ('Brit. Med. Journ.,' April 23, 1887). In Dr Buzzard's second lecture (p. 64), Harada "Die Japanische Kakké" (1882), and Palm ('Edin. Clin. and Path. Journ.,' Sept., 1884), are also quoted.

AFFECTIONS OF THE SPINAL CORD

PARAPLEGIA

Difficulties of this section—Arrangement of diseases of the cord proposed.
Symptoms of paraplegia—Localization—Superior and inferior limits—Incomplete paraplegia—Reflex movements—Knee-jerk and clonus—Muscular atrophy and rigidity—The bladder in paraplegia—The rectum and genitalia—Ammoniacal urine and bedsores.
Intrinsic Paraplegia—*Myelitis—Histology of acute myelitis—Of chronic myelitis or sclerosis—Ætiology—Course and symptoms—Prognosis—Diagnosis from paraplegia due to Spinal Weakness, Anæmia and Congestion of the cord, from Reflex and Hysterical paraplegia, Concussion of the spine, Acute Ascending paralysis, Alcoholic and Syphilitic paraplegia, spinal Hæmorrhage, and Tumours of the cord, with pathology and symptoms of these affections severally—Treatment of intrinsic paraplegia generally.*
Hemiparaplegia—Rachialgia.
Extrinsic *or* Compression Paraplegia—*from vertebral caries or cancer—from aneurysm, hydatid cyst, or meningeal tumour—Symptoms—Diagnosis—Prognosis and Treatment.*
Spastic Paraplegia—*in adults—in children—Primary and secondary—Anatomy—Symptoms—Prognosis and Treatment—The Author's experience.*
Meningeal Affections—*Hæmorrhage—Acute and chronic spinal meningitis.*

Within the last few years our knowledge of affections of the spinal cord has made great progress; certain lesions can now be positively diagnosed which were entirely unknown to the pathologists of the last generation. Unfortunately, however, the advance has been mainly in regard to diseases which are comparatively rare. Upon the more common ones much less light has been thrown; and their diagnosis still remains a matter of presumption rather than of certainty; for it is based mainly upon our knowledge that under given circumstances one kind of disease is more frequently met with than another. No doubt the same interpretation of symptoms and signs by help of probability often applies in the diagnosis of diseases of the brain or the abdomen; but compared with the spinal cord, I think there is no other region of the body in which the morbid changes revealed by an autopsy are so often unexpected.

Classification.—The arrangement of diseases of the cord is not easy. Very few can be grouped on an ætiological system, since their origin is often most obscure. The pathological processes are comparatively few, and vary greatly in the symptoms they produce according to their precise localisation. Anatomical lesions are peculiarly difficult to determine, from the extreme slowness of their course in most cases from the difficulties in histological

examination, and from the rarity of some of the most obscure. Moreover, some diseases of the cord have, so far as the most skilled observers working with modern methods can determine, no anatomy at all. A clinical classification is, therefore, in the present state of our knowledge, most practicable; but the distinction between functional and organic affections, the local distribution of chronic myelitis, and the ætiological facts which bear upon treatment or prognosis must also be regarded.

The plan here adopted will be to describe, first, the most frequent and obvious symptom in lesions of the cord, namely, Paraplegia. The varieties of this according to its pathological cause when known, or according to its concomitant or causative conditions, will naturally follow. First we will take Paraplegia from Myelitis, and from other causes operating *within the cord.*

Next will follow an account of Paraplegia, the result of direct compression of the cord *from outside*, which differs in prognosis and treatment from all the preceding forms.

We shall next consider a form of paraplegia in which the paralysed muscles become affected with *spasm;* it is sufficiently separated by other clinical features and by the anatomical lesion which usually accompanies them, to be classed separately.

An account of the symptoms produced by affections of the spinal *meninges* will complete the present chapter.

The *atrophic* forms of spinal paralysis, some paraplegic but others not, are conveniently grouped together, and will form the next division of the subject.

Most of these various spinal paralyses are defined by the anatomical lesions to which they may be traced; but there remain two remarkable nervous disorders which have also been satisfactorily associated with definite changes in the cord, but which have far more constant and characteristic clinical features in origin, course, symptoms, and event than the preceding affections, and may be regarded as natural pathological series of events. These two "diseases"—Tabes and Insular Sclerosis—complete the list of definitely spinal maladies.

PARAPLEGIA.—This is a form of paralysis, possessing two essential features: first, that it affects both sides of the body, and generally to an equal extent and in the same degree; and, secondly, that it affects all parts, up to a certain level, according to the functions of the several pairs of spinal nerves, ascending from the sacral to the cervical. It is easy to see why affections of the cord should produce such a symptom. On the one hand, the motor tracts belonging to the two halves of the body are in the cord so close to one another that they are generally involved in the same lesion. On the other hand, since each segment * of the cord contains fibres belonging to all the nerves below, they are all likely to suffer together.

* A "segment" of the cord is included between any two adjacent horizontal sections. It takes the *whole thickness* of the cord, and a single pair of nerves. We thus recognise eight cervical, twelve dorsal, and five lumbo-sacral segments, although the last are too closely packed together for us to be able always to distinguish their several lesions.

It must be carefully borne in mind that while each segment generally resembles the rest, there are important differences between them. First, in the total size and shape; secondly, in the proportion of grey to white matter, and the size of the anterior cornua; thirdly, in the development of the several "tracts" of the cord.

Thus as we pass downwards the crossed pyramidal tract becomes smaller and more superficial; the direct cerebellar and direct pyramidal tracts gradually disappear; the

Although Sir William Gull and other writers have spoken of a "cervical paraplegia," when the arms are paralysed without the legs, I think the expression is incorrect. In such cases, the affection, if it lies within the cord at all, must be definitely limited to a part only of the segmental area; and between them and cases of ordinary spinal paralysis there are many important differences. One cannot insist too strongly on the fact that the morbid changes which cause true paraplegia acknowledge no such limitations. They affect one or more segments in their entire width, sometimes by gradual extension, more often at once, and always at last.

Locality.—As regards their distribution in the length of the cord, the lesions in question vary widely. They may be strictly confined to a single spot, or may extend from the cauda equina to the cervical region, or may transcend the limits of the cord and invade the bulb as well.

The *upper limit* of a spinal affection is, as a rule, roughly indicated by the extent upwards of the paraplegia to which it gives rise. Disease of the lumbar enlargement causes paralysis of the lower limbs; disease in the dorsal region, paralysis of the abdomen and chest, corresponding with the level to which it reaches; disease of the cervical enlargement, paralysis of the upper limbs; disease still higher up, paralysis of the diaphragm and of the cervical muscles.

In some cases the feet and legs become powerless before the thighs and the hips. This is generally supposed to mean that the lesion is at first confined to the extreme lower end of the lumbar enlargement; but, since Woroschiloff found that in the lateral columns of the lumbar cord of the rabbit the motor paths for the distal parts of the lower limbs lie outside those for the proximal parts, it has been conjectured that the symptom in question may perhaps sometimes be due to gradual extension of a morbid process from the surface inwards. When paralysis attacks the upper extremities in detail, as the result of disease spreading upwards through the cord, I believe that the muscles of the hands are affected before those of the elbow and of the shoulder. The fact that the nerves to the ulnar side of the hand come chiefly from the lowest part of the brachial plexus would lead one to expect that the inner fingers would suffer earlier than the outer fingers or the thumb. I do not know whether this is the case; if not, we have here a distinction between lesions of the cord itself and those of the nerve-roots.*

When any segment of the cord is completely destroyed, there is of course an absolute loss of *sensation*, as well as of motion, in all parts of the body below. And when a total disorganisation spreads upwards through its

postero-median column of Goll becomes smaller and deeper. The anterior cornua are very large in the cervical and lumbar enlargements, very small in the dorsal region; while the important group of cells known as Clarke's vesicular column is well represented in the dorsal (especially the lower dorsal) region, and almost absent in the cervical and lumbar.

Lastly, it must be remembered that the lumbar or lumbo-sacral enlargement is so named from its giving origin to the nerves of the lumbar and sacral plexuses, but that it does not, like the cervical and dorsal regions, correspond to the vertebræ of the same name. The cord ends, in the adult, at the first lumbar vertebra, and from the mid-cervical region the nerves pass down to their foramina of exit from the vertebral canal, with increasing obliquity, until they are collected together into the cauda equina.

* According to Erb ('Ziemssen's Handbuch,' xi, 2, p. 62) paralysis of all four limbs, and of the trunk muscles generally, sometimes occurs without any interference with the respiratory movements, as the result of an exceptional limitation of disease to a part of the segmental area of the cord in the cervical region, leaving the lateral columns intact. Strictly speaking, we should not call such an affection paraplegic, in the sense in which I have defined that term.

substance, the gradual progress of the disease from day to day can be determined from the extension of the anæsthesia with far greater accuracy than is ever possible from that of the motor paralysis alone. But in the great majority of cases, parts affected with paraplegia either retain perfect sensation, or have that function but little impaired.

This preponderance of loss of motion over loss of sensation is not only met with in all diffused spinal affections, but belongs to them in common with diseases of the peripheral nerves and of the brain itself. And the true explanation is, no doubt, that which was given when the diseases of the nerves were under consideration; namely, that sensory impulses are transmitted far more easily than motor ones, so that unless the conducting fibres are completely disorganised anæsthesia does not occur. Many patients, however, complain of *parœsthesiæ*, such as "pins and needles," sensations of "pricking" or "tingling" in the toes or in other parts, "creeping" or "crawling" feelings, subjective sensations of heat or of cold.

Physiological results.—If the account of paraplegia in the last few sentences is correct, it would follow that this symptom, as it is seen in man produced by disease, has received little direct elucidation by the experiments of those physiologists who have devoted such infinite pains to the investigation of the exact paths for the transmission of sensory and of motor impulses respectively within the substance of the cord. And this, I believe, is really the case. I shall hereafter have to describe certain affections which are strictly limited to particular tracts of the grey or of the white matter; but we shall find that none of these are characterised by simple akinesis or simple anæsthesia of the parts below. On the other hand, the lesions which cause paraplegia tend to diffuse themselves over the whole segmental area of the cord, and no special localisation of them is concerned in causing the preponderance of loss of motion over loss of sensation, which is so commonly observed.

All physiologists, however, are agreed that volitional motor impulses pass mainly along the lateral columns. With regard to the paths for sensory impulses, there still is doubt. One opinion was maintained by Brown-Séquard, that they are situated in the grey matter. Another, which was first taught by Schiff, is that tactile sensations are transmitted through the posterior columns, but sensations of pain through the grey matter. A third and better supported hypothesis places the chief sensory tracts in the deeper part of the lateral columns of the opposite side. But this most difficult problem is still far from satisfactory solution. So far as I can judge, neither view, supposing its truth to be demonstrated, would at present be practically available in the regional diagnosis of affections of the spinal cord as we see them at the bedside. We shall find that a central tubercular nodule is occasionally the cause of paraplegia. I have not read, nor have I observed, that anæsthesia precedes the motor paralysis in cases of this kind, which seem to approach more nearly than any others to the conception of a lesion destroying the grey matter only, such as, on Brown-Séquard's theory, should produce that effect. On the other hand, it ought to follow, if Schiff's doctrine were correct, that such an affection should rather cause analgesia, tactile sensation being retained. But as yet, so far as I know, nothing of the kind has been noticed. It may of course be replied that such a nodule does not completely disorganise the grey matter before it presses on the white columns; but this is the very point on which I would insist, namely, that the diseases which cause paraplegia are not so localised as to admit of the application of laws deduced from experiment. One point, however, which is

of great clinical importance, is more than probable, namely, that the sensory fibres do not cross at the decussation of the pyramids in the bulb, but pass over to the opposite side, at once or gradually, at no great distance from the posterior roots.

Partial paraplegia.—Hitherto, I have spoken only of complete paraplegia, a condition in which the affected muscles are altogether incapable of being influenced by the will; but in a large number of cases the paralysis is incomplete. Every degree of loss of power may be noticed. One patient can just move his toes, another can flex the knees so long as they lie horizontally on the bed; a third can draw the thighs up to the abdomen; a fourth can stand for an instant and then falls down; a fifth can walk a few paces, and so on, up to a condition in which all that can be detected on the most careful examination is a slight uncertainty of step, with a tendency to catch one of the feet against anything that happens to project above the pavement. When the paresis is a little more marked than this the gait is peculiar, and it is well worthy of study because it contrasts with that which is observed in some other spinal affections that will be discussed further on. Erb describes it in the following terms:—"The foot hangs down in walking, the toes are dragged, the sole is brought to the ground clumsily, and for the most part upon its outer edge; the knee is too much raised or carried forwards without being bent; there is generally a kind of stiffness about the legs. The patient uses one or even two sticks, or he is held up by crutches or by the arms of other persons; he totters but slightly and stands quietly and steadily. When left unsupported he sinks down upon the ground. The gait of course varies somewhat according to the number of muscles which are paralysed. When only those below the knees are affected it is waddling and very peculiar." In those cases of partial paraplegia in which sensation is also impaired the patient feels when he stands upon his feet as though they were wrapped in wadding or covered with thick woollen stockings; or a hard floor may seem to him like a thick felt carpet. I believe that he then always requires the guidance of vision to enable him to stand firmly or to walk evenly. We shall presently find that where a patient who has his feet close together totters or falls, as soon as he is made to close his eyes, many observers infer that he is suffering from a special disease of the cord—"sclerosis of the posterior columns" or "locomotor ataxy." I quite agree with Erb that although the symptom in question is often present in that disease, it is yet by no means peculiar to it, being met with whenever there is an imperfect transmission of those sensory impulses from the soles of the feet, and from the other parts of the lower limbs, by which the equilibrium of the body in the erect posture is normally maintained.

Reflexes.—The lower limit of a lesion of the cord cannot be determined with any great accuracy. We have seen that disease in the cervical region renders the legs as well as the arms paraplegic, and this equally whether the affection is confined to a small part of the length of the cord or involves the whole of it down to the cauda equina. A very simple observation, however, enables us in many cases to say that at least some of the lower segments retain their functions, although their connection with the brain is cut off. We have but to see whether *reflex movements* can be excited in the legs by impressions upon the cutaneous nerves. If this experiment succeeds, it proves that the lumbar enlargement is still undestroyed. On the other hand, its failure is not to be taken as conclusive evidence that the disease

extends throughout the whole of the lower part of the cord. On the contrary, it often happens in cases of very limited lesions that reflex movements can only now and then be elicited. For example, surgeons find, when there is fracture with displacement of the upper dorsal or cervical vertebræ, that for a few days after the accident there is an entire absence of reflex excitability in the cord; it seems to have been in some way deprived of its functions by the shock.* So also, we shall hereafter learn that the movements are generally less readily obtained when disease has begun in the interior of the cord than when it is merely compressed from without, the reason being that in the former case an "inhibitory" influence is transmitted downwards upon the lower centres. Even when these retain or have regained their susceptibility, it may sometimes be easily exhausted for a while by repeated calls upon it. In a well-known paper, read before the Royal Medical and Chirurgical Society in 1839, Dr William Budd mentions the case of a young lady in whom the slightest disturbance of the bedclothes caused very energetic contractions of the right leg, which was paralysed; but there was a successive diminution in the vigour of the spasms on each renewal of the stimulus. The impressions that produce reflex movements are sometimes felt, and in the case of this young lady the movements themselves were attended with pain like that of cramp. On the other hand, Dr Budd relates an instance in which all the limbs were paralysed by an injury to the cord in the cervical region, but in which convulsions in the arms were excited by drawing on the hair of the head or by tickling the chin; the efferent impulses must then have traversed the seat of lesion, through which no volitional efforts could be made to pass. As a rule, however, cases in which all communication with the brain is absolutely cut off are those in which the reflex activity of the cord is most marked; the patient then has no consciousness either of the application of the stimulus or of its effect, unless he sees his legs jerked upwards. It seems probable that the susceptibility of the lower spinal centres is often directly augmented by an irritant influence transmitted downwards from the diseased part, just as it constantly is by the administration of strychnia, which excites spasms in paralysed limbs much earlier than in those which remain obedient to the will. Sensitive impressions upon the skin of the soles of the feet seem to set up reflex movements in the legs much more easily than similar stimuli applied to any other part of the cutaneous surface; lightly touching the hollow of the foot with a feather often throws the whole limb into spasm. As might be expected, a hot metal plate produces much more marked effects than one at an ordinary temperature, even when the transmission of sensory impressions is so imperfect that the patient cannot tell the difference. But the most violent convulsions of all are those which are sometimes excited by micturition and by defæcation. One of Dr Budd's patients was obliged, whenever he was placed upon the night-stool, to have his feet inserted into two large loops of saddlers' webbing which were nailed to the floor, to have two larger loops adjusted over his knees, and to hold down his thighs; otherwise he was liable to be thrown forwards upon the ground.

Reflex movements are more or less co-ordinated. There seems no reason to suppose that the muscles in general use are individually represented in the spinal centres. And whatever may be the machinery in the brain by which the several groups of muscles are harmonised and co-ordinated

* See the instructive remarks of Prof. Goltz on this subject in his 'Verrichtungen des Grosshirns,' S. 78-81.

in their actions so as to produce definite changes in the position of a limb, it is certain that cross-connections exist within the cord by means of which a similar result can be brought about. We do not in a paraplegic man see such marked purposive reflex movements as are observed in decapitated frogs. The nearest approach to them that I remember to have read of was in a case of Dr McDonnell's, recorded in the 'Dublin Quart. Journ.' for 1871. The patient was paralysed in all four limbs; when a catheter was passed into his bladder his left arm would "wriggle up and down, and the hand come fluttering over the genital organs, although he was absolutely unconscious of the operation, and even when his eyes were shut." Another curious case is related by Virchow at p. 684 of his 'Gesammelte Abhandlungen.' It is that of a man who was paraplegic, and who generally lay in bed with his lower limbs rigidly flexed. When he wanted to stand upright he would pinch the right thigh sharply, or give it a smart blow on its outer surface; this threw both the legs into a position of extension. Still the gastrocnemii remained contracted, and before he could put his foot to the ground he had to bring down the heels with both hands. As a rule, when reflex movements are excited by impressions upon the cutaneous surface they remain confined to the limb on the same side. Leyden says that they do not spread to the opposite limb except when the lesion is in the bulb, but many instances to the contrary may be found. The movements generally consist in a series of jerking spasms rapidly following one another, in which all the joints are flexed, but sometimes the contraction is rather of a tonic character. A curious illustration of the way in which one set of reflex actions may be made to counteract another set is afforded by a case of Dr Budd's. The patient was recovering from paraplegia and could walk a few steps when the contact of the soles of his feet with the ground would make his knees bend beneath him; this he was able to overcome by rubbing the surface of his belly, the legs being then extended with a jerk.

Beside the well-known reflex movements called *plantar* and *cremasteric*, several others may be obtained and their absence or excess may be of diagnostic value.

(1) Some are obtained by touching, pinching, or stroking the skin so as to produce contraction of the underlying muscles. They are called superficial reflexes, and have been carefully investigated by Dr Gowers. The following are the most important:—*abdominal*, when the external oblique muscle contracts; the *epigastric*, when tickling the skin over the fourth to the sixth ribs produces contraction of the upper segment of the rectus; the *gluteal*, when irritation of the skin of the buttock leads to dimpling over the insertion of the glutæus maximus into the fascia lata; the *scapular*, a slight movement of the teres major in the fold of the axilla when the skin between the shoulders is pinched. These are most easily produced in children, and others may be discovered at an early age, as the *palmar* reflex or closure of the fingers produced by gently touching the palm of a sleeping infant. In elderly people several are absent, and all are less readily elicited.

(2) There are what may be termed visceral reflexes:—*vesical*, when the bladder is caused to contract by irritation of the urethral orifice, of which the incontinence of urine in children, by reason of a long prepuce, is an example; *penile*, when erection is produced by the passage of a catheter or by the slightest contact, a condition sometimes seen in cases of partial paraplegia, short of that which produces priapism; *rectal*, when contraction

of the bowel follows the introduction of a suppository; the familiar reflex action of *vomiting* from tickling the fauces; *sneezing* from a draught of cold air or a brilliant light, *blinking* from touching the cornea, and contraction of the *pupil* to light and accommodation.

As criteria of the seat of a spinal lesion:—the plantar reflex corresponds to the sciatic nerve and lower part of the lumbar enlargement of the cord, situated in the adult opposite the last dorsal spinous process; the gluteal reflex corresponds to the posterior fourth and fifth lumbar branches, the lumbar enlargement, and superior gluteal nerve; the cremasteric to the first and second anterior lumbar branches; the abdominal to the lower three or four intercostal nerves and dorsal cord; the epigastric to the middle ones (fifth to seventh), and the upper dorsal cord; and the scapular to the upper bundle of the brachial plexus, and cervical enlargement of the cord.*

The movement of the ribs in respiration also enables us to judge of the integrity of the connection of the intercostal nerves with the brain, or of the point where it is severed by transverse lesion of the cord. The act of swallowing depends on the centre situated in the bulb, and that of contraction of the iris on the centrum ciliospinale in the same region.

(3) A closely connected set of phenomena have been called *deep reflexes*, but it is doubtful whether they are due to reflex or direct stimulation of the muscles involved.†

The most important was first described in 1875, by Westphal and Erb independently; it was called by the latter the "patellar tendon-reflex," by the former the "knee-phenomenon." The best English name is *knee-jerk*, and it has been long known to schoolboys. A smart tap is given with the side of the hand upon the tendon of the great extensor muscle of the leg, above or below the patella, while the knee is crossed over the opposite thigh; and the foot is jerked upwards. A similar elbow-jerk may be obtained in most healthy persons by striking the stretched tendon of insertion of the triceps.

The absence of the knee-jerk is usually, perhaps always, a morbid sign; but different persons vary much in the readiness with which it is procured. It is exaggerated in paraplegia, but, contrary to what we should expect, is diminished in the paralysed leg of hemiplegia. In morbidly excitable conditions the jerk may sometimes be obtained by stretching the ligamentum patellæ and striking the subcutaneous surface of the tibia; or by stretching the tendo Achillis and tapping the tibialis anticus and flexors of the toes, an extension of the foot may be obtained (Gowers' front-tap contraction).

The so-called deep reflexes do not always increase or diminish, in correspondence with the true or superficial reflexes.

Ankle-clonus is the name given to the clonic contractions produced in some patients by forcibly bending up the foot so as to make the tendo Achillis tense. It was first described by Charcot, and is included by Erb and Westphal among tendon-reflexes. It usually goes with exaggerated knee-jerk, and is probably always a morbid phenomenon. But a somewhat different kind of clonic spasm of the gastrocnemius can be obtained in most healthy persons by bending the knee and ankle with the ball of the foot resting on the ground, and moving the knee rapidly up and down.

* See an instructive diagram by Dr Gowers ('Diagnosis of Disease of the Spinal Cord,' p. 58).

† There is much to be said against the reflex character of this phenomenon. See papers by Tschirjew ('Arch. f. Psychiatrie,' 1877, Du Bois's 'Arch.,' 1879), Gowers ('Lancet,' i, p. 156, 1876), Waller ('Brain,' July 1880), and De Watteville ('Brit. Med. Journ.,' 1882 p. 736).

The paralysed muscles.—The condition of the centres in the lower part of the cord has a direct influence on the nutrition of the paralysed muscles, and on their electrical reactions. We have seen that when disease destroys the entire substance of the cord up to a certain level, no reflex movements of any kind can be obtained in the limbs below. Under such circumstances the muscles become very rapidly deprived of their electrical contractility, and they undergo marked *atrophy*. Before there is a complete loss of susceptibility to galvanic currents, there is generally a period in which the "reaction of degeneration" is present. On the other hand, when paraplegia is due to a lesion limited to one of the upper segments of the cord, so that reflex movements persist in the paralysed limbs, the muscles, as a rule, retain perfectly normal electrical relations, and they remain well nourished for months and years, or at most show a slight flaccidity and wasting, which may fairly be attributed to disuse. They are still in connection with their trophic centres in the anterior cornua. Again, paraplegic muscles may become *rigid*, so that the legs are either forcibly extended, or drawn up in a state of flexion. Cases of this kind will be separately described hereafter, under the name of "spastic paraplegia." The knee-jerk is almost always exaggerated and clonus present.

The bladder in paraplegia.—Non-striated, as well as striated muscles can be excited to reflex contractions. Erb mentions a curious case of paraplegia, in which a fluid fæcal evacuation was passed whenever a large bedsore was dressed. And in other instances micturition was directly produced by pressure over the bladder, or by the introduction of the finger into the rectum. It does not follow that the impulses were reflected upon the vesical muscular fibres after being conveyed to the cord by cutaneous nerves, for the slightest compression of any part of the bladder appears to be sufficient to make its mucous membrane the recipient of stimuli from the urine contained in its interior. Indeed, physiologists now believe that when micturition seems to be a volitional act, the influence of the will is really limited to relaxing the sphincter, and throwing the abdominal muscles into forcible contraction, so as to press upon the bladder, and to set in action its reflex machinery. The experiments of Goltz upon young dogs ('Pflüger's Archiv,' 1874) have shown that in these animals the bladder is capable of emptying itself at regular intervals, and in a perfectly normal manner, after the cord has been completely cut across by a sharp knife. Thus physiology would lead us to expect that micturition should take place naturally in those cases of paraplegia in which the lesion is limited to the upper part of the cord. In practice, however, it is found that retention of urine almost invariably occurs, even when reflex movements can be easily excited in the lower limbs, proving that the lumbar centres are intact. The explanation doubtless is that an inhibitory influence is transmitted downwards upon those centres from the diseased part above. Goltz, in fact, states that in some of his experiments it was not until several days after the operation of section of the cord that the animal was able to micturate properly; and he suggests that in cases of injury to the spine in man the bladder would after a time be found to have regained its functions, if catheterism were not continued as a matter of routine. However this may be, it is certain that the theoretical accuracy of Goltz's views with regard to micturition is established by some very exceptional cases of paraplegia, in which the urine is passed quite naturally. It is remarkable how many of the patients whose cases are related by Dr Budd in his well-known paper were able to micturate, though

not always in a perfectly normal manner. In the case which I have already cited as presenting such violent convulsive movements of the legs, the urine at first began to flow in interrupted jets, but as the spasms grew more eeble its stream became continuous. Another patient, at a certain period n his illness, emptied his bladder involuntarily and with a sort of jerk, about every two hours. In a third case, one of fracture of the spine, in which there was at first retention, the power of voiding urine quite naturally was regained on the fifth day, whereas no volitional movements in the legs occurred until the ninth day. So, again, one of the cases related by Ollivier, in his classical work,* is that of a man who had complete paralysis of his lower limbs as the result of caries of some of the higher dorsal vertebræ, but in whom the bladder performed its functions normally.

It is an interesting question whether the converse can occur, that is, whether the lumbar centre for micturition is ever the only part of the cord diseased, so that while the bladder is paralysed the lower limbs nevertheless retain the power of movement. I have notes of more than one case in which retention of urine has preceded all other indications of the onset of paraplegia by an interval of a few days, but I do not remember any instance in which it has continued to be the sole or even the main symptom of a spinal affection. Some years ago I saw a gentleman who was one of the sufferers in the Thorpe railway collision, and whose chief complaint was that he could pass his water only in a very feeble stream; he said that "it flowed from him like oil," and it quickly became ammoniacal and very fœtid. He had pain and tenderness in the back, but the only sign of any loss of power in the lower limbs was that in walking he sometimes seemed to totter, especially if his foot caught against any slight obstacle; he also had "twitchings" in the legs three or four times a day. He was not long in getting perfectly well.

In some instances in which the power of micturition is partially impaired one can distinguish paralysis of the *sphincter* from that of the *detrusor*, the muscular coat of the bladder itself, by which expulsion of the urine is mainly effected. It might have been supposed that in the former case there would be complete incontinence, the fluid dribbling away incessantly. But physiologists have found that even after destruction of the lumbar cord the neck of the bladder can withstand a pressure of six inches of water (as compared with one of twenty inches when the cord is intact). And this result is entirely confirmed by some observations of Hutchinson ('Brit. Med. Journ.,' 1877) upon persons who have the remains of a shrunken spina bifida, involving the fourth pair of sacral nerves, so that the sphincters of the bladder and rectum alone are permanently paralysed. In such cases he finds that the bladder may be able to hold a large quantity of urine, but if once its reflex contractions are excited the will is, of course, powerless to restrain them, and therefore there is a constant risk of accidents. So in many cases of partial paraplegia the patient says that as soon as he feels the inclination to micturate he is obliged to indulge it, so that the urine is apt to escape before he can get to the chamber-vessel. But when the *detrusor* is paralysed, the complaint is that he has to strain for a minute or two before he can pass any water, that it comes away slowly in a feeble stream, and trickles for some little time after voluntary effort has ceased.

On the other hand, when the "micturition centre" in the lower part of

* Traité de la Moelle Épinière et de ses Maladies,' par C. P. Ollivier. 1st Ed., 1821, 3rd, 1837, with plates.

the cord is wholly deprived of its functions by disease or injury, the sphincter and the detrusor of the bladder are of course paralysed together. The result is that the organ becomes distended, and that the excess of urine beyond what it can hold dribbles away through the urethra. The Germans call this condition "ischuria paradoxa." It has often been mistaken for incontinence; an unskilled nurse is very apt to suppose that there can be no retention of urine in the case of a patient who is constantly wet, and her statements may mislead a careless practitioner; but one can always avoid such an error by passing one's hand over the lower part of the abdomen. In real incontinence of urine the bladder keeps firmly contracted, and almost perfectly empty. I believe that its walls invariably become greatly hypertrophied. It seems to be still doubtful whether this symptom always depends upon an exaltation of the reflex activity of an intact lumbar centre, or whether it may occur after that part of the cord has been destroyed, as the result of contractions of the detrusor, which one could either regard as spontaneous or suppose to be dependent upon minute ganglia connected with the vesical nerves.*

Rectum and genitalia.—Some other points connected with the pelvic organs may be conveniently mentioned here. One of them has reference to the *rectum*. It is well known that the peristaltic movements of the intestine are not directly dependent on the spinal cord. But the sphincter ani is under the control of a lumbar centre. Consequently, as a rule, it becomes paralysed with the bladder; and incontinence of the fæces is generally associated with retention of urine.

Another point concerns the male genitalia. *Priapism*—a more or less complete unconscious erection of the penis—is of frequent occurrence in paraplegia. It is one of the results of the reflex activity of the lower part of the cord. Sometimes it is dependent upon distension of the bladder; sometimes it is directly excited by the operation of catheterism. Goltz, in his experiments on dogs, traced out carefully the various ways in which it could be induced by irritating the skin of the abdomen, or of the thighs, or the sheath of the penis. He also found that he could easily inhibit this reflex action by simultaneously irritating the sciatic nerve, or by pinching the hind foot. The last observation—showing that the centre for erection is very sensitive to disturbing influences—may serve to explain the fact that a temporary loss of sexual power is not infrequently observed in persons who have been in a railway accident, but in whom there are no other symptoms of serious spinal injury. When the lumbar enlargement of the cord is destroyed, priapism is of course altogether absent.

As regards the female pelvic organs, the only fact worthy of mention seems to be that *parturition* may take place naturally in a woman suffering from paraplegia, provided that the lumbar centres remain intact.

The urine in paraplegia.—We have still to consider certain changes in the urine itself, and in the organs that secrete and expel it, which are of the highest clinical importance. Sir Benjamin Brodie many years ago pointed out (in the 'Med.-Chir. Trans.' for 1836) that in cases of injury to the spine the urine is often voided alkaline, ammoniacal, and turbid, even as early as the second or third day; it is now well-known that in most cases of disease of the cord, attended with paraplegia, the same thing occurs,

* See a paper on these intra-vesical ganglia by Dr Francis Darwin, 'Quart. Journ. of Micr. Sci.,' 1874, p. 109.

but generally not until a longer time has elapsed. Its explanation is still a matter of doubt. Charcot believes that it is in some way due to a direct action of the nervous centres upon the urinary organs. Most of the other writers who have recently expressed opinions upon the subject think that it is simply a result of decomposition of the urine, after secretion, while it is stagnant in the cavity of the bladder. Such a view must not be taken to mean that only those patients who have complete retention pass urine which is alkaline and fœtid, for there can be no doubt that a very slight impairment of the power of the bladder may prevent it from emptying itself completely. But the difficulty, at the present day, is to understand how the change in the urine is brought about, apart from the access of air. Traube, of Berlin, was, I believe, the first to suggest that a badly-cleansed catheter might set up decomposition in it; and the importance of employing antiseptic precautions before one introduces any instrument into the bladder cannot be overstated. But I have seen more than one case in which catheterism was never necessary, but in which the urine nevertheless became putrid.*

Moreover, although we may admit that decomposed urine is capable of exciting inflammation of the bladder and of the urinary passages, it does not appear that there is always such an interval of time as would be required for the production of this effect. Charcot cites cases in which the urine contained blood as early as the third and the fourth day respectively. And the presence of blood is generally followed in a very few days by that of pus, or of the gelatinous substance which is formed out of pus in a strongly alkaline liquid.

On *post-mortem* examination, in cases of this kind, one finds the most intense cystitis and suppurative nephritis; and, indeed, these affections have most commonly been the direct cause of death. The mucous membrane of the bladder is ecchymosed or swollen, infiltrated with inflammatory products, lined with a diphtheritic layer or with a granular deposit of phosphates, or actually sloughing. Its muscular wall is often free from hypertrophy, but sometimes I have found it greatly thickened; once it measured at least one twelfth of an inch across, although the disease had lasted only four or five weeks. The kidneys are enlarged, intensely congested, and full of suppurating points and streaks; in other words, they present all the characters of an "ascending inflammation."

Bedsores.—A circumstance which points strongly towards the conclusion advocated by Charcot,—that morbid changes in the urinary organs are at least sometimes directly dependent upon an influence transmitted to them from the nervous centres,—is that their development is commonly coincident with that of a gangrenous condition of the skin over the upper part of the sacrum and the gluteal regions adjacent. An "acute bedsore," in fact, sometimes begins to form as early as the fourth day. Many instances of this kind are quoted by Charcot; but perhaps there is not one more striking than a case recorded by Sir William Gull, of a man who on November 22nd, 1856, was working in the docks, when he felt a sudden pain in the back after lifting some deals; he became paraplegic on the 24th, and on the 26th was admitted into the hospital with a small bedsore already present. It is clear that in such cases the affection cannot be regarded as a mere result of pressure upon the skin, nor of the irritant action of excreta passed

* I do not know whether the difficulty is met by a suggestion made by Dr Goodhart, in the 'Guy's Hosp. Rep.' for 1874, that an almost stagnant column of urine filling the urethra would suffice to enable organisms, without which it is believed that putrefaction does not occur, to pass into the bladder.

without the patient's knowledge; but there seems to be no doubt that each of these conditions is concerned in the production of the more chronic bedsores which do not appear until after the lapse of some months. An acute bedsore may sometimes be made to heal, if carefully attended to; this occurred in the case which I have just quoted; but afterwards another one formed at the same spot, and it rapidly increased in size, so that, when the disease ended fatally, six weeks from its commencement, the whole of the sacrum was exposed. Indeed, this affection may directly destroy life, either by exhaustion, or by pyæmia, or by extension of putrid inflammation into the spinal canal, when the dura mater has sloughed.

The morbid conditions that give rise to paraplegia fall naturally into two groups. The first includes all those affections which have their seat *within the cord itself*. The second is made up of those in which the primary lesion is *outside the cord*, and subjects it to *slow compression*, so as to annihilate its functions. The division is not exhaustive; but it is one of practical importance, because in the second group there is a characteristic chain of symptoms, dependent upon the interference with the nerve-roots at the level of the lesion.

MYELITIS.—Among the affections beginning within the spinal cord itself and capable of causing paraplegia, the most important and by far the most frequent is one which is now believed by most pathologists to be a more or less diffused inflammation of its substance, and which they therefore term *myelitis*. This, again, presents many varieties in its symptoms and course, and in the morbid appearances to which it gives rise; but in the main they may be arranged into two classes, according as their progress is *acute* or *chronic*.

*Acute myelitis.**—It has long been known that in many cases of paraplegia the cord is found after death to be more or less extensively softened; and from the time of Ollivier, the opinion has been maintained by most pathologists that the morbid process in question is of an inflammatory nature. There are, indeed, obvious objections to such a view. It involves the admission that a remarkable contrast exists between softening of the cord and a similar change in the brain. The latter (as we shall hereafter find) is most frequently the result of a deficient supply of arterial blood to the affected part of the cerebral substance; and, when it is inflammatory, it is almost always traceable to some definite local irritation. The former, on the other hand, is very seldom secondary to any interruption of the blood-supply, and its chief causes are of a general kind, such as functional exhaustion, exposure to cold, &c. A more serious difficulty is that the histological appearances correspond only partially with those which are seen in inflamed tissues generally, and that even the naked-eye changes are not altogether such as are met with in the spinal cords of animals, when inflammation has been artificially set up in them. Leyden, for example, induced myelitis in dogs by injecting Fowler's solution of arsenic, and obtained greenish patches of purulent infiltration, surrounded by vascular zones. Now, in human pathology, the presence of anything that can be recognised as pus in the spinal cord is in the highest degree exceptional.†

* *Synonyms.*—Acute softening of the cord—myelomalacia. Red and yellow softening of the cord.

† I find a reference to a case of Jaccoud's as one of the chief instances of it that have been recorded ('Les Paraplégies,' &c., p. 545); but this writer, although he speaks of a "*myelite suppurée*," does not actually say that the material which took the place of the affected part of the cord was proved to be true pus.

On these and other grounds Dr Bastian regards most cases called acute myelitis not as strictly inflammatory, but as a process of softening secondary to thrombosis or other vascular changes.

Anatomy.—The colour of the spinal cord when softened is very variable. Sometimes its tissue is pink or bright red from vascular injection; sometimes it is mottled with small ecchymoses; it may then be so saturated with extravasated blood (hæmato-myelitis) that doubts arise as to whether the case is not simply one of hæmorrhage, an affection, however, of which the occurrence is denied by many modern pathologists, as we shall presently see. Probably all these appearances are confined to an early stage of the inflammatory process. Later on the blood undergoes changes which give to the diseased parts a tawny yellow tint, or if no hæmorrhage has occurred, it may be perfectly white. Whether white softening is ever observed when death has occurred at a very early stage I am not sure. I have not found any instance of it among those cases which have proved rapidly fatal at Guy's Hospital.

But the fact that one part of the spinal cord is softer than the rest, or that it looks diffluent when cut across, is not of itself a proof that disease is present. It may result from bruising with the bone forceps during the autopsy though made by the hands of a practised assistant. The microscope here affords valuable assistance. In the great majority of cases one has only to spread out a little of the softened tissue between slide and cover in order to find with a quarter objective numbers of opaque mulberry-like aggregations of fatty granules, the well-known "compound granule-masses" or "corpuscles of Gluge." The exact pathological nature of these bodies is, indeed, still very uncertain; but there is scarcely any doubt as to their practical significance, as indicating that the part of the cord in which they are detected is in a morbid condition. Leyden, indeed, quotes statements made by different observers that they are normally present in the fœtus, and that they may also be found in persons at an advanced age, or after death from tuberculosis, pneumonia, or anæmia, when no spinal symptoms had existed during life. But at any rate one need not hesitate to regard them as a proof of disease when they are thickly scattered through some parts of a cord and absent elsewhere. The difficult question of their origin may be left to be discussed hereafter, when I shall describe softening of the brain. The most remarkable points about them are that they are observed under such diverse pathological conditions, and are fully developed at so early a period. Thus, there were large numbers of them in the spinal cord of a patient who was under the care of Dr Frederick Taylor in Guy's Hospital in 1873, and who died within eleven days from the commencement of his symptoms, and not more than fourteen days after the occurrence of a slight injury that perhaps set up the disease. In such cases they are not generally accompanied by an obvious infiltration of leucocytes, or by the presence of any diffused granular matter, but lie embedded among nervous elements which to a superficial observer seems to have a normal appearance.

Recent investigations, however, have shown that the histological changes are really far more considerable than would at first sight appear, and it is to be noted that granule-masses are not infrequently discovered when there is no obvious softening, the texture being, of the two, rather firmer than is natural; and, again, when there is no marked change of colour, or, at most, only a pinkish-grey tint, with some blurring of the pattern on a cut surface. Nor is even the failure to detect granule-masses a proof that morbid changes

may not be recognised where the cord has been specially prepared for the purpose. In many cases after it has been soaked in a diluted solution of chromic acid for two or three weeks one can see at a glance what parts of it are diseased, since they remain colourless instead of turning green. And a microscopical examination of thin sections shows that the nerve-fibres have their axis-cylinders swollen and varicose, that the ganglion-cells are enlarged and granular or perhaps vacuolated; and (according to Erb) that the network of the neuroglia is thickened, while its cells are increased in size and augmented in numbers, and sometimes contain more than one nucleus. Erb also says that a colloid substance is sometimes found diffused along the course of the swollen connective-tissue bundles, and Leyden that a scanty infiltration of leucocytes is now and then seen in the interstices between the nerve-fibres. Such appearances are doubtless conclusive as to the inflammatory nature of the process, and so is the presence of recently effused lymph in the tissue of the pia mater or in the subarachnoid space. This I have myself found in sufficient quantity to be plainly visible to the naked eye, and it probably might be discovered with the aid of the microscope in many cases in which it has hitherto been overlooked.

Acute myelitis is never limited to the anterior or posterior cornua of the grey substance of the cord, nor to any of its columns. It is a "diffuse" or "indiscriminate," not what the German pathologists call a "system-lesion," or, as we may say, a "columnar" disease. It may begin from the surface, secondary to meningitis, or it may occupy the grey matter around the neural canal ("central" or "periependymal" myelitis), or it may affect the entire breadth of the cord, including both cornua and columns (transverse or segmental myelitis); or lastly, it may spread over large portions at once as a progressive ascending myelitis.

*Chronic interstitial myelitis.**—The disease which I have just been describing does not in all cases run such a course as might seem at first sight to warrant our speaking of it as an *acute* inflammation. Sometimes, indeed, it sets in with fever and ends fatally in a few days, but in many instances it is unattended with febrile disturbances, and goes on for many months before it terminates, either in the death of the patient or in his more or less complete recovery. What then justifies its name is the fact that its onset is more or less sudden and its development rapid, the paralysis being complete within a week or two.

In marked contrast with that affection—even in the most protracted cases—is one which always begins insidiously and gradually, of which the duration is never less than two or three years, and which, instead of leading to a softening of the cord, rather renders it firmer and harder than before. The naked-eye appearances in this form of myelitis are variable. Sometimes it is altogether impossible to distinguish those parts of the cord that are diseased from those that are healthy; in such instances no morbid change was recognised until the accurate microscopical investigations of the last few years revealed its presence. In other cases the substance of the cord is obviously tougher than natural. Ollivier long ago very aptly compared it to boiled white of egg; the knife meets with resistance in cutting through it, and the exposed surface is smooth and even. On close scrutiny it often looks gelatinous and has a greyish or greyish-yellow tint, instead of the milk-white colour of a healthy cord. It is rather shrunken than increased in size. When held up to the light the diseased parts are

* *Synonyms.*—Sclerosis of the cord. Grey induration.

translucent. When stained with osmic acid they are untouched by the re agent. When stained with carmine they take the colour much better than the unaffected white columns.

With the aid of the microscope the neuroglia is found to be thickened and to have a fibrillated texture, or even, in extreme cases, to be converted into a dense mass of connective tissue with delicate, parallel, wavy fibres. Its cells and nuclei are increased in numbers, and are far more conspicuous than in the healthy spinal cord. Cells possessing a large number of radiating processes (Deiters' cells) are often seen; it is said that similar cells may be discovered in the normal neuroglia, but they are at any rate much more obvious in cases of chronic myelitis. The nerve-fibres are generally atrophied; they have lost their medullary sheaths, and their axis-cylinders are described as having a hard, glistening look. The ganglion-cells are shrivelled and granular, or may be converted into homogeneous, bright-looking, angular bodies without processes, and having apparently no nuclei, so that they can hardly be identified. At the same time granule-masses are present, often in large numbers, but sometimes very few. Corpora amylacea are generally abundant. The smaller blood-vessels are thickened.

This morbid process is now generally known as "sclerosis." It is not, however, peculiar to cases of diffused or segmental chronic myelitis, such as are attended with paraplegia, but lies at the foundation of several other affections of the spinal cord, which will be described separately, because their limitation to special parts of its substance gives them an independent place in nosology. In the cases now under consideration, the lesion so far extends over the segmental area of the cord, that it interferes with or altogether prevents the transmission of motor impulses from the brain to the parts below. Its longitudinal extent may, of course, vary greatly. Sometimes it is confined to a single spot, and it is often spoken of as "chronic transverse or segmental myelitis;" sometimes it creeps slowly along the entire length of the cord (generally from below upwards) until it may even reach the bulb.

Ætiology.—According to Erb, inflammation of the cord is more frequent between the ages of ten and thirty years than in older persons. But I find that twenty-five consecutive cases of fatal primary diffused myelitis at Guy's Hospital were pretty uniformly distributed over the several decennial periods from ten to fifty, and that three of them occurred in patients between fifty and sixty years old. Most cases occurred in men, the proportion being 19 to 6. This, however, depends, not on the existence of any predisposition to the disease in men, but on the fact that they are much more exposed than women to its various exciting causes. For, although myelitis is certainly not always due to any one morbid influence, its direct ætiology can often be traced with remarkable accuracy.

Sometimes it is clearly the result of *exposure to cold.* Several instances of this have been recorded, and our records at Guy's contain others scarcely less striking. Thus in 1876 a custom-house officer was admitted into the hospital with nearly complete paraplegia, who had been well until eight days previously, when he was obliged to remain for twenty-four hours in a barge, where everything was soaking wet. On the very next day he was attacked with pains in the joints, which he believed to be rheumatic; his urine began to run away from him; and two days later his feet and hands and back already felt numb. Another patient, who was brought in on January 29th,

1875, had on the 18th of that month got wet and caught cold; three days afterwards he passed his water into his bed without knowing it, and his legs became numb and powerless. Among the conditions which are mentioned as especially apt to set up myelitis are sleeping on the damp ground, or in snow, and prolonged immersion in water, as when a person narrowly escapes drowning.

Great *bodily fatigue* also seems often to play an important part in the ætiology of the disease. When it occurs in soldiers on active service, as is said to be frequently the case, it may either be due to exhaustion of the spinal cord from forced marches, or to exposure to cold and wet, or to both causes combined. A case in point has been recorded by Sir William Gull ('Guy's Hosp. Rep.,' 1856). A young man, after walking twenty-eight miles to seek for work, passed the night of July 8th, 1855, in a brickfield. On the 15th he walked thirty-two miles; the day was wet, and he allowed his clothes to dry upon him. Next day he felt as well as usual, but in the afternoon, while he was sauntering in a garden, his legs suddenly gave way under him, and he fell down. He was admitted, six days later, completely paraplegic. He was healthy and florid looking; but it should be stated that he had, about a fortnight previously, been unable to pass his water for a period of twelve hours. He died in less than a month.

A great muscular effort, such as often causes a pain in the back, is now and then followed, after an interval of a day or two, by paraplegia. An instance of this has already been cited (at p. 444) in the case of the dock labourer. Another example of it occurred in Guy's Hospital in 1863 in the person of a railway porter, aged thirty-two, who was pushing a carriage with his back, when he suddenly gave a jump to escape falling into a pit, and so ricked his spine. For a moment he felt powerless, but he afterwards resumed his work, and worked as usual on the next day. The day after that, however, he was walking in the street when he suddenly became paralysed and fell down. Each of these cases terminated fatally in about six weeks; at the autopsy no injury to the bones or the ligaments could be discovered; but in the dock labourer there was softening of the cord in the dorsal region, the affected part having a greenish or brownish colour, no doubt from admixture of a small quantity of extravasated blood. The microscopical examination revealed no trace of any inflammatory exudation; but I do not doubt that, if modern methods of investigation could have been used, we should have been more successful.

Violent mental emotions,—of terror, grief, or anger,—are mentioned as possible causes of myelitis by some writers. Thus Leyden met with a case of paraplegia, apparently due to acute myelitis, which appeared to be caused by fright from the breaking out of a fire.

Again, it occasionally happens that paraplegia develops itself as a sequel or complication of an *acute disease*, especially during convalescence; and in cases of this kind, myelitis has been discovered on post-mortem examination. Virchow has recorded ('Ges. Abhandl.,' p. 683) an instance in which chronic myelo-meningitis came on a few months after recovery from enteric fever, and seems to have thought that this occurrence was more than a coincidence. Westphal examined the bodies of two patients, each of whom became paraplegic during the eruptive stage of smallpox (variolois), and in both cases he found disseminated myelitis. Either pregnancy or the puerperal state, too, may be attended with paralysis of the lower limbs; and it would seem that the cord has been found softened. But the most remarkable case of all,—if

it was correctly interpreted,—is one which Baumgarten supposed to be due to infection with the poison of the epizootic Splenic Fever; it ended very quickly in death, and he found the characteristic bacilli in the blood as well as in the spinal cord ('Arch. f. Heilk.,' 1876).

Erb and many other authors believe *Syphilis* to be an undoubted and important cause of myelitis. He states that the affection may then run either a very rapid or a chronic course. Of cases of the latter kind he saw at least a dozen in the course of a few years, and some of them came to an autopsy. This question, however, I shall have to discuss further on (p. 464).

Lastly, there is reason to believe that *sexual excesses* play a considerable part in the ætiology of diffused inflammation of the spinal cord, although it is very difficult to say exactly how far this influence extends. Men who have been indulging their passions too freely, especially if they are weakly or very young, often experience pains in the back and limbs, which seem to be due to exhaustion of the lower spinal centres, and not merely to fatigue of the muscles. And it is probable that these symptoms may after a time pass into those of actual myelitis. Whether coitus in the erect posture is especially injurious I do not know, nor whether onanism is so, except in so far as it is apt to be repeated frequently, without instinctive desire, and at an early period of life. On these points there is no evidence, nor can one desire to collect it, since, whatever the physical injury of such practices, it is far outweighed by their moral evils.

Symptoms and Course.—I must remind the reader that on grounds of practical convenience I have set aside for separate description certain groups of cases which are included under myelitis by the morbid anatomist, namely those of secondary softening due to direct extension from some affection of the parts outside the cord ("compression-myelitis"), and also those of sclerosis limited to certain definite tracts within the cord, or disseminated in a number of isolated patches throughout its substance. I am at present confining myself to the *primary* form of acute myelitis, and to the more or less *diffused* forms of chronic myelitis. Now, in such cases the principal symptom is paralysis of parts below the seat of lesion. Indeed, the whole account of paraplegia above given (pp. 435—445) may be taken as applicable to the disease now under consideration.

Beside, however, causing akinesis, impairment of sensation, dysæsthesiæ, disorder of the reflex functions of the cord, and the like, myelitis is often attended with another set of symptoms, which I have hitherto avoided mentioning, namely, *painful sensations* referred to various parts of the body. In some instances the seat of pain is in the back; it there varies greatly in intensity; it may either be confined to one or two spinous processes, or diffused along the whole length of the cord; there may be extreme tenderness on pressure, or this may be altogether absent. Occasionally spinal pain may be the earliest symptom of the disease, but the notes of cases which I have before me seem to show that this is rare. In other instances the pain is referred to the front of the chest, or to the epigastrium, or to one or more of the limbs. One patient, a girl of nineteen, was attending as an out-patient for pain in the chest, when she one day fell down in the waiting-room, struck with paraplegia. Another, a man aged forty-nine, had complained of a fixed pain in the left hip for eight weeks before any definite sign of spinal mischief showed itself. A third, a man aged fifty-one, suffered at first from severe burning pains in the soles of the feet, which continued night and day for a considerable time. These cases all terminated fatally,

and in each of them the bones and ligaments and membranes were found to be healthy, and the disease to be limited to the substance of the cord itself.

Sometimes, when pain is referred to the fore part of the body it is accompanied by a sense of constriction, as if a cord were tightly bound round the waist, or as if the chest were fixed in a vice. We shall hereafter see that this symptom, to which German writers give the special name of *Gürtelgefühl* (girdle-feel), is particularly marked in that other form of paraplegia due to compression of the cord by disease of the surrounding structures. But I believe that there can be no question as to its occurring now and then in cases of primary myelitis. On the other hand, I must insist on the fact that a large majority of cases of this kind are altogether unattended with pain, whether in the back or elsewhere.

The *course* of myelitis differs very widely, not only according to the nature and seat of the morbid change in the cord, but also in individual cases which appear to resemble one another closely. Erb speaks of the disease as sometimes setting in with malaise and fever, and even with rigors. A case in point occurred at Guy's Hospital in 1872. A man aged fifty-one, who was much exposed to weather, one day began to shiver, and was attacked with severe pains running down the back of the thighs and calves, and the insides of the arms. He remained in bed for four days, after which the pains left him, and he went to work again. Next day he lost all power in his limbs, and he died of dyspnœa about eight days afterwards. Paralysis often shows itself suddenly in that form of myelitis which leads to "softening" of the cord. Indeed, as Erb remarks, the rapid development of this symptom affords the chief justification for our calling cases of this kind "acute," since they are often unattended with febrile symptoms, and their duration may be prolonged over a period of many months. In most cases of acute myelitis however, death takes place in a few days or two or three weeks. It is comparatively rare for the disease to be prolonged in a chronic form, and then also it is almost always fatal.

Prognosis.—Until within the last few years there were no grounds upon which writers could speak with confidence of the possibility of complete recovery from myelitis. It was well known that many patients suffering from paraplegia got perfectly well, but whether inflammation of the cord existed in such cases was uncertain. The discovery of the remarkable power of regeneration possessed by nerve-fibres did much to clear up the doubt. Eichhorst and Naunyn found in experiments upon young dogs, in which they cut through the spinal cord in the lower dorsal region, that the affected part ultimately underwent partial repair, nerve-fibres with double contour being developed in small numbers in the new material which filled up the gap, while motor power and sensation were to some extent regained. These results have not been confirmed by the more recent investigations of Goltz and Freusberg ; nor in any case could one lay much stress upon them, so far as human pathology is concerned, since it is certain that in man the cord is very far from possessing such powers of restoration. But, on the other hand, I shall presently cite an observation made by Michaud, which shows that, in spite of the occurrence of well-marked myelitis, recovery from compression-paraplegia may take place, though the affected part of the cord continues to display obvious morbid appearances. Surely this case may be taken as proving the possibility of the complete subsidence, with return of normal function, of those local and slight forms of myelitis which may fairly be supposed to be present in partial and transitory forms of spinal paralysis.

Such a conclusion is further supported by the fact that the recovery is often incomplete in cases of this kind; since it is more reasonable to suppose that an organic affection of the cord should in some cases subside than that a merely functional disorder should in any case be permanent.

However this may be, it is certain that, as a rule, the prognosis must be unfavourable in cases of complete paraplegia, with paralysis of the sphincters, which depend on a primary myelitis. But it is another matter if the paralysis is incomplete or limited to certain muscles.

Diagnosis.—The decision that myelitis is the cause of partial or complete paraplegia is generally effected by a process of exclusion. We have first to determine that the paralysis is more than functional, and that the original seat of the disease is in the cord itself, and that the case is not one of compression of the cord, nor of meningitis. I cannot, however, discuss the grounds upon which these inferences must be based until I shall have described the diseases in question. Secondly, we have to decide *what* affection of the substance of the cord is most likely to be the cause of the paralysis. Now, myelitis is at once the most frequent of these affections and by far the most varied of them all in its symptoms and course. But I have already virtually admitted that doubts still prevail as to which cases should, and which should not, be set down as due to myelitis, at least so far as concerns the less fatal forms of paraplegia. I therefore think that the only convenient way of dealing with the subject of diagnosis will be for me here to interrupt my account of myelitis, and to introduce, as briefly as may be, descriptions of the other morbid conditions, seated in the cord itself, with which it may be confounded.

Neurasthenia Spinalis. — "*Spinal weakness.*"— Among the patients who seek medical advice, believing that they are suffering from spinal disease, there are some—chiefly youths and men not far advanced in adult life—whose principal complaint is of muscular fatigue and weakness. If they attempt to walk far, they experience aching pains or stiffness in their limbs. When they have to stand for more than a short time, their legs tremble and seem to give way under them. They feel tired and weary all day long, even before they get out of bed in the morning. On account of the resemblance which such cases undoubtedly bear to some others in which there is early mischief in the cord, it is desirable that a special name should be given to them; and that of "Spinal Neurasthenia," originally proposed by Drs Beard and Rockwell in 1871, appears to be suitable. It has recently been adopted by Erb, who in 1876 had already collected notes of more than two dozen cases. Most of them occurred in persons belonging to the middle or upper classes and to families with marked tendencies to suffer from nervous disorders. The exciting causes appeared generally to be either over-exertion of body or mind (especially if carried on during the hours that ought to have been devoted to sleep) or the various forms of sexual excess. Among the symptoms were an "irritable weakness" of the generative organs, sometimes a little dribbling of the urine after micturition, and very rarely a slight sensation of numbness or formication in the feet. In some cases dragging or tearing pains in the limbs were complained of; but generally these were not severe. Very often there was a peculiar pain in the back, seated apparently in the muscles, increased by movements of the spine or of the scapulæ. Sometimes local tenderness of certain spinous processes was present, exactly as in the affection which will hereafter be

described as "rachialgia" or "*spinal irritation*;" it may then be said that the two neuroses were combined in the same patient. Often there was functional disturbance of the brain, indicated by sleeplessness, timidity, and hypochondriacal depression of spirits.

In fact the condition is that long recognised as a form of hypochondriasis, or, as it used to be called, tabes dorsalis.*

It is supposed by Erb that the state of the nervous elements in the spinal centres in such cases is in fact the same as in the healthy cord when exhausted by the discharge of its functions. The difference is that a natural period of rest fails to restore to them their vigour and activity. This view appears to me to be very reasonable.

The *diagnosis* from myelitis and from other serious spinal diseases must be based mainly upon the absence of objective symptoms of a definite lesion of the cord, in contrast with the vehemence with which the patient complains of his subjective sensations.

Spinal neurasthenia sometimes develops itself in the course of a few days, but much more frequently its advent is gradual and slow. It may reach such an intensity as to compel the sufferer to give up his occupation and to renounce all society. It is often obstinate, lasting for many months, or for years. Relapses sometimes occur. Whether it ever is incurable, or passes on into any organic affection, is as yet doubtful.

Treatment is directed entirely to the general health, and away from the spinal symptoms. Cold sponging, muscular exercise, abstaining from liquor, and the exhibition of steel, strychnia, or cod-liver oil are the successful measures.

Anæmia of the Spinal Cord.—Unless the modern interpretation of a very old observation is incorrect, one of the possible causes of paraplegia is an arrest of the arterial blood supply to the lower part of the cord. Nicolas Steno in 1667 described, and Haller afterwards confirmed, as a result of tying the abdominal aorta in animals, that the lower limbs became powerless, and remained so until the ligature was removed. This experiment was long afterwards again repeated by Sir Astley Cooper and by others; but until 1869 the explanation universally accepted was that the muscles were paralysed because they and the peripheral nerves were deprived of blood. Schiffer, however, has recently shown that if this were the case an interval would elapse, instead of the effect being observed a few seconds after the operation. Whether a similar form of paralysis is ever seen in man as the result of disease appears to be somewhat doubtful. The most frequent cause of obstruction of the abdominal aorta is embolism; but in that affection, as might be expected, the clot becomes impacted close to the bifurcation of the vessel—too low to interfere with the circulation in the lumbar enlargement of the cord.

In the 'Guy's Hospital Reports' for 1857, Sir William Gull gave the clinical history of a patient whose aorta was from some cause or other obstructed, and who was attacked with paralysis which, as he thought, was peripheral, rather than spinal, in its origin. The man was a shipwright; and in the beginning of March, 1855, while apparently in perfect health, he was suddenly seized, being in a stooping position, with pain in the loins. This went off after he had rested for a few minutes; but on his resuming

* The cases cited in a work by Surgeon-Major Neale, published under this title in the year 1806, are many of them of this character.

his work it returned and extended down the legs, with a sense of numbness, soon followed by entire paralysis, both of sensation and motion, from the loins downwards. The sphincters were involved. After a few days sensation returned, and he was able to take a few steps unsupported. He gradually improved, but the legs remained unsteady. The paraplegic symptoms led to his coming under Gull's care in June of the same year. It was found that there was no pulsation in the abdominal aorta, nor in the arteries of the lower limbs. The right superior epigastric artery was already distinctly enlarged, and could be seen pulsating. In the course of the two following years he regained power to walk tolerably well, but at the end of that term his muscles were still thin and his gait was languid, and from time to time he had slight returns of weakness and numbness in the legs. His feet also were cold and damp. Leyden has suggested in reference to this case that a sudden obstruction of the abdominal aorta could scarcely have occurred, without more serious symptoms developing themselves, and that a collateral enlargement of the epigastric artery would hardly have been seen at so early a period afterwards. And it must be admitted that the case resembled one of "coarctation of the aortic arch" in many respects; and, among others, in the presence of a systolic murmur at the lower angle of the left scapula. But, on the other hand, the arterial anastomoses over the back and the abdomen underwent gradual enlargement while he was under observation, apparently to a very marked extent. And even if the obstruction had existed for a length of time before the paralytic symptoms appeared, it still may have caused them; a supply of blood adequate to the ordinary requirements of the lower limbs (or, on the other view, of the spinal cord) may have been insufficient for some specially prolonged effort, or when the man was exhausted by overwork or by some other cause. One remark made by Gull is interesting: namely, that "using the legs soon brought on increased weakness and numbness." For an intermittent paraplegia has been noticed in horses, as a result of obstruction of the aorta; a loss of power in one or both of the hinder limbs coming on when they are driven, and subsiding when they stand still. Charcot is said to have observed a similar condition in a patient whose right common iliac artery was obstructed. When this man was walking he was obliged to stop every fifteen or twenty minutes, on account of paralysis of the corresponding limb; after a few minutes' rest the symptoms passed off and he was able to go on again.

Leyden found capillary embolisms in the cord in ulcerative endocarditis, but I am not aware that any impairment of its functions has been traced to them; probably the arteries which enter it are not large enough for obstruction of any one of them to produce clinical effects.

There is some evidence that a defective blood supply to the spinal cord may lead to the occurrence of paraplegia in persons who have suffered severely from hæmorrhage, or who are highly anæmic. Among the cases of this kind quoted by Leyden there is one of a woman, aged twenty-four, who had lost blood profusely after a confinement; she was exceedingly weak, but she was resuming her domestic duties when, at the end of about a month, she suddenly became unable to stand. Another case, published by Dr Moutard Martin, occurred in a man as the result of severe hæmorrhage in the course of dysentery. Others were in persons who had suffered from menorrhagia, but it must have been difficult, if not impossible, to exclude hysteria, which, as we shall presently see, may itself cause paraplegia. The

same source of fallacy exists in reference to those cases which are recorded as examples of paraplegia due to chlorosis in women. I am not aware that such an affection has hitherto been observed in the intense and fatal form of idiopathic anæmia to which men are liable.

But, after all, the mere fact that persons who have lost blood are now and then attacked with this form of paralysis is far from showing that anæmia of the spinal cord is capable of directly producing the effect in question. For the proof to be complete there must be no interval of time during which impairment of the nutrition of the cord could have occurred as an intermediate link in the chain of events. In any future case it would be important to note whether the patient, after the hæmorrhage, took violent exercise or worked hard in any way, using his legs more than his arms, so as to cause the lower spinal centres to make special claims upon the nutrient powers of the blood. We might thus be able to see why the lower limbs should alone be paralysed and not all parts of the body alike.

In his Croonian Lectures before the College of Physicians in 1881 the late Dr Moxon drew attention to the great length and obliquity of the spinal arteries which supply the lower third of the cord, as a probable cause of temporary anæmia and an explanation of its affecting the lower extremities rather than other parts of the trunk and limbs.

Some years ago, Brown-Séquard professed to be able to distinguish paraplegia due to anæmia from that due to hyperæmia by the simple test that, whereas the former was benefited by keeping the patient recumbent on the back, the latter underwent aggravation when this posture was adopted, but was lessened if he lay on the abdomen, or stood upright, or walked. This method of diagnosis has recently been endorsed by Dr Hammond, of New York, who mentions a case which he considered to be one of congestion, and in which "all the symptoms were worse in the morning." But it seems to me that the value of his opinion is greatly diminished by the readiness with which he adopts this kind of hypothetical pathology, assuming the existence of two altogether distinct forms of spinal anæmia—the one limited to the antero-lateral, the other to the posterior columns. The last affection is, indeed, one with which we are all well acquainted under the name of "spinal irritation" (or rachialgia), which I shall describe in the next chapter. Nor could one have a more striking proof that such speculations are baseless than the fact that Stilling and Ollivier had each referred this disease to the very opposite condition—congestion of the cord—for reasons which are neither better nor worse than those of Dr Hammond.

Hyperæmia of the Cord.—Passing now to consider whether paraplegia may depend upon congestion of the cord, I will frankly state my own conviction that we are never justified in making such a diagnosis. All pathologists are agreed that distension of the blood-vessels within the spinal canal, if found in the dead body, affords no proof that they were overfull during life: it may be due either to the mode of death, or, if the corpse lies face upwards, to *post-mortem* gravitation.

There are sometimes good reasons for believing in the occurrence of morbid processes, in spite of the fact that they cannot be recognised by the anatomist. What chiefly leads me to reject the doctrine of a persistent congestion, independently of any mechanical cause for it, is my opinion as to the origin of that doctrine. In the early days of pathology, writers proceeded to assign to all diseases their anatomical lesions in an ascending order of

severity. They found that almost every organ was liable to certain chronic changes which could be regarded as inflammatory, and any cases which seemed to stand lower in the scale were handed over either to hyperæmia or to anæmia, according as the one condition or the other seemed the more easily to be brought into harmony with the various symptoms.

Even now a similar method prevails. In 1866 Dr Radcliffe ('Reynolds' System,' ii, p. 619) had under his care a woman who had been found paralysed in all her limbs the morning after having had her menstrual flow checked by an alarm of fire. Among the other symptoms were tingling in the fingers and toes, some degree of general hyperæsthesia, and a dull burning aching in the limbs and along the spine, without special tenderness on pressure over the vertebræ, but with increased sensitiveness to a hot sponge. The bladder and rectum performed their functions naturally. There was no over-excitability of the reflex centres in the lower part of the cord. Within two or three weeks she began to improve, and at the end of five months she left the hospital convalescent. Considering the circumstances under which her attack commenced, we can see that the hypothesis of spinal congestion is not unreasonable, although other interpretations are possible. It is easy enough to carve out of the whole mass of cases of paraplegia a group presenting the same symptoms as those which existed in Dr Radcliffe's patient and to label them accordingly; but I fail to see any ground for supposing that these cases have more claim to the title of spinal congestion than any others in which the paraplegia is incomplete and terminates in recovery. Dr Radcliffe lays stress upon the sudden commencement as characteristic. Now, it is obviously a question that can be settled by direct observation, whether a paraplegia of which the onset is sudden does really get well more often than one which begins gradually. But in the meantime, I find that Erb lays stress upon a *slow* development of the symptoms as distinguishing hyperæmia from other morbid states of the spinal cord. He also mentions, as one of the most important signs of this affection, its fluctuating and changeable course. I have, however, seen at least one case of myelitis in which inexplicable variations in the severity of the paralysis and of the anæsthesia occurred from day to day.

Reflex Paraplegia.—It has long been suspected that a loss of function in the spinal cord, causing paralysis of the lower limbs, may be an indirect result of certain visceral diseases. Such cases would be fairly comparable with two already mentioned (p. 390) in which paresis of one arm was caused by a carious tooth. Hammond relates the case of a girl who was brought to him on account of paraplegia which had suddenly developed. He administered several doses of santonine, followed by castor-oil; a number of round-worms were passed, and the paralysis disappeared in the night.

Within the last few years there has been much discussion as to the way in which this kind of paralysis is brought about. Mr Stanley, who was perhaps the first to describe it ('Med.-Chir. Trans.,' 1833), was contented to refer it to "irritation, propagated through the sentient nerves to the spinal cord," whence he supposed that "the impression" was "transmitted through both the motor and sentient spinal nerves to the limbs, here occasioning an impairment both of sensation and of the power of motion." Obviously, however, this explanation goes but a very little way. In 1860 Dr Brown-Séquard published in the 'Lancet' an elaborate theory, according to which irritation of afferent nerves of a diseased organ was supposed

to set up a reflex spasm in the blood-vessels of the spinal cord, so as to render it anæmic and impair its nutrition and its functions. In the following year, however, Sir William Gull refuted this opinion in the 'Guy's Hospital Reports,' and no one now accepts it. Nor has a suggestion of Jaccoud's been accepted that reflex paralysis is due to "exhaustion" of that portion of the cord upon which fall the stimuli conveyed by the sensitive nerves of an irritated part. Thus the field remained clear for the entrance of the modern doctrine of "inhibition," which in theory meets all the difficulties of the case satisfactorily. Its application to paraplegia seems to have been first made by Lewisson ('Reichert's Archiv,' 1869). In a series of experiments on rabbits, he succeeded in paralysing the lower limbs by forcibly squeezing between his fingers the kidney, or the uterus, or a large loop of intestine. The loss of power is described as lasting only as long as the pressure was continued, or a little longer, and as always disappearing abruptly. Now, nothing is easier than to refer nervous phenomena of all kinds to "inhibitory" influences; but this very fact makes it essential that the pathologist should never extend the area of inhibition a hair's breadth beyond the limits to which a strict interpretation of physiological observations would confine them. The paralytic affections already referred to as being caused by diseased teeth, and the paraplegia from worms in Hammond's case, were all in perfect accord with Lewisson's experimental results, since they subsided as soon as the source of irritation was removed. Graves placed on record the case of a man who was admitted into the Richmond Hospital in 1835 with partial paraplegia of two weeks' duration, and a tight stricture, which had existed for some months. He had recently been much exposed to cold and wet, and this might have been regarded as the cause of the paralysis. But in a very few days after the first introduction of a catheter a remarkable improvement took place in his legs and in his back, where he had had much pain. In fact, the change was almost sudden; and within a month the power of the lower limbs was completely restored. It would seem that we may fairly regard this as an instance of reflex paraplegia. I believe that cases have been recorded in women in whom the reduction of a displaced uterus has been followed by a marvellously rapid recovery from paralysis; but then it is scarcely possible to exclude hysteria as a cause of the loss of power. On the other hand, the immense majority of cases which are supposed to be of reflex origin run a protracted course. It is true that the disease which is supposed to be the starting-point of the inhibitory influence is often itself permanent and incurable; but sometimes it subsides entirely, and yet the paralysis persists.

In such cases, it is only reasonable to admit that there is something more than a mere reflex paraplegia; and, indeed, myelitis has sometimes been discovered after death. A particular group, in which the paralysis has appeared to be secondary to some affection of the bladder or of the urethra has been known by the distinctive name of "urinary paraplegia." Three instances of it were recorded by Gull in 1856 ('Med.-Chir. Trans.,' xxxix); in each of them a part of the spinal cord was found softened. Two similar cases have come under the observation of Leyden; the paralysis seemed to have started from a vesical affection, the result of a long-standing stricture; there was a limited inflammation of the upper portion of the lumbar enlargement with granule masses. Leyden remarks that the seat of this lesion, corresponding exactly with the spot at which the nerves of the bladder enter and leave the cord, suggests the conclusion that the morbid action had extended along their fibres. This opinion has since been supported

by experiments made by Tiesler and Feinberg and Klemm, each of whom succeeded in generating an inflammation of the lower part of the spinal cord in animals by setting up neuritis of the sciatic nerves. In a dog operated on by Tiesler there was actually a collection of pus in the substance of the cord "at the point of exit of the sciatic plexus." Roesingh, however, has repeated some of these experiments with negative results, so that the question can hardly be regarded as finally settled.

Leyden ascribes most cases of reflex paraplegia to progressive ascending neuritis, and believes that the inflammatory process can be traced step by step along the whole course of the nerves.

Sir William Gull several years ago proposed a different route for the transmission of the morbid action, namely, by the veins. In a man who died of paraplegia, consecutive to a long-standing stricture, he found a small quantity of pus lying outside the sheath of the dorsal part of the cord; and one of the vertebral veins in the lumbar region was full of pus. But it is mentioned that there was a large slough over the sacrum, and, as we now know that a putrid inflammation often enters the lower end of the spinal canal from a bedsore, I think that most probably the suppuration there and the venous thrombosis arose in that way shortly before death, and were, in fact, indirect consequences of the paralysis.

There would be no difficulty in collecting from medical works and journals a large number of cases of supposed reflex paraplegia, but they would need to be most carefully criticised. In the Guy's Hospital Library copy of the 'Medico-Chirurgical Transactions' for 1833, containing Mr Stanley's paper, there is a pencil-note by Dr Wilks, to the effect that the majority of his cases were wrongly interpreted, and were really examples of a primary inflammatory softening of the cord, attended with secondary cystitis and nephritis. There are, indeed, two cases to which this remark does not apply: one is that of a patient admitted into St Bartholomew's Hospital for retention of urine (attributed to a severe gonorrhœa, the discharge of which he had stopped by injections), who became paraplegic and died in about a fortnight; the other, of a man who was getting well of a gonorrhœa when he was seized with paralysis extending up to the umbilicus, and fell back dead in his bed sixteen hours afterwards. But I shall hereafter have to relate instances in which septicæmia set up rapidly fatal cerebral symptoms for which no adequate explanation could be found at the autopsy; and it seems to me likely that the cases which I have just quoted of Mr Stanley's were really of that nature, rather than merely reflex, or due to inhibition of the spinal centres.

Again, when chronic paraplegia develops itself in a person who has for years had a stricture, or who has recently had dysentery or some other intestinal affection, or who has a retroflexed or prolapsed uterus, one must not forget that this may be a mere coincidence. Brown-Séquard formerly laid down a series of fifteen criteria, by which he thought that a reflex paraplegia might be distinguished from one due to an organic lesion in the cord. But now that we know that myelitis is actually present in most of the cases supposed to be of reflex origin, these criteria would have to be set aside, even if experience had not long since shown that no reliance could be placed on them. And I think the only way in which one can exclude the possibility that the relation between the primary disease and the paralysis is purely accidental is to show that the cases in question are too numerous to admit of such an explanation. But can this be shown?

I must for my own part confess that I have never yet had under my care a patient who appeared to me to be suffering from reflex paraplegia.

Hysterical Paraplegia.—It is a well-recognised fact that hysteria is a not infrequent cause of paraplegia; the proof being that young women who have previously suffered, or are actually suffering, from other hysterical symptoms are apt to be affected with paralysis of the legs, which gets well after a time, or sometimes suddenly, under the influence of some mental or moral shock. To define as accurately as possible the characters of such an affection must evidently be of great importance in reference to prognosis and treatment, for the diagnosis cannot be taken for granted upon the mere fact that the patient has hysteria, and (on the other hand) affections really hysterical are occasionally met with in persons who do not present obvious indications of that disease. But there is a remarkable want of agreement among writers as to the kind of paraplegia which is most likely to be due to such a cause. Radcliffe and Bastian say that the paralysis is usually incomplete, Wilks that completeness is, in a doubtful case, an argument for paraplegia being due to hysteria. According to Wilks there is loss of sensation, and most writers agree with him. Duchenne attached much importance to the presence of a diminition of "electro-sensibility" without loss of "electro-contractility;" but Reynolds has related two cases in which tactile sensibility, sensibility to electricity, and electro-contractility were all perfect. He remarks that although each of these patients could raise her legs from the bed, yet when she was made to attempt to walk, no amount of help could prevent her from staggering and tumbling down to within a few inches of the ground, and then she would recover herself without assistance. As regards micturition, Reynolds states that while retention of urine is common in hysteria, loss of power over the bladder and rectum is not generally present in hysterical paraplegia; Wilks, that the patient "at all events does not wet the bed, she merely has retention;" Radcliffe, that "the bladder and rectum are little, if at all, under control; less so, as a rule, than in common paraplegia." Wilks remarks that hysterical girls remain plump in spite of their being affected with paralysis. I remember that Sir William Gull used to point out the coldness and pallor of the surface of the legs in such patients, as contrasting with their condition in cases of organic disease of the cord. Early rigidity is another characteristic of hysterical paraplegia.

Dr Reynolds has described a closely allied functional paralysis, which he called paraplegia dependent on an idea ('Brit. Med. Journ.,' Nov. 6th, 1869).

The treatment of hysterical paraplegia is that of hysteria, and electricity should only be used, if at all, for its effect upon the mind.

Concussion of the Cord.—In ordinary cases of fracture and dislocation of the spine, such as come under the care of the surgeon, the paralysis, which is commonly present from the first, is attributed either to crushing of the cord by a displaced vertebra or to effusion of blood. A case recorded by Gull shows how a comparatively slight accident may be attended by fatal consequences in this way. A man was carrying coals on his back down some cellar stairs when his foot slipped, so that he fell, and the sack of coals upon him. He died in thirty-four hours. Two of the cervical vertebræ were found torn asunder, but this was not the cause of the paraplegia from which he had suffered immediately after his fall, for there was no displacement of the bones nor any injury to the posterior ligament of the spinal membranes.

There were, however, several spots of ecchymosis in the posterior grey cornua of the cord, as well as in the posterior white columns and the posterior half of the left lateral column. It is perhaps worthy of notice that even these lesions did not actually explain the paraplegia, for there is no proof that the motor tracts on both sides were injured. This, however, illustrates exactly the view which should be taken of capillary ecchymoses of the nervous centres, whether cerebral or spinal. It is not that they themselves produce paralytic or any other symptoms, but that their presence, showing that the violence was sufficient to tear asunder vessels, shows also that it must have been enough to lacerate the nervous elements themselves, which are more delicate.

These remarks tend, I think, to explain the fact that spinal injuries are sometimes followed by paraplegia, and may even terminate fatally without any morbid change whatever being discoverable even with the microscope. Leyden says that he met with an instance of this kind in which death occurred within five days, and in which he failed to detect any lesion, even after having hardened the tissues with chromic acid. Such cases are commonly described as cases of *spinal concussion*, and it is probable that they depend upon a molecular change in the nerve-fibres and cells of the cord due to the jarring force of the injury. The occurrence of this change would of course be prevented if the vibrations should in any way be hindered from reaching the nerve-centres. Mr Erichsen, for instance, says that after a railway accident those persons who were sitting with their faces to the engine are less likely to suffer from spinal injuries because they would be thrown forwards and could break their fall with their arms and hands, whereas those who sat the other way would be driven against the back of the carriage when its motion was suddenly arrested so that there would be scarcely anything to prevent the spinal cord from being violently shaken. But I must confess that I am unable to understand another statement of Mr Erichsen's, namely, that persons who are asleep at the time are less apt to be affected than those who are awake. Even if we admit that the functional activity of the spinal centres is diminished during sleep it seems impossible that this can prevent their undergoing the direct physical effects of the shock.

The cases of which one can most confidently speak as being examples of concussion of the cord are those in which a blow or a fall is instantly followed by paralysis, and in which recovery takes place so rapidly that the alternative diagnosis—hæmorrhage—can be satisfactorily rejected. Such an instance is perhaps one related by Erb. A man, aged fifty-five, fell twenty feet from a tree upon his heels and ischia. He was not unconscious, but was at once unable to walk, and had to be carried home. He experienced severe pains in the sacrum and in the lower limbs, but these passed off after a time. There was no loss of sensation, but the legs were said to be entirely motionless for a week. When he came under medical observation at the end of four weeks he could only make a few steps at a time, and slowly and with hesitation. Reflex movements could be produced as usual. The excitability of the nerves and muscles by electricity was much diminished. Galvanic treatment, applied both to the spine and to the legs, was attended with great success. He was soon able to walk well, and in three weeks he was discharged cured.

But in most cases in which railway accidents are followed by paralysis this does not manifest itself until some time has elapsed. The interval is

often of several days' duration. Mr Erichsen says that it may even last two or three months. During this time the patient is not, indeed, well; he is suffering from other effects of the injury, but he frequently has no idea that his spine has been hurt. I really do not see how the diagnosis of "concussion" is to be established as against that of myelitis, when the symptoms are thus remote. However slight they may be, one may surely conceive them to be caused by an inflammation of the cord limited to a sufficiently small area of its substance. And the fact that they generally subside after a time affords no argument to the contrary, now that we know that myelitis is a disease from which recovery is possible.

Acute Ascending Paralysis.*—I have already alluded to the fact that myelitis sometimes spreads rapidly upwards along the cord, and destroys life in a few days. To such cases, which at Guy's Hospital have not been very infrequent, the name of acute ascending paralysis might be fairly applied. Recent writers, however, reserve it for a different affection, one in which the most minute scrutiny has hitherto failed to reveal any lesion of the tissue of the cord. The earliest notice of this form of paraplegia appears to be one by Landry, in the 'Gazette Hebdomadaire' for 1859. It is said to be definitely characterised by its symptoms. For in a case of quickly fatal myelitis there is marked anæsthesia, the bladder and rectum are totally paralysed, bedsores are formed at a very early period, and the faradic excitability of the muscles soon becomes extinguished. But in the acute ascending paralysis of Landry none of these phenomena are observed.

Onset, course, and symptoms.—I take the following description from the account given by Erb in 'Ziemssen's Handbuch.' † Sometimes there are prodroma, consisting of slight febrile disturbance, malaise, dragging and shooting pains in the back and limbs, sensations of numbness and formication in the feet and in the tips of the fingers, and, above all, a feeling of great muscular exhaustion and weakness. The patient may go on complaining in this way for a day or two, or for a week, or even (in one recorded instance) longer. More often no such symptoms arise, the earliest indication that anything is the matter being a loss of strength in the lower limbs, which rapidly passes into complete paraplegia. The feet first become motionless, then the legs, and afterwards the thighs. Soon the trunk is involved; straining becomes impossible during defæcation, and in coughing or sneezing; the intercostal muscles are paralysed in succession from below upwards. At the same time, or even earlier, the hands are affected; the patient is unable to write or to feed himself; his grasp rapidly becomes enfeebled; the loss of power extends to the arms and shoulders. Lastly, the muscles supplied by the upper cervical nerves, including the diaphragm, fail in their turn; the act of swallowing becomes impossible, and death by suffocation closes the scene. Towards the last it is sometimes noticed that the speech is embarrassed, that liquids regurgitate through the nose in consequence of paralysis of the palate, and that the masticating muscles and those of the face are weakened. The pupils are sometimes unequal, and a transitory diplopia has twice been observed. There may also be a very rapid action of the

* *Synonym.*—Paralysie ascendente aigue—Landry's paralysis. Poliomyelitis anterior acutissima was a name given on a wrong diagnosis of its nature, both clinical and pathological. Kussmaul's two cases of Fatal Paraplegia without assignable cause (cited by Ross) were published in the same year, and belong to the same group.

† There is also a full account in Dr Ross's work, and another by Remak, for a knowledge of which I was indebted to Dr Gowers. It occurs in the article "Spinallähmung" (p. 651) of Eulenburg's 'Real-Encyclopädie.

heart; this is believed to depend on the lesion having reached the upper part of the cervical cord.

In some cases the order in which different parts are paralysed is said to be reversed. The fatal illness of the great naturalist Cuvier was of this kind, and terminated in less than seven days. His first symptom, a sensation of discomfort at the epigastrium, appeared on May 7th, 1832. Next morning he experienced a difficulty in deglutition, and in the evening he could swallow nothing, and had marked loss of power in the upper limbs. The paralysis gradually became absolute, and affected the lower limbs also. Such a course of events can only be explained on the supposition that the morbid change (of whatever nature) descends the cord, instead of ascending; and that it is limited to grey matter, leaving the white columns unaffected, which of course contain at each level fibres belonging to all parts of the body below. It might be thought doubtful whether these cases can fairly be brought within the very narrow definition of Landry's paralysis; but they resemble the typical cases, not only in there being no recognisable lesion, but also in the fact that the sensibility of the paralysed parts remains perfect.

For, as I have already observed, what is really most remarkable and characteristic of the disease is the complete absence of all other symptoms, with the exception of the loss of motor power. The affected limbs lie flaccid and free from spasm. At first one can excite reflex movements in them; after a few days this is no longer the case, and the "deep reflexes" are lost; but the electrical contractility of the muscles persists to the last, nor do they ever waste. The functions of the bladder and rectum are unimpaired; there may for a time be retention of urine, but this quickly passes off, so that a catheter has seldom to be used. The patient may complain of slight feelings of numbness, or of formication, but the paralysed parts are still sensitive to touch and to painful impressions. No pains are experienced in the affected limbs, nor is there any tenderness of the spinal column. The patient may be so completely free from discomfort, and from all the ordinary signs of serious illness, that it may be difficult for those about him to realise the gravity of his state. In the case recorded by Landry, M. Gubler, who had the charge of it, thought for a day or two that the paralysis was feigned, nor was there any apprehension of danger when the disease was within eight hours of a fatal termination.

Pathology.—As I have already stated, pathological anatomy has hitherto thrown no light whatever upon the nature of this disease. Westphal ('Arch. f. Psych.,' 1876) examined the spinal cord by the most approved methods in three typical cases, and with absolute negative results. The same result followed autopsies by Vulpian and Hayem.

Prognosis.—The mean duration of fatal acute ascending paralysis is said to be from eight to twelve days. It has been known to destroy life in two or three days, but sometimes it runs on for as many weeks. Such cases, however, end in the patient's recovery. Landry spoke of eight out of his total of ten collected cases (only four of which he had seen himself) as having terminated favourably; but his paper contains no details which might have enabled one to form an opinion as to the validity of this conclusion. Erb says that the disease may stop at any period of its course, even when the breathing is embarrassed and the power of swallowing is impaired. One must therefore be careful not to give a bad prognosis too positively.

Ætiology.—With regard to its causes, nothing is known. It appears to occur mostly in young adults, and more frequently in males than in females, the proportion in Pellegrino-Levi's cases being twelve to four. Some cases have been ascribed to exposure to cold, or to some of the other supposed causes of myelitis described above (pp. 448–9); some have occurred during convalescence from acute diseases, as enteric fever and diphtheria, a statement that must not be taken as referring to the ordinary diphtheritic paralysis, of which the characters are altogether different. Westphal thinks that the affection of Landry may be due to the operation of some hitherto unrecognised poison; Erb suggests that its nature is the same as that of tetanus—an analogy to which one may fairly object that it explains nothing.

Rarity.—Landry's paralysis is of exceedingly infrequent occurrence in England, if I may judge from the fact that not a single typical case has been recorded in Guy's Hospital. Dr Wilks ('Lectures,' p. 274) relates seven cases of acute ascending paralysis, and in three of them the cord was found normal. But apart from the difficulty of accepting negative conclusions before modern histological methods were in use, the presence of anæsthesia, of vesical symptoms and of pain in all these cases, brings them under the clinical category of acute ascending diffuse myelitis. A case in a young man which ended favourably is recorded by Dr Ross, and another which proved fatal; but the latter he regards as extremely doubtful. Other authorities, Bristowe, Buzzard, Gowers, Bastian, Bramwell, describe the disease from French or German accounts, not from their own experience. The early cases of Walford ('Brit. Med. Journ.,' November, 1854), Handfield Jones (ibid. October, 1866), and Harley ('Lancet,' October, 1868), in which last Lockhart Clarke found extensive lesions in the cord, cannot be admitted as genuine cases, and many of those published abroad are either myelitis or subacute atrophic spinal paralysis.

The fully described and probably genuine cases are certainly very few, even abroad, perhaps not thirty. The rapid course, the successive implication of legs, arms, and trunk, and finally of the bulbar nerves, including the portio dura, the absence of marked anæsthesia or pain, the absence of bedsores and rectal or vesico-genital disturbance, the loss of knee-jerk, the absence of atrophy or spasm of the paralysed muscles and maintenance of electrical contractility—make up a striking combination. The usually fatal course and negative anatomical result complete the type.

There is no febrile reaction, but Westphal observed the spleen to be enlarged, and this has been regarded as evidence of the disease belonging to the group of "blood diseases."*

No treatment can be said to be indicated, but the application to the spine of ice, of heat, of the actual cautery, and of other counter-irritants, has been recommended, as well as galvanism, ergot, and other drugs.

ALCOHOLIC PARAPLEGIA.—Under this name Wilks has described a form of partial motor paralysis of the lower limbs, of which he has observed many instances, chiefly "in ladies, who have given themselves up to brandy-drinking." Pains in the legs and anæsthesia are also present.

These symptoms, with the dragging gait from dropping of the toes, the integrity of the functions of the pelvic organs, and the favourable pro-

* An unpublished case of paralysis, recently fatal, at St Thomas's Hospital, resembled Landry's cases: it was that of a man who had been inoculated for hydrophobia, and suggested to Dr Bristowe a comparison with the paralytic form of that disease in dogs and in rabbits.

gnosis under proper treatment, make up a recognisable clinical "disease." Dr Buzzard has suggested that symmetrical and multiple peripheral neuritis is the pathological cause of these symptoms (*vide supra*, p. 432). Dr Gowers takes the same view, and regards as minor cases of the same malady those which have been described as Alcoholic Ataxia and Pseudo-tabes potatorum. This was in fact Wilks's own opinion, for he says that sometimes the symptoms are almost confined to the legs, and resemble in character those of locomotor ataxia.

In Dr Broadbent's remarkable case of alcoholic paralysis ('Med.-Chir. Trans.,' 1884) the cord was found normal, but the nerves were not examined. It was in some respects unique; but the other cases he associates with it agree with Buzzard's in occurring in women, in being chiefly paraplegic, and in the muscles being tender on handling, with in one case at least sharp pains, dropped-wrist, and sphincters unaffected. He compares it with Landry's paralysis.

Syphilitic Paraplegia.—That paralysis of the lower limbs may be one of the remote effects of syphilis has for several years been a well-known fact, but morbid anatomy has hitherto done very little to define the exact nature of the changes which occur in such cases. As to their relative frequency there is some discrepancy of opinion. Dr Buzzard has expressed his conviction that, in a person between twenty and forty years of age, a case of paraplegia, when not associated with Bright's disease or embolism, is (like hemiplegia under the same conditions) in nineteen cases out of twenty the result of syphilis. But in this statement he appears to ignore the important distinction between the pathology of the brain and that of the cord, which is represented by the common occurrence of idiopathic myelitis as compared with the infrequency of primary local encephalitis leading to softening; and I cannot doubt that Heubner is right when he asserts that the *lues venerea* affects the spinal cord far less often than the brain. On the other hand, Buzzard's limits of age appear to be too narrow, for in a case observed by Wilks the patient was a woman of fifty-three, and among the five or six other cases which have been examined in the *post-mortem* room of Guy's Hospital within the last few years, one occurred in a man aged fifty-seven, another in a woman aged forty-seven. But, in truth, this form of paralysis has hitherto been chiefly studied at the bedside. Many years ago Sir Benjamin Brodie said that he had several times seen paraplegia stopped in its course by bichloride of mercury or by Plummer's pill. The inference has since been drawn that the cases in question were syphilitic, although there is nothing to warrant it in Brodie's brief statements concerning them. A similar conclusion has sometimes been based upon the striking results which now and then follow the administration of iodide of potassium for paraplegia, a wonderful instance of which I remember seeing in the practice of Dr Wilks about twenty-five years ago.

The frequency with which nodes are seen upon the inner surface of the skull, pushing the dura mater inwards, at one time led to the supposition that a similar affection of the bony walls of the spinal canal might occur. But in the few autopsies which have been made of late years, since the attention of pathologists has been specially directed to the subject, the vertebræ themselves seem always to have been healthy. In some instances gummata have been found upon the membranes, or growing from them into the substance of the cord. Such cases, however, come properly under the head of Compression-Paraplegia, and I shall therefore refer to them in the next

chapter. A gumma beginning in the interior of the cord is, I believe, an affection as yet unknown to pathologists.

On the other hand, in certain cases supposed to be examples of syphilitic paraplegia, no obvious morbid change has been discovered, or only a softened state of the cord; and, as I have already remarked, Erb mentions syphilis as an important cause of myelitis. In 1878 I examined the body of a man who had died of the effects of paraplegia, and who was said to have suffered from venereal disease seven years previously, this having been followed by an eruption and by recurrent sore-throats, and by the occurrence of a miscarriage on the part of his wife—whom he married after having the chancre. I found a common myelitis with recent slight meningitis, but as there was no other evidence of syphilis in any organ, I came to the conclusion that this affection was probably not syphilitic. A year before, however, I made an observation which seems to me of considerable importance. A man, aged twenty, was actually under treatment by Mr Davies-Colley for syphilis, when he became paraplegic and was transferred to the care of Dr Wilks. He died two months later. The cord in the mid-dorsal region was flattened and soft for about an inch and a half of its length; the antero-lateral columns and the grey matter were especially affected, the latter being of a rusty brown colour. To the naked eye there was no obvious change in the pia mater. But when a piece of it, corresponding with the softened part, was placed on a microscopic slide and examined with a lens, the walls of the arteries were at once seen to be enormously thickened and degenerated. By reflected light they looked like solid, opaque, white cylinders; by transmitted light their tissue appeared black. Neither Dr Goodhart nor I could find any in which the affection was in an earlier stage, so that we might have compared it with that of which Heubner has given so complete a description, as occurring in the cerebral arteries. I think it is very likely that many cases, such as have hitherto been supposed to be examples of softening from syphilitic myelitis, may hereafter be traced to the defective blood-supply consequent on a syphilitic affection of numerous vessels in the spinal pia mater; and it may be that the same lesion will be found to account for those other cases in which as yet no morbid change whatever has been detected.

Hæmorrhage into the Cord. "*Hæmatomyelia.*"—As a primary cause of paraplegia this is very rare; so rare, indeed, that Charcot and Hayem* have recently expressed doubts as to whether it has ever been observed, and have endeavoured to show that in all the cases which have been recorded since those of Ollivier and Cruveilhier as examples of it, there was an antecedent softening, the result of myelitis. This view would dissociate it altogether from the common cerebral hæmorrhage of old people; for the softening which so often precedes that affection is not inflammatory, but depends upon disease of the arteries of the brain, and appears to have no representative among the morbid changes to which the cord is liable. But there certainly seems to be some instances in which extravasations of blood into the spinal cord and those into the brain are strictly comparable with one another. A case in point is one of Jaccoud's—that of a woman, aged sixty-two, who was

* His monograph, 'Des hémorrhagies intrarachidiennes,' 1872, contains a collection of thirty-two cases with copious references. Goltdammer finds about thirty cases recorded of non-traumatic hæmatomyelia, and most of these were hæmorrhagic myelitis ('Virchow's Archiv,' lxxi.)

brought into the hospital with complete paraplegia, which had suddenly appeared four days before. The diagnosis was given that there was hæmorrhage into the lumbar enlargement of the cord. Six days afterwards she was found dead in her bed. The autopsy showed that the grey matter of the cord, up to the highest roots of the lumbar plexus, was occupied by a clot which was beginning to undergo disintegration. The cause of death was an immense effusion of blood into the left lateral ventricle, the crus cerebri, the pons, and the bulb. Erb says that the part of the cord into which hæmorrhage is most apt to occur is the central grey substance. Sometimes the blood is diffused throughout its whole length; in other cases it destroys the entire thickness of the cord at a certain level, so that a bluish discolouration can be seen through the pia mater.

This affection is of more frequent occurrence in men than in women, in persons between the ages of fifteen and thirty-five than in those who are older. A striking instance is recorded by Goltdammer ('Virchow's Archiv,' 1876) in a girl between fifteen and sixteen years of age. She was one day about noon sitting quietly on a chair, when she suddenly experienced a severe stabbing pain in the back between the shoulder-blades, which compelled her to cry out. The pain quickly passed into the right arm, and then into the left arm and the lower part of the chest: at the pit of the stomach it extended round the body like a girdle. She fell from her seat, and at once noticed that she could not move her right leg; half an hour later her left leg also was paralysed; within two hours she was taken into the hospital and was found to be perfectly paraplegic, with anæsthesia up to the nipples, and loss of power over the bladder. Reflex movements could be excited in the lower limbs. The case was diagnosed as one of hæmorrhage into the cord below the cervical enlargement. She lived almost exactly a year. At the autopsy there was found a firm cicatricial mass in the grey and white substance of the upper part of the dorsal region of the cord; the nervous elements were completely destroyed; hæmatoidin crystals and granular pigment were present in large quantity, giving a reddish-brown colour.

Hæmorrhage into the cord, though often fatal, may be completely repaired by absorption of the clot, although this event belongs rather to meningeal hæmorrhage. A remarkable case in a young man which, after severe symptoms terminated favourably, is recorded by Ross (vol. ii, p. 154).

Tumour within the Cord.—For practical purposes one may include under this head tubercle of the cord, just as we shall hereafter classify the so-called "solitary" tubercles among tumours of the brain. Even so, however, the group consists of a very small number of cases in comparison with any of the more common ætiological varieties of paraplegia.

Tubercle in the cord forms a rounded or elongated mass, which seldom reaches any considerable size. Two cases were observed in Guy's Hospital in the year 1870; in one the tubercle was no larger than a pea, in the other it was compared to a small cherry. It is worthy of note that in one of them no sign of the lesion was visible on the surface of the pia mater. I made a series of sections at the usual distances from one another, and missed it altogether; it was afterwards found by Dr Reginald Stocker when cutting across the cord in fresh places. The main substance of a spinal tubercle is firm and caseous; its centre may be softened into a creamy liquid; its periphery is sometimes grey, showing a well-marked lymphoid structure; it is surrounded externally by a soft, vascular, pinkish zone, so that it readily

slips from its bed when it has been cut across. Leyden says that tubercles more often occur in the upper than in the lower part of the cord. But of three cases of which I have notes there are two in which the lumbar region was the seat of the disease, while in the third it was in the lower dorsal region. Two of the patients were women, aged twenty-eight and fifty-eight respectively; one was a man, aged thirty-six. Dr Frederick Taylor has told me of a case that he saw in a child. In two of my cases there was extensive pulmonary phthisis; in the remaining one the peritoneum was tuberculous, and the mesenteric glands were caseous.

A new growth in the cord is probably a *glioma;* for gumma and sarcoma are almost unknown. One that was found in the body of a woman, aged twenty-six, at Guy's Hospital in 1860, was an oval soft mass made up of spindle-cell tissue. They are somewhat apt to undergo mucous softening, (myxoglioma and myxosarcoma); but the most remarkable change that is observed in connection with them is the occasional development of an elongated cavity in the centre of the cord, which looks exactly like a dilated central canal, but which is really not so, as is proved by the fact that in transverse sections the remains of the canal can still be seen in front of it. This condition is described by some recent writers under the name of "syringomyelus." Dr Frederick Taylor has shown a beautiful specimen of it, in which, however, it is doubtful whether the cyst was not a primary lesion, for no very clear indications of the presence of a new growth could be made out ('Path. Trans.,' 1878 and 1884).

The symptoms produced by tumours of the cord vary widely in different cases. As a rule, paraplegia is present and differs in no respect from that dependent on any other cause. But sometimes the paralysis is of a more limited kind. In a case under the editor's care in Stephen Ward (Nov. 1878) the tumour (a glioma) occupied the cervical region of the cord and caused acute hæmorrhagic myelitis around it. The symptoms were paraplegia and cystitis with paresis of the right arm, and the temperature rose to 108·5° F. shortly before death. Theodor Simon has recorded several remarkable cases in which there would appear to have been a complete absence of all symptoms ('Arch. f. Psych.,' 1874).

Diagnosis.—Reverting now to the question of the diagnosis of Myelitis from the various affections now described, we see how obscure and difficult the decision during life must often be. The occurrence of paraplegia in a person known to be suffering from phthisis or any other "scrofulous" disease would lead one to suspect the presence of a *tubercle*, but, as I shall presently have to point out, it would be very difficult to exclude the case of caries of the vertebræ (without deformity) producing compression of the cord. So far as I can judge, there is no possibility of distinguishing the paralysis caused by a *tumour* within the cord from that due to a circumscribed transverse myelitis, unless, indeed, there should be severe and long-continued pain, in which case one would be almost certainly driven into the opposite error of supposing that the lesion was in the bones or in the membranes. I have cited two cases, in both of which *hæmorrhage* was, in fact, correctly diagnosed; but, taking into consideration its extreme rarity, I must confess to thinking that each of them was quite as likely to have proved an example of myelitis. At any rate, it is certain that suddenness of onset is not of itself a sufficient ground for attributing paraplegia to effusion of blood. As regards *syphilis*, the recognition of it as

being positively the cause of a paralytic affection of the lower limbs must be based upon the clinical history and upon the presence of other syphilitic lesions rather than upon any peculiarities in the spinal symptoms themselves. But I have no doubt that, as is the case with all the other remote effects of the venereal poison, there are many instances of syphilitic paraplegia in which the most careful scrutiny would fail to elicit any evidence of their real nature; and, seeing how important it is that such cases should not be overlooked, I think that we are often fully justified in prescribing anti-syphilitic remedies in the dark. There is, however, one positive indication of myelitis that must always be carefully noticed, namely, a slow creeping up of the paralysis and anæsthesia from the thighs to the groins, and from thence to the abdomen. I believe that it warrants a confident diagnosis of the presence of diffuse inflammation of the cord. We have seen that an *acute ascending paraplegia* may be independent of any recognisable morbid change in the cord, and the early stage of acute progressive myelitis is probably indistinguishable from Landry's paralysis; but after a time the integrity of the recto-vesical functions, and the other negative characters of the latter, separate it clearly from myelitis.

Among the other forms of paraplegia which have been enumerated the *hysterical* seems to be the only one that can be placed definitely apart from myelitis. I do not wish positively to assert that all the rest are really examples of a partial or local inflammation of the cord, but I think I am justified in maintaining that, if they are not so, one cannot at present give a more accurate account of them than by saying that they are among the spinal representatives of the Neuroses or functional disorders of the encephalon, of which the true pathology is as yet unknown.

Exceptional cases.—I have spoken of myelitis as occasionally terminating in recovery; but I have also laid down the rule that the prognosis is generally unfavourable when there is complete paraplegia. To this rule, however, there are some remarkable exceptions. Among the most striking that I know of are two which have been recorded by Wilks, in his 'Lectures on Diseases of the Nervous System.' One is the case of a gouty man, aged fifty-two, who had been losing power over his lower limbs and his bladder for a fortnight, so that on admission into Guy's Hospital he could not move his legs, and had partial loss of feeling up to the umbilicus. Reflex movements persisted. The urine, which was ammoniacal, had to be drawn off twice daily. Afterwards he had a feeling of tightness round the lower part of the chest and the abdomen, and he experienced numbness passing down the arms into the fingers. He gradually got worse; and about a fortnight after his admission he became feverish, with a quick pulse, a red tongue, rigors, and hiccough; a bedsore was forming, and the urine ran away from him; moreover, his mind became clouded. Dr Wilks thought that he was suffering from suppurative nephritis and had not many hours to live. For some days he remained in a precarious state, and then his constitutional symptoms abated. He began to regain some degree of power in his legs, and from this time he made a rapid recovery; he sat up in bed, he ceased to require the catheter, he got into a chair, he asked for crutches, he walked about the ward, and finally he left the hospital; this was exactly two months after his admission, and six weeks from the time when his paralysis became complete.*

The other case is that of a woman of middle age, who came in suffering from almost complete paraplegia, which had begun a few days previously.

* This patient showed himself several weeks later, still quite well.

It was accompanied by pain and swelling of the joints—a symptom of spinal disease which I have not yet mentioned, but which I shall presently have to describe. There was some febrile disturbance. She complained of great pain in the limbs, with twitching of the muscles, and of a sense of constriction round the waist. The urine was ammoniacal and was passed involuntarily, and the sphincter ani was paralysed. A bedsore formed, which extended until there was a deep slough on the back. All the other symptoms continued for three weeks, after which she began to recover. She then took tonics and was galvanised; and at the end of five months she was discharged perfectly well. Dr Wilks, who, I may say, rejects absolutely all refined diagnoses of reflex paraplegia and of anæmia and hyperæmia of the cord, confesses that he does not know what was the nature of the disease in these cases.

Treatment.—The question now is, whether we can by medical treatment influence the course of myelitis so as to increase the proportion of cases in which such a happy result occurs. And here I fear that we have very little positive knowledge to guide us. Of late years it has not been the practice in England to adopt very active measures in the acute form of the disease. The modern German plan seems to be to use leeches or cupping-glasses freely, to apply bags of ice along the spine, to rub in blue ointment or to give calomel internally, in fact, to carry out the "antiphlogistic" treatment very much as English physicians used to employ it thirty years ago. In proof of the value of *icebags* a case recorded by Dr Tibbits, of Clifton ('Med. Times and Gaz.,' 1871), is quoted: that of a man suffering from myelitis, which was rapidly spreading upwards, which had already produced paralysis of the arms, and which was attended with a temperature of 103·6°, while the pulse was at the rate of 132, and the breathing at 40 a minute. Soon after trial was first made of the cold applications he became easier; the temperature fell to 99·2°, the pulse to 100, and the respirations to 24. In the course of the next ten days he completely regained the use of his arms, but no recovery of power took place in the parts which were first paralysed, and he died six weeks later of exhaustion from bedsores. The suggestion is that the issue of the case might perhaps have been more favourable if the icebags had been employed earlier; paralytic symptoms had been coming on for twelve days, and there had been complete paraplegia for eight days. Erb recommends blistering the back and even the actual cautery, undeterred by the risk of setting up bedsores, which he fully recognises. In "Landry's acute ascending paralysis" he would adopt the same course, besides rubbing the body with cold water, and similar means.

A point of great importance is that the patient should, at the earliest possible period, be placed upon a water bed. Probably he should not be allowed to lie constantly on his back, but should be shifted from time to time. I believe it is sometimes dangerous to allow him to sit up. Some years ago a man, who had recently been admitted into hospital with symptoms of acute myelitis limited to the lower part of the cord, was taken out to "have his bed made." When put back he seemed much exhausted, and very soon afterwards he died.

The utmost care must be taken to prevent the formation of *bedsores*. The sacral region must be kept dry by being cleansed and wiped with soft towels. Some good may also be done by sponging the skin with whisky, or alternately with hot and with cold water. Or a large piece of felt plaster may be applied, having a hole in the centre, if there should be a spot already reddened. When an ulcer has formed, Hammond speaks in the highest

terms of Mr Golding Bird's plan of placing a thin plate of silver over it, of exactly the same size as the sore; while a wire six or eight inches long is carried from this plate to another, made of zinc, which is laid on some part of the skin above, but separated from it by a piece of wash-leather, soaked in vinegar. Galvanic action is set up; and Hammond states that he has "frequently seen bedsores three or four inches in diameter, and half an inch deep, heal entirely in forty-eight hours." Sir Spencer Wells is said to have obtained no less striking results. I regret to say that I have no experience with regard to this plan of treatment.

Retention of urine must not be allowed to occur, or the bladder to remain distended. A soft French catheter should be used, if interference is necessary, and care must be taken to depress the free end of the instrument between the thighs, so as to draw off the whole of the urine; for if any should be left it will quickly decompose. Probably carbolic oil should be employed to purify, while it lubricates, the catheter. But I believe that the tendency to putrefaction may be completely checked by the administration of salicylic acid, or of some other antiseptic medicine, by the mouth. This practice seems to have been first proposed by Fürbringer. I believe Dr Pavy introduced it at Guy's Hospital, and I have seen excellent results from it. From half a drachm to a drachm must be taken in the course of the twenty-four hours; it often restores the urine from an offensive alkaline condition to one of normal acidity and odour. Increased experience has confirmed this statement. Benzoate instead of salicylate of soda may be used. When pus is discharged in any quantity the bladder should be regularly washed out with a weak antiseptic solution.

In some of the less complete and acute forms of paraplegia there is evidence of the value of *ergot* administered internally; and I have prescribed it in one or two instances, though not with any striking results. Hammond relates a case which he considered to be one of "congestion of the cord," and in which it seemed clearly to be of great service. The patient was a Mr W—, of Tennessee, who had become affected with partial paraplegia, and afterwards with paralysis of the bladder, from exposure to cold and damp. He was unable to walk unless he used crutches and had a man on each side of him holding his shoulder. He also had a constant dull aching pain in the loins, and occasional startings of the legs while in bed. All his symptoms were worse in the morning. A teaspoonful of the extract of ergot, taken three times a day, cured him entirely in a month. He had a relapse a few weeks later, but recovered in ten days under the same treatment. No other medicine was given.

The tincture of *belladonna* in fifteen minim doses is said to be useful in similar cases, especially when there is severe pain in the back.

When there is evidence of syphilis there is little doubt that active mercurial treatment by inunction or otherwise is of great value. In more chronic cases of paraplegia the Liquor Hydrargyri Perchloridi is also of undoubted service.

Many writers recommend full doses of iodide of potassium, not only when there is probability of syphilis, but in most forms of myelitis.

Authorities are agreed that *galvanism* should not be employed early in cases of acute myelitis, but Erb says that it may be used from the very first in the ascending paralysis of Landry. The electrodes should be large sponges placed at a considerable distance apart, one upon the neck, the other over the lumbar vertebræ; they may in turn be moved gently up and down, and

their direction may be from time to time reversed. When there is reason to believe that the disease is limited to a segment of the cord, one pole may be placed on the affected spot, the other over the front of the chest or of the abdomen. Faradisation of the paralysed muscles may be useful if they are flabby or wasted.

Strychnia seems to be valuable only in the later stages of myelitis, and is certainly harmful before. But both electricity and strychnia are often very useful in such cases as raise doubts whether they are hysterical or reflex, or due to concussion of the cord. In spinal neurasthenia their employment from the first is strongly indicated, and in this disease, quinine, iron, and other tonics are also of service, while early hours, good food, and exercise, short of fatigue, in the open air, are essential.

On the Continent certain spas have great reputations for the treatment of the more chronic forms of paraplegia, with what amount of justice it is hard to say. Erb says that hot baths are apt to be injurious, except at very advanced periods of such cases. Among the places most frequently visited by patients suffering from paralysis I may mention Schlangenbad, Ragatz-Pfeffers, Gastein, Wiesbaden, Teplitz. It is said that in proportion as the elevation above the sea is greater, baths can be borne at higher temperatures without ill effects.

HEMIPARAPLEGIA.—There is a curious and rare kind of paraplegia due to a lesion which is limited to one lateral half of the cord, but destroys that half completely at a certain level. As might be anticipated, there is loss of power in that half of the body which is on the same side as the disease in the cord, and the upper limit of the paralysis varies with the distribution of the highest nerves coming off from the part of the cord below, or involved in, the disease. Such a condition may fairly be termed Hemiparaplegia; and this, whether it is limited to the leg or involves also the corresponding arm. But what could not have been foreseen, at least until Brown-Séquard in 1860 demonstrated the fact in animals, one half of whose spinal cord had been cut through, is that anæsthesia is present, not on the same side as the disease, but on the opposite side. That half of the body which retains its motor power loses its sensibility, while the paralysed half has its sensory functions unimpaired or rather exalted. The explanation of the crossed anæsthesia is the fact—which is now after a long contest admitted by most physiologists—that the fibres belonging to each sensory nerve-root decussate in the substance of the spinal cord itself, immediately above their entrance, or at least before they reach the level of the decussation of their motor tracts in the pyramids. This applies equally to the fibres which convey all sorts of impressions,—whether of touch, or pain, or heat and cold; but according to Brown-Séquard, with the curious exception that the fibres belonging to the muscular sense run upward without crossing. Thus, there is said to be loss of muscular sense, and with it, of electro-muscular sensibility, on the paralysed and not on the anæsthetic side of the body. Why there should be hyperæsthesia of the skin on the paralysed side,—the susceptibility to tactile and to painful impressions and to change of temperature being all increased,—is not so clear. The inflammatory action which develops itself in the neighbourhood of the lesion may render the fibres unduly sensitive, or there may be a suppression of some normally controlling influence.

Thus it would appear that no disease of the spinal cord proper can give rise to paralysis and anæsthesia limited to one and the same side of the

body. If a patient is found to have one of his lower limbs, for example, affected in this way, we are justified in concluding that the lesion must concern the corresponding half of the nerve-roots which form the cauda equina—unless, indeed, it is seated above the decussation of the pyramids in the opposite half of the encephalon.

On the other hand, it must not be supposed that every affection of one side of the spinal cord necessarily causes paralysis of the same side, and anæsthesia of the opposite side of the body. This effect is observed only when the abolition of function in that half of the cord is complete or nearly so. I have repeatedly had occasion to insist that all morbid changes in the nervous structures produce loss of motor power more constantly than loss of sensation. Unilateral lesions of the cord offer no exception to this rule. It is quite possible for such a lesion to give rise to complete paralysis of one lower limb, or of the arm and leg on one side of the body, without there being any anæsthesia of the limbs on the opposite side. Such cases were formerly known under the name of "spinal hemiplegia." The explanation of them is that the disease, while it is all included within one half of the cord, nevertheless leaves a part of that half functionally active. The best marked cases are those which will be hereafter discussed as essential spinal paralysis in infants and in adults. Here the anterior cornua are the seat of the lesion.

The vaso motor nerves are said to be affected on that side which is the seat of the lesion, especially when this has occurred suddenly or developed itself rapidly. The temperature of the paralysed limb or limbs is at first higher than that of the corresponding parts by a difference amounting to 1° or 2° Fahrenheit or even more. After a time, however, it becomes normal, and sometimes falls to a still lower level.

With regard to the state of the reflex excitability of the lower spinal centres in cases of hemiparaplegia there is still some uncertainty. On the paralysed side it has in some cases been increased, in others it has been diminished; on the anæsthetic side it has generally appeared to be normal, but occasionally it has been exalted. Erb says that the bladder and rectum have generally been paralysed—always when there has been a sudden traumatic lesion—with either complete retention or complete incontinence of the urine, and involuntary passage of fæces. This appears to lend powerful support to the view that the paralysis of these parts in cases of ordinary paraplegia, due to a bilateral lesion limited to the upper part of the cord, depends on an inhibitory influence transmitted downwards from the diseased parts to the healthy centres below. An analogous fact, which has now been noticed in several cases, is that the muscles on the paralysed side in hemiparaplegia sometimes have their faradic contractility lowered in a marked degree; they may also undergo rapid wasting.

Hemiparaplegia may be due to many different *causes*, but hardly to so many as those which may produce paralysis on both sides of the body alike, and some of the more rare lesions of the cord are far more apt to give rise to it than others which are of more frequent occurrence. Thus it has been observed in patients who have been stabbed in the back by a knife or dagger. One is apt to wonder that a chance wound should make a section of one half of the cord with the accuracy of an incision performed by an experimental physiologist; the shape and size of the spaces between the arches of the vertebræ are possibly such as to prevent a cutting instrument from passing across the median line within the spinal canal.

Now and then a fracture or a dislocation of the spine has, in man, been attended with the symptom in question, and perhaps an effusion of blood into the membranes on one side may cause it. A tumour outside the cord, compressing one half of it, has more than once caused hemiparaplegia. In other cases this form of paralysis has been due to disease limited to the interior of the cord on one side, whether this be an effusion of blood, a patch of sclerosis, or of some other form of myelitis, an intrachordal tumour, or a syphilitic lesion.

It is obvious that of these various kinds of unilateral lesions some must be more apt than others to affect a considerable length of the cord, and at the same time to involve the roots of a greater or less number of spinal nerves, whether within or without its substance. This is a point of some importance, because it leads to the development in many cases of hemiparaplegia of a fresh set of symptoms due to interference with nerve-roots. Thus there is commonly an anæsthetic half-zone of greater or less depth passing round the paralysed half of the body from back to front, and dividing the hyperæsthetic part of the surface below from the normal part above, and the upper edge of this anæsthetic space is said sometimes to present a narrow hyperæsthetic border. The explanation is obvious: the nerves of the region deprived of sensation have had their roots destroyed by the disease; those of the region which is over-sensitive have merely had their roots irritated The other (or anæsthetic) half of the body sometimes also has a narrow hyperæsthetic half-zone, limiting the anæsthesia above, and due to irritation of fibres which have just decussated at the upper edge of the lesion in the cord. In some cases the patient experiences a disagreeable sense of constriction or severe burning or shooting pains round the trunk, at a level corresponding with that of the nerves whose roots are involved in the disease, and such sensations may be limited to one half of the body or affect both sides alike. We shall presently see that exact counterparts for all these symptoms are met with in certain forms of paraplegia which depend on slow compression of the cord.

The *course* taken by hemiparaplegia varies in different cases, according to the nature of the disease that gives rise to it. It very rarely remains stationary. Most commonly it soon undergoes conversion into ordinary paraplegia, as the result of the development of myelitis, which affects the whole thickness of the cord around the original lesion. This, again, often ends fatally, but sometimes, according to Erb, it subsides and allows the symptoms of a unilateral lesion to reappear. These may then persist for years without change, and in certain cases they have been known to terminate in recovery. According to Brown-Sèquard, the power of motion is then regained earlier and more completely than that of sensation. If this is the fact, it is in opposition to what is observed in all other affections, whether of the centres or of nerve-trunks.

RACHIALGIA. "*Spinal irritation.*"—In 1828 Dr Thomas Brown, of Glasgow, drew attention to an affection attended with pain and tenderness in one or more of the vertebræ, and termed it *spinal irritation.* He was followed by Mr Teale, of Leeds (1829), by the brothers William and Daniel Griffin, of Limerick (1834), and by Stilling, of Cassel (1840), the last of whom devoted a volume of 540 pages to the subject. All these observers gave a very wide scope to the definition. They detected tenderness on pressure over certain spinous processes in persons suffering from various neuralgic and other affec-

tions, and they maintained that in all such cases a morbid state of the spine was really the fundamental disease. They even enlarged their definition, so as to include transitory paralytic affections and a variety of other neuroses. In this they were no doubt partly influenced by the name which they had adopted, for it would obviously be difficult to exclude from "spinal irritation" almost any "functional" disorder of the lower nervous centres from tetanus to pleurodynia.

It is not surprising that these views met with much opposition. They were criticised in trenchant style, but very justly, by Romberg, and they have never met with general acceptance in this country. As I have already remarked, Stilling and Ollivier maintained that "spinal irritation" was due to congestion of the cord. More recently Hammond has asserted that the essential condition in that disease is spinal anæmia. The boundary lines between it and other affections have been repeatedly shifted, and, on the other hand, many observers have ignored it altogether. This is much to be regretted, because there are undoubtedly some cases which naturally fall under this head, and which have hitherto found no other place in the nosology. For my own part—while I altogether reject the doctrine that the discovery of tenderness over one or more of the vertebræ affords grounds for referring, whatever other pains or neurotic symptoms a patient may present to a particular morbid condition of the spinal cord—I yet think it of great clinical importance to recognise an independent neurosis, characterised by pain and tenderness in the back, but unattended with any symptoms of organic lesion of the cord, of the vertebræ, or of the cartilages and ligaments. All the most recent writers do, in fact, describe "spinal irritation," but many of them still seem to me to give it too wide a range. I think that they all regard it as an affection of the spinal cord itself. It may, however, be urged that the organic affections which most closely resemble it lie outside the cord and (at least in this early stage) affect only the spinal nerves. I would, in fact, propose to look upon it as a neuralgia (in the sense in which I have defined that term in pp. 417—419) belonging to the posterior branches of the spinal nerves, and perhaps to filaments distributed to the various structures which enclose and support the cord. It is true that the law of Hilton and of Van der Kolk—applied precisely as we shall find necessary for migraine and some other painful affections of the brain—would explain the circumstance that pain should be referred to the cutaneous nerves of the back, even though its starting-point might be within the spinal cord; and such a view as to the nature of "spinal irritation" is undoubtedly supported by some facts which will presently be mentioned as to the transference of impressions from the posterior to the anterior branches of the nerves in cases of this kind. But, after all, this difficulty as to the centric or excentric seat of rachialgia is not peculiar to it, but concerns every other form of neuralgia likewise.

It might be thought that if spinal irritation is a neuralgia, its description would properly have come in the previous chapters, but I prefer to give it in this place on account of the importance of the diagnosis between it and the more grave diseases of the vertebræ and of the cord—just as it is convenient to classify pleurodynia with affections of the chest, and gastralgia with those of the stomach; and the analogy leads me to suggest that, seeing how open to misconstruction the term "spinal irritation" is, we shall find great advantage in discarding it, and in substituting for it the name of

Rachialgia, which was long ago employed by Joseph Frank, and corresponds with those applied to other local neuralgic affections.

Symptoms.—The severity of the pain varies infinitely in different cases. Sometimes there is no spontaneous pain at all; on pressing upon the different spinous processes we may find that some of them are tender; the patient shrinks or complains that we are hurting him, but he may hitherto have had no idea that anything was the matter with his spine. Upon cases of this kind great stress was laid by those who developed the theory of "spinal irritation" in its wildest proportions, but I very much doubt whether any clinical significance can in general be attached to such a discovery. The observers in question were in the habit of directing their treatment mainly to the supposed spinal affection, and applied leeches, blisters, tartar-emetic ointment, over the vertebræ. Romberg, however, tells us that very little was effected by these measures, and in my view of the nature of rachialgia one does not see why attention should be concentrated upon it to the exclusion of the more obvious complaints from which the patient may be suffering.

In other cases there is a dull aching sensation in the affected part of the spine, or even severe pain. Its development is usually gradual. At first it may be felt only after great fatigue or excitement, but as time passes slighter causes excite it, and it is less ready to subside; so that it ultimately becomes constant. It is almost always increased by muscular efforts and by movements of the vertebræ. Thus the patient is unable to walk far, or to stand upright for more than a short time; even sitting at the piano, the writing desk, or the sewing machine may be too painful to be borne.

Sometimes the best way of bringing out the increased sensitiveness of the affected nerves is to pass one's fingers along the spine, so as to press upon the vertebræ in succession; sometimes it is more manifest when a sponge wrung out of hot water is drawn down the back. To the patient this seems to scald at the tender spots, while he experiences no discomfort elsewhere.

There is often a close anatomical relation between the seat of rachialgia and that of any other neuralgia which happens to be present in the same case; the one corresponding with the posterior, the other with the anterior main branch of a spinal nerve. This statement is, indeed, almost identical with one which used to be made, and insisted on, by Trousseau; namely, that a "*point apophysaire*" is discoverable in most instances of neuralgia. Pressure on the tender vertebra will often bring out, or increase, the pain in any other part that happens to have its nerves in an irritable condition.

Thus one of the cases related by Dr and Mr Griffin (at p. 19 of their work) is that of a girl who complained of headaches and pains in all parts of her body. Her whole spinal column was acutely tender. Pressure upon the first or second cervical vertebra caused a pain which shot forwards from the occiput to the brow; a little further down, it excited pain in the larynx; over the lowest cervical spine, at the spot where the trachea dips behind the sternum; and still lower, at the middle of the sternum, the ensiform cartilage, and the pubic region successively. This certainly was not due to any direct mechanical impression upon the cord; for when pressure was made behind the trochanter pain was felt at the iliac crest, at the inside of the thigh, and even in the opposite hip; while pressure upon the thigh or knee set up pains in the shins and toes.

In other cases there is a fixed spot in the front of the body, to which pain is always referred, whenever any one of several tender vertebræ is pressed upon. Thus another of Dr Griffin's patients had great tenderness

of all the dorsal and lumbar, but not of the cervical spinous processes. Pressure on the upper dorsal vertebræ caused pain at the middle of the sternum; from the third or fourth dorsal down to the sacrum it excited pain, not in the corresponding points as usual, but at the ensiform cartilage. Pain at this spot was even brought on by pressure behind the trochanter, upon the muscles of the thigh, or over one knee-joint, and the patient felt the same pain if she chanced to tread on uneven ground, or if a pebble came beneath her feet in walking. To ascertain whether the seventh and eighth dorsal vertebræ were, as usual, more affected than other parts of the spine, firm pressure was made upon them. The result was that she suddenly tumbled forwards insensible, and would have struck her face against the floor had not someone caught her. Such an occurrence might well appear almost incredible; but the same observers have placed on record other similar cases.

Another of their patients was a lady who complained of pain in the face, but who "had no conception that her spine was at all affected." When the second cervical vertebra was touched she sprang up with frightful suddenness, as if a needle had been driven through the cord, and then fell in a state approaching to insensibility. Out of this stupor she twice started up in the same way, and as often dropped back powerless, her countenance evincing the utmost terror and agitation. As soon as she could speak she said that she had felt a numbness and sensation as of pins and needles in all parts above the ensiform cartilage. She would on no account permit her neck to be touched again.

A third instance was that of a young gentleman, who described himself as suffering from a chronic liver complaint. Pressure upon the spinal column was excessively disagreeable to him. When the finger rested on one of the dorsal vertebræ he grew pale and terrified, experiencing a sudden sensation or thrill through every nerve in his frame. He had an unpleasant feeling about the part for the remainder of the day, and shuddered at the idea of allowing the pressure to be repeated. After a few weeks, however, the experiment was tried again, and with precisely the same results.

A fourth case was that of a boy, aged twelve, who fell forwards insensible, as if he had been shot, as soon as slight pressure was made upon the second lumbar spine.

I believe that phenomena somewhat similar, although in a less marked degree, are not uncommonly observed. They undoubtedly lend support to the view that the seat of rachialgia is in the cord itself rather than in the nerves. Anstie met with a case in which pressure on one spot, over the lowest cervical vertebra, caused exquisite pain, a sensation of extreme nausea, and disappearance of the pulse at the right wrist, that on the left side remaining unaltered. The patient, a young lady, was also seen by Dr Walshe and Dr Reynolds. Dr Frederick Taylor has mentioned to me the case of a child, brought to him at the hospital, in whom it had been noticed for a considerable time that pressure upon the seventh cervical vertebra at once gave rise to a marked pallor of the left side of the face, which lasted a few minutes.

Ætiology.—Dr Radcliffe has suggested that the cause of rachialgia (or, as he terms it, "spinal irritation") is often a strain of the back, or a blow, the occurrence of which may have been forgotten by the patient. This supposition seems to me to be strikingly confirmed by the fact that slight railway accidents so often give rise to it. The so-called "Railway-spine" is, in fact,

in the immense majority of cases an affection of this kind. Anstie's patient, to whom I just now referred, had received a very slight contusion in a collision. At the same time her sister was severely injured; she nursed her assiduously for three or four months, and then her own health began to be seriously impaired. Many observers think that no reliance can be placed on the statements of persons who have met with such accidents, so far as their subjective symptoms are concerned. But I have seen several cases in which there was abundant collateral proof of good faith. A general "nervous susceptibility"—if I may use the term—also plays an important part in the ætiology of the complaint. It is far more common in women than in men, and in persons between fifteen and thirty years of age, than in those at earlier or later periods of life. An inherited "neuropathic tendency" predisposes to it. Like other neuralgiæ, it often affects women who at the same time are obviously suffering from hysteria; and thus it may be associated with any of the varied symptoms of that morbid state. Among other causes which are mentioned are over-fatigue, exhaustion by night-watching, sexual excesses, onanism, violent mental emotions, and insufficient food; but probably none of these are capable of directly causing it.

Prognosis.—There may be a question whether rachialgia ever undergoes an actual conversion, or development, into some one of those diseases in progress of time. Erb leaves this question an open one; but such a supposition seems to me to be improbable. The course of the affection is towards recovery, but relapses are common. Very often, too, the subsidence of pain in a particular spot in the back is followed by the appearance of neuralgia elsewhere, or of a different neurosis, either hysterical or of some other type.

The *diagnosis* of rachialgia from organic disease compressing the cord, and from hysterical and other spinal neuroses, is best considered afterwards.

Treatment.—A most important point to be determined is whether or not rest in the horizontal posture should be insisted on. Some of the patients whom the late Mr Hilton used to keep lying on their backs for months together, with sandbags to prevent any movements of the spine, were, I believe, sufferers from rachialgia rather than from actual spinal disease; and he undoubtedly obtained good results. Mr Teale, however, says that he succeeded in curing several persons belonging to the poorer classes while they were still pursuing their laborious avocations; and in many cases the general nervous state renders it exceedingly desirable that the patient should have fresh air and change of scene, and should even be encouraged to take moderate exercise, short of fatigue. An abundant supply of good food is of great importance. Dr Radcliffe says that a somewhat bold use of alcoholic drinks is a cardinal point in the treatment; but I strongly incline to the opinion of Dr Anstie that they should be taken in great moderation, and only with the meals. Quinine, the tincture of steel, strychnia, and cod-liver oil are each of them valuable medicines. The application of blisters to the spine is recommended by modern English authors. The older writers speak no less favourably of leeches and of cupping. These measures are now out of fashion; but in one very severe and obstinate case which arose out of the Thorpe railway accident, and which I saw with Mr Erichsen and with Mr Robinson, of Norwich, nothing gave so much relief as the repeated application of leeches at intervals of a few weeks, and especially at the catamenial periods. I must, however, add that the patient, a married lady, was stout and florid. Other applications that have sometimes proved useful

are turpentine liniment and unguentum veratriæ; or a bag of hot sand may be placed along the spine, or a galvanic current passed through it in the manner directed at p. 427, for neuralgic affections in general.

I have now to describe the second group of affections causing Paraplegia, to which I referred at p. 434, namely, those in which the primary lesion is outside the cord, and acts by subjecting it to *slow compression*, so as first to produce pain by interference with the posterior roots of the nerves, and finally to destroy the cord completely at the point affected.

Compression-paraplegia. — Forty or fifty years have passed since Ollivier and Cruveilhier described as a separate form of paraplegia that which is produced by the "slow compression of the cord." The importance of thus grouping together various affections of the parts which surround and support the spinal marrow seems not to have been recognised by the writers who followed them. But, recently, several observers have worked out the idea in detail; among others Charcot, who, however, seems to me to err in including in his description tumours and tubercles lying in the interior of the cord itself. For, as he himself admits, some of the most characteristic phenomena are absent in such cases. In the present section I propose to deal only with those lesions of the vertebræ or of the spinal membranes which exert pressure from without upon a more or less limited part of the cord, and so cause paralysis. I will first enumerate the diseases themselves and the conditions under which they occur, and then describe their symptoms and how they are to be distinguished from one another. This will be dealing with some of them in rather a fragmentary manner, for they are not always attended by paraplegia, and of cases in which this symptom is absent I shall take no heed. But the boundary lines, within which I shall keep, will be almost exactly those which divide the practice of the physician from that of the surgeon.

Causes.—In the first place it is to be remarked that some affections of the spine never cause paraplegia. Thus the cord seems always to escape compression in cases of lateral curvature (or *scoliosis*, as it is often called), however extreme may be the deformity. Leyden, indeed, alludes to one instance in which the patient was unable to stand or walk for more than a short space of time, but such an effect is quite exceptional. Whether the vertebral canal is ever narrowed in cases of osteo-arthritis I am not sure. In 1838 Aston Key related in the third volume of the 'Guy's Hospital Reports' two cases of paraplegia in each of which Wilkinson King—a pathologist of deserved reputation—found an intervertebral substance projecting backwards with raised lips upon the edges of the two adjacent vertebræ, and in one instance with an ossified posterior common ligament bridging over the space between them. It was taken for granted that this was the cause of the paralysis, but, so far as I am aware, no similar case has since been observed. I have already remarked that syphilitic nodes seem never to grow from the bodies or arches of the vertebra inwards, so as to interfere with the cord.

1. *Caries of the spine. Pott's disease.*—The most frequent, and therefore by far the most important, of all the affections that are really capable of giving rise to a "compression-paraplegia," is that which, from the kind of deformity produced by it, is in this country commonly known as "angular curvature," but which on the Continent is universally called after one of the greatest

English surgeons of the last century, Percival Pott. It consists in the destruction by caries, with or without necrosis, of the body or bodies of one or more vertebræ, which then fall together beneath the weight of the head and upper part of the trunk. The necessary result is a displacement of the corresponding arches and spinous processes, which become bent into a sharp angle or rounded curve, according to the number of the bones which are diseased. Opinions differ with regard to the mode of commencement of this morbid process. German pathologists agree in stating that it begins in the bodies themselves, the intervertebral discs escaping or being implicated only at a late period, and I have seen at least one case in which several of the vertebræ contained caseous masses in their interior, or even irregular cavities, which nowhere touched their surfaces. But, as Wilks long ago pointed out, there is in some cases a source of fallacy in the fact that when a disc is completely destroyed the adjacent vertebræ often lose each about half of its substance, and the remaining halves, coming together, look exactly like the fragments of a single bone ulcerated through by the disease. And I have repeatedly found intervertebral discs above and below the main seat of mischief presenting early changes of such a kind as to convince me that in the particular case under observation they, rather than the bones, were primarily affected. So, in an instance recorded by Sir William Gull ('Guy's Hosp. Rep.,' 1856, p. 179), death actually occurred from an affection of the cord at a time when the only change was softening of three of the discs, with the formation of a cheesy substance in the middle one and a little "absorption" limited to the adjacent part of the bone.

Another question is, whether caries of the vertebræ is to be regarded as "scrofulous." According to my view, this question is to be decided mainly by the presence or absence of associated changes in other parts. Cases of tubercular pyelitis and of Addison's disease are, each of them, now and then accompanied by an affection of the vertebræ immediately adjacent; and I could cite several instances in which a similar affection has occurred in conjunction with pulmonary phthisis, or tubercular disease of the testis, or dis ease of other bones or joints. Their frequency seems to me to prove that Pott's disease of the spine is often tubercular; but I must confess that in looking over a list of sixteen cases, all fatal by paraplegia, I find less evidence of this kind than I should have expected. It may be that a simple spinal caries will hereafter be recognised as distinct from the "scrofulous" affection. Dr Moxon is inclined to such an opinion; he would trace the former disease to accidental injuries, and thinks that it may be characterised by occurring in adults, and by its having a tendency to indurate the bones and to terminate in repair. But it seems to me, on the contrary, that in most instances of permanently cured angular curvature with anchylosis the active stage of the disease was passed through in childhood. Of the sixteen cases just referred to, in fifteen the patients were adults; four between twenty and thirty years of age, three between thirty-one and forty, seven between forty-one and fifty, and one at the age of fifty-six. The only case in which anchylosis existed was one in which the spinal disease began when the patient was six years old; he died at thirty-two.

Considering how great is the deformity in many of these cases, and how much it alters the relations of the arches of the vertebræ to one another one could not be surprised if the displaced bones often compressed the cord directly. That this does sometimes occur appears clear from a case of Brown-Séquard's, in which a paraplegia which had set in suddenly was

removed in twenty-five hours by extension of the spine. But every pathological museum contains specimens which show that the spinal canal generally remains of its full width, however much its direction may be altered. Moreover, as Charcot points out, paralysis often occurs in cases in which there is no curvature at all, while in other cases, in which curvature exists, the patient regains the use of his limbs, notwithstanding that the state of the bones remains unaltered. What really presses upon the cord when there is caries of the spine is, as Gull showed in 1856, a mass of cheesy *débris*, which collects between the affected vertebræ and the dura mater, having, perhaps, been extruded from the diseased fibro-cartilages or from the carious bones as the result of destruction of the posterior common ligament. Michaud has further observed that the outer layers of the dura mater become in their turn converted into a thick yellow caseating mass.

2. *Malignant disease of the spine.*—This is the only other frequent cause of extrinsic paraplegia. It assumes a variety of forms. Sometimes it is secondary to carcinoma of the breast, or to sarcoma occurring in the neck or in one of the long bones, or to cancer of the œsophagus or colon, the uterus or testicle. The nature of the disease is sufficiently obvious if there has been a primary affection in any of these parts, especially if a surgical operation should have already been performed; but it is otherwise when it is seated internally, as (for instance) in the mediastinum. Sometimes the vertebræ become affected with malignant disease by direct extension, as from a canccer of the kidney. Sometimes the spinal lesion appears to be itself a primary new growth. In these cases it is generally a sarcoma of one kind or another, and not infrequently it affects simultaneously a number of vertebræ in different regions, as well as other bones, such as those of the limbs, the ossa innominata, or the skull. Thus a careful search during life may lead to the discovery of a swelling in some distant part of the body, and so may clear up a case that would otherwise have remained altogether obscure.

Sometimes there is a distinct projection of one or more spinous processes, or a mass of the growth can be felt within the substance of the erector-spinæ muscle. Sometimes a tumour in connection with the front of the spinal column is discovered when deep pressure is made in the middle of the abdomen, or, if the cervical vertebræ are diseased, manipulation of the neck may reveal an enlargement on one side corresponding with the transverse processes. But in the great majority of cases one can feel nothing abnormal. The growth lies entirely within the spinal canal, except in so far as it occupies the substance of the bodies or laminæ of the vertebræ themselves.

Of seventeen cases of malignant disease of the spine, collected from the records of *post-mortem* examinations at Guy's Hospital, twelve were in males, five in females. The patients were of all ages, from sixteen to sixty-eight.

In one case in Philip Ward a correct diagnosis was possible, from the age of the patient excluding caries, while the severe pain and its increase on pressure of the spine downwards pointed to the vertebræ. It was confirmed before death by the detection of a tumour in the abdomen, which proved to be a cancerous gland.

In another case, occurring in a man aged 50, the paralysis was very slight, and the pain and other symptoms subsided so much after rest in bed

that the patient was allowed to get up. The same day he was attacked with renewed pain, and paraplegia became complete. Death speedily followed, and we found that while secondary cancer of the vertebræ had compressed the cord, the weight of his body in walking had crushed the bones together, and destroyed that segment.

3. *Erosion of the vertebræ by aneurysm.* — Common as it is for an aneurysm of the aorta to eat out the bodies of the vertebræ with which it comes into contact, it very rarely penetrates the spinal canal so as to interfere with the cord. We have in Guy's museum specimens from two cases, in each of which paraplegia developed itself before death at a period long after the discovery of a pulsating tumour in the back. But the most remarkable instance of this kind with which I am acquainted is one on which I made a *post-mortem* examination in 1871. A man, aged thirty, was admitted into hospital for paraplegia, which had begun three months previously. He had first complained of pain in the shoulders, then his left leg became weak and numb, and afterwards his right leg, and he experienced a pain as though the abdomen were constricted by a cord. He died without any suspicion of the real nature of his disease. When the erector spinæ were cut into, a large mass of laminated clot was found in its substance on the left side. This belonged to an aneurysm of the descending aortic arch, which had destroyed several ribs, and laid open the spinal canal for a space two inches in length. Some of the clot adhered to the outer surface of the dura mater; the cord itself was flattened, white, and very soft.

4. *Erosion of the vertebræ by a hydatid.*—This also is very rare. A striking instance of it is one recorded by Cruveilhier. A large echinococcus lay behind the vertebræ; it filled the groove on each side of the spine beneath the lumbar muscles so as to form two elongated pouches, which were connected together by a narrow neck that also penetrated into the spinal canal. A somewhat similar case occurred to Dr Moxon in our *post-mortem* room in 1871, a few months before I met with the aneurysm just referred to. The parasite was "multilocular," budding externally. It formed a large elastic swelling on the left side of the spine, and made its way into the canal through the second and third lumbar vertebræ. It compressed the cord, but did not penetrate the dura mater. The patient, a woman of fifty-eight, had suffered for eleven months from pain in the side, and had been paraplegic for six weeks.

5. *Meningeal tumour.*—New growths of various kinds occasionally form in connection with the spinal membranes. Sometimes a lipoma or an enchondroma is formed outside the dura mater of the cord, having developed itself in the connective tissue which is usually present there. More frequently, one of the spaces within the sheath is the seat of the affection. According to Charcot, tumours are particularly apt to grow from the inner face of the dura mater—generally sarcoma or psammoma.

I have met with only one specimen of a growth in this position; it consisted of perfectly developed fibrous tissue; its upper end lay close to the foramen magnum; it extended downwards for more than two inches, and pressed upon the back of the cord on the left side. In another case I found a soft, granular, reddish-grey mass, smooth and lobulated on the surface, which lay loosely attached beneath the arachnoid membrane, between it and the pia mater, in the dorsal region. It measured an inch and a quarter in length; it was made up partly of fibrous and partly of spindle-cell tissue.

6. *Meningeal gumma.*—This affection also seems to be very rare, even more so than tumour. Virchow has related a case in which the dura mater in the lower cervical region was increased to three times its normal thickness, and was bound down to the bodies of the fifth and sixth cervical vertebræ by a large quantity of firm connective tissue, which doubtless had been developed out of a syphilitic new growth. Wilks once found a hard, irregular, yellow mass three quarters of an inch long, lying on the right side of the cord within its sheath, and adherent to the pia mater and to the posterior roots of the spinal nerves, which last were compressed by it. In a case of Dr Moxon's ('Guy's Hosp. Rep.,' 1871) there were a number of brownish or blackish patches, from the size of a barleycorn to that of a pea, with soft yellowish centres, penetrating into the substance of the cord from the pia mater. In one described by Heubner, in 'Ziemssen's Cyclopædia,' a gelatinous mass extended from the floor of the fourth ventricle into the bulb for a depth of one twelfth of an inch; while in the cervical region the dura mater and the other membranes behind the cord were pressed together into a callous mass, which was firmly adherent both to the arch of the atlas and to the posterior columns of the cord itself.

Symptoms of compression-paraplegia.—At the bedside we recognise and distinguish this secondary paralysis from primary affections of the spinal cord, not so much by peculiarities in its proper symptoms as by the fact that they are accompanied, and in most cases preceded, by symptoms due to interference with the roots of the nerves coming off from the cord at or just above the level of the lesion. By Charcot these are termed "extrinsic" symptoms, while he gives the name of "intrinsic" symptoms to the loss of movement in parts lower down, to the anæsthesia, and to the other effects of pressure upon the cord itself.* Erb describes the former as belonging to a "first stage," the latter as characterising a "second stage" of the disease.

In attributing the earlier phenomena to the circumstance that some of the spinal nerves have their roots directly involved in the lesion, one is not expressing a merely speculative opinion. They have been observed on dissection to be reddened and greatly swollen, although not showing any very serious histological change, while in advanced cases at a late period they have been found transparent, greyish, and atrophied, with their fibres in a state of fatty degeneration.

Chief among the early symptoms is *pain.* And few points in clinical medicine are more important than the fact that this is referred by the patient, not to the seat of mischief, but chiefly or solely to those parts to which the affected nerves are distributed. Thus, there is always danger of mistaking cases of spinal disease for various other affections; now for occipital or brachial neuralgia, then for pleurodynia, and again for gastrodynia, for colic, or for sciatica. Very often the pain appears to be fixed in a single spot, or to occupy only a very small part of the whole surface supplied by a single nerve. In other cases it shoots through all the main trunks belonging to a limb. Charcot lays stress on its burning character, and on the absence of *points douloureux* as distinguishing it from a mere neuralgia; but as I believe with the late Dr Anstie that the *points* are to be detected only in nerves which have already been the seat of pain for a considerable length of time, I doubt whether the distinction can be maintained.

* Extrinsic and intrinsic are also applied to denote lesions of the cord itself, and those which affect it from outside, respectively—intramedullary and extramedullary, as they are called by some writers, with a reference to the medulla spinalis.

Associated with the pain there is often an extreme degree of hyperæsthesia. The patient may be unable to bear even the slightest touch without crying out, and every movement may be excessively painful. An eruption of herpes zoster has now and then been observed in the course of some of the nerves which are most seriously involved in the disease. On the other hand, there is sometimes impairment of sensibility or actual anæsthesia over a more or less extensive part of the surface to which the affected nerves are supplied. And the muscles which receive branches from them may show tonic or clonic spasms, or become paralysed and even atrophied, with loss of faradic contractility and an inability to respond to reflex stimuli.

It is to be observed that in speaking of these phenomena as symptoms of compression-paraplegia, we are adopting an expression which may prove to be incorrect. Precisely similar pains may occur in any case in which spinal nerves or their roots are involved in disease, though the cord may to the last remain intact. Thus lateral curvature of the spine, which probably never causes paralysis, is frequently accompanied by a fixed pain in one or more of the ribs or intercostal spaces, which seems to be due to pressure upon nerves as they are passing through the intervertebral foramina. And in the most remarkable case of medullary sarcoma of the vertebræ which I have ever seen there was at no time any well-marked paralysis. The patient was an errand-boy, aged sixteen, who, five months before his death, began to experience pain first in the loins, then in the shoulders, and then in all his limbs. His sufferings became intense and hardly bearable. The whole surface of the body was excessively tender, but especially the lower part of the back. The pain was constant, but it varied in position. There was numbness and a sensation of tingling in each hand; this was first noticed in the fingers supplied by the ulnar nerve on either side. He became wasted to the most extreme degree. The only way in which he got any ease was by lying flat on his back, with his legs drawn up. Every movement of his neck, and of any part below it, caused him severe pain. One day he became insensible and had a series of epileptiform seizures, in which he died. A mass of white medullary new growth (possessing microscopical characters such as are now known to belong to the sarcomata) occupied the lumbar glands, and spread from them to the intervertebral discs, eating also into the lumbar vertebræ themselves. It passed up in front of the spine into the neck, where it involved part of the brachial plexus on each side. It nowhere penetrated into the spinal canal or implicated the membranes of the cord.

There seems to be no doubt that pain is more marked as an early symptom in cases of malignant disease of the spine than in those of other forms of compression-paraplegia. Perhaps one ought to be contented to accept this as an example of the rule that cancerous affections in general are apt to be very painful. There are many nervous filaments ramifying in the periosteal and other tissues connected with the vertebræ, and one can easily understand their leaving the patient no peace when involved in a new growth.

Cruveilhier long ago associated together under the name of *paraplégie douloureuse* certain cases attended with intense lancinating pains in the nerves of the sciatic and lumbar plexuses, of which the muscular branches are paralysed. Recent writers have remarked that a large majority of the cases in question are instances of malignant disease of the spine, involving the roots of the nerves for the lower limbs in the cauda equina, or where they lie by the side of the lower end of the lumbar enlargement of the spinal

marrow. But I do not think that the reason for this has been pointed out, namely, that whereas the massive lumbar and lowest dorsal vertebræ frequently become cancerous, they are comparatively seldom affected with caries. In fourteen consecutive cases of paraplegia, due to Pott's disease of the vertebræ, which I have collected from our *post-mortem* records at Guy's Hospital, the affection was not once in this lower part of the spine. I found a few other cases in which it occupied this position, but without paralysis. Thus, in one instance the third and fourth lumbar vertebræ were found "extensively diseased," and in another the eleventh and twelfth dorsal vertebræ had entirely disappeared, so that on straightening the spine a large space was seen in front, in which the sheath of the cord was exposed. But in each case the disease had been entirely latent during life, the patient had been kept in bed for a considerable time by some other malady, and this no doubt had enabled the spinal cord to escape being compressed. In the report of the second case it is expressly noted that repeated questions failed to elicit any complaint of pain, whether in the back or elsewhere.

Hitherto I have purposely deferred mentioning pain in the back itself, —rachialgia, already described as a neurosis,—because, although it is not infrequently present as an early symptom of compression-paraplegia, it is far less constant than one might have expected. It may assume different forms. Certain of the spinous processes may be tender on pressure, or oversensitive to a slight blow or jar. Or the patient may be conscious of a feeling of stiffness in moving the back or the neck, according to the seat of the mischief. Sometimes—as in a case of cancer of the cervical vertebræ placed on record by Mr Cæsar Hawkins—there is a great increase of pain in the neck when the face is turned over on the pillow to one side or the other, so that the hands are used to steady the head in every change of posture.

After the "extrinsic" symptoms have lasted for months or even for years,—having been perhaps regarded as neuralgic or rheumatic,—they are succeeded by others of which the spinal origin is obvious to the most uninstructed observer. These "intrinsic" symptoms are by no means merely results of mechanical pressure upon the cord. Very gradual external pressure by itself may probably alter the shape of the cord, but symptoms only begin when the pressure interferes with its circulation and nutrition. As far back as 1856, Gull discovered in a case of this kind granule-masses in the tissues of the cord. Within the last few years the state of the spinal cord in compression-paraplegia has been thoroughly investigated by Michaud and other French observers, and they found that the affected part becomes reduced in size (so that sometimes it is scarcely as thick as a quill) and flattened or distorted in shape. Its tissue may be pale, but otherwise natural to the naked eye; or it may obviously have lost its normal structure. It is either softened or (in a more advanced stage) harder than usual. Under the microscope the neuroglia is seen to be thicker and more fibrous; the nerve-tubes have no longer any medullary sheaths, but their axis-cylinders persist and may even be increased in size. Granule-masses are abundant. The ganglion-cells are swollen, vacuolated, and pigmented, or sometimes they are degenerated and broken down. In other words, there is a chronic transverse myelitis, and it extends a little way above and below the spot which is actually compressed.

The course of the paraplegia is such as might be inferred from the nature of the lesion causing it, according to the principles laid down in the

last section. Sometimes its development takes place very rapidly, within two or three days, or even in a few hours—much more often it occupies several weeks. The patient finds his legs more and more heavy, especially in going up and downstairs; he becomes unable to stand; at last he cannot even move his toes while lying in bed. Subjective sensations of numbness, pins-and-needles, &c., often precede the motor symptoms, but, later on, loss of voluntary power over the muscles generally preponderates over loss of sensation. The upper limit of the spinal lesion is commonly well marked, especially if there is much anæsthesia. It corresponds more or less exactly with the seat of the early "extrinsic" pains and tenderness, which, indeed, often persist after paralysis has set in. A point of great importance is that, as a rule, there is no tendency for secondary changes in the cord to spread upwards in the main tracts beyond the point of compression; but Erb quotes Michaud as having observed that in some very rare cases a morbid action may ascend along the lateral columns, so that, for example, the upper limbs may after a time become paralysed, although the part of the cord which is compressed is in the thoracic region. Sometimes the local seat of changes by compression gives rise to ascending sclerosis of the posterior median columns without special symptoms; more frequently to *descending* sclerosis of the lateral columns. The lower limit of the lesion cannot be defined with exactness, but at least one can generally say that the lumbar enlargement of the cord is healthy, the only exceptions being those cases in which it is itself the seat of the pressure. Reflex contractions in the lower limbs are usually very readily excited, much more so than under normal conditions. Leyden, indeed, remarks that in old people the sensibility of the spinal centres to stimuli from below is comparatively little marked, so that compression-paraplegia is generally more easily recognised in children than in persons advanced in years.

The state of the bladder is very variable; it often continues to expel its contents quite naturally for some time after the legs have begun to be paralysed, but whenever the paraplegia is complete it almost always becomes paralysed.

Charcot lays stress on some modifications of sensibility which, he says, although not peculiar to compression-paraplegia, are observed more frequently in these cases than in other affections of the cord. One is a retardation in the transmission of sensory impressions when there is not absolute anæsthesia; fifteen, twenty, or even thirty seconds may elapse between the application of a stimulus to the cutaneous surface and the perception of it by the patient. I shall have to discuss this symptom more fully hereafter, when I am describing "locomotor ataxy." Another is a peculiar dysæsthesia, a very painful feeling, which is excited by slightly pinching a limb or touching it with anything cold; this lasts for several minutes, and apparently no more exact description of it can be given than that it is a sort of diffused vibration bearing no relation to the nature of the impression which calls it forth. Another is an "associated sensation" which is referred symmetrically to a spot upon the opposite limb corresponding exactly with that to which the stimulus itself is applied.

Diagnosis.—The determination of compression as the cause of paraplegia may be either very easy or very difficult. The simplest cases of all are those in which there is spinal deformity, and in determining the fact that the cord is pressed on from without one can often at once define the nature of the lesion, for the short round bend, or the sharp angle, produced by Pott's disease is quite unlike anything else. But often the natural configuration is preserved.

Leyden says that if caries occurs in either the lumbar or the cervical region there is rarely any projection of the spinous processes, the reason being that the natural curve of those parts of the column is in the opposite direction; but, when a person stoops, both the cervical and the lumbar vertebræ are normally convex backwards, and I think it is not at all uncommon for some one of the spines to appear unduly prominent when disease is present. At any rate I remember several instances in which I was in doubt as to whether the projection was or was not excessive. In such cases it is important to observe whether the vertebral column retains its flexibility. In malignant disease, if there is anything to be detected on manipulation, it is the presence of a new growth and not a mere displacement of the bones. Cruveilhier, in his hydatid case (to which I have referred at p. 481), discovered at the bend of the twelfth dorsal and first lumbar vertebræ a spot of the size of a sixpence, which felt like a depression surrounded by a bony ring, and which he took for the remains of a spina bifida. As he says with regret, if he had rightly interpreted what he observed, and had ventured upon making a puncture, the patient might perhaps have been cured.

On the other hand, meningeal tumours and gummata are of course altogether beyond the reach of the fingers. A careful analysis of the symptoms affords the only chance of distinguishing cases in which such affections are present from those of disease of the vertebræ unattended with deformity. I have already noticed the peculiar severity of the pain caused by malignant growths affecting the spinal column. But just in proportion as this enables a diagnosis to be made between that particular form of compression-paraplegia and the rest, do difficulties arise in distinguishing cases of caries of the vertebræ or of meningeal tumour from those of primary *transverse myelitis*, or of any other affection of the interior of the cord limited to a particular segment of it. In describing myelitis I have mentioned that it is often accompanied by painful sensations referred to various parts of the body, and by the peculiar "girdle-feeling,"—phenomena which are strictly comparable with the "extrinsic" symptoms of compression-paraplegia, and which do not necessarily differ from them even in degree. I am disposed frankly to admit that in some cases a diagnosis between the two forms of disease is impossible.

It is chiefly at an early period that a case of compression-paraplegia might be mistaken for one of *rachialgia* or *vice versâ*. The suggestion has, indeed, been made that, considering the frequency with which this spinal neurosis is associated with hysteria, one ought to be prepared for its being now and then accompanied by hysterical paraplegia, so as very closely to simulate a grave disease of the vertebræ. But I do not know that a case in point has ever been recorded, and it is probable that a careful examination of the patient would show a want of correspondence between the apparent upper limit of the lesion in the cord, and the seat of tenderness and pain in the spinal column. When unattended with paralysis, cases of rachialgia may generally be distinguished from those of incipient disease of the bones without any great difficulty.

Prognosis.—Most forms of compression-paraplegia terminate inevitably in the death of the patients, but their duration is very variable. According to Leyden, malignant disease of the vertebræ commonly runs a course of some months, or even of more than a year;* it kills, he says, by a slow cachexia and wasting with dropsy, or directly by interfering with the cord. In some cases,

* Among fifteen recent cases in Guy's Hospital I find that the duration, from the

when its seat is in the upper cervical vertebræ, the fatal issue is preceded by delirium and stupor, for which no explanation can be found in the autopsy. Meningeal tumours are described by Erb as slower in their effects; some cases end in eight or ten months, others in from two to five years, others last longer still, even to a period of fifteen years. It accords well with his statement that in one of the two instances in which I found the cord interfered with by a growth on its exterior the duration was sixteen months, and in the other two years, if we reckon from the time when pain in the back was first complained of; and both patients may be said to have died accidentally, the one of pneumonia, the other of suppurative inflammation of the kidneys. But in a man, aged sixty-two, who had a malignant growth extending from the vertebræ into the spinal canal, death took place within a few days of the development of paraplegia, and not more than two or three months after the first complaint of pain in the front and sides of the chest. He was so florid and healthy looking that until the paralytic symptoms showed themselves I thought the case was one of hypochondriasis.

Treatment.—All that can be done in cases like the last is to relieve the pain and to prevent the development of cystitis and the formation of bed-sores. The application of a belladonna or opium plaster to the back or an occasional blister may meet the former indication, but in most cases morphia has to be given internally or subcutaneously.

It is probable that mercury and iodide of potassium are capable of curing a paraplegia which is due to syphilitic gummata pressing on, or growing into, the cord; and I have already remarked that this treatment should never be omitted in any case which can possibly be of venereal origin.

The only other variety of compression-paraplegia in which recovery of power over the limbs appears to be possible, is that due to Pott's disease of the spine. Of such cases a great many do well; among six which came under Erb's observation during the year before he wrote his article on this subject, only one ended fatally, the remaining five being cured or greatly relieved. Charcot refers to two patients, who had their lower limbs completely paralysed for eighteen months and two years respectively, and who nevertheless recovered perfectly. He speaks in decided terms of the value of the application of the actual cautery along both sides of the projecting spinous processes. But it seems likely that all other plans of treatment will in future be superseded by Sayre's method of applying a plaster-of-Paris bandage, rolled round the body, while the patient is suspended from the head, chin, and armpits by a suitable apparatus. Cod-liver oil and other tonics should, however, also be administered.

One might well have doubted whether in these cases of caries of the vertebræ, in which recovery takes place from secondary compression-paraplegia, inflammation of the substance of the cord has ever occurred. But an observation of Michaud, which is cited by Charcot, shows that this really is so. A woman who had regained the use of her legs for more than two years, died of hip-joint disease. The cord, at the level of the spinal affection, was found reduced to the thickness of a goose-quill, its sectional area being not more than one third of that of a healthy cord in the same region. It was of firm consistence, and grey in colour; in other words it seemed to be affected with sclerosis in an advanced stage. The microscope, however, showed that a considerable number of nerve-fibres,

earliest symptoms till death, was three months in six, under a year in eight, and fifteen months in one patient.

possessing medullary sheaths, were embedded in the thick dense fibrous material which gave it these appearances. Only one of the grey cornua remained, and this displayed but a small number of uninjured nerve-cells. I have already cited this case as proving the possibility of recovery from myelitis, and from that point of view its importance can scarcely be exaggerated.

Spastic Paraplegia.*—As I have already more than once had occasion to point out, rigidity of the paralysed limbs is no uncommon feature in cases of segmental disease of the cord, whether primarily affecting its substance or secondary to compression of its surface. It is only when the lowest lumbar centres are destroyed that the legs are of necessity flaccid. Thus in paraplegia from angular curvature they are often spasmodically flexed whether in a child or in an adult; sometimes, on the other hand, they are as forcibly extended. These conditions are closely related to exaggerated knee-jerk and to ankle-clonus; and I think it is reasonable to refer them to an exalted susceptibility of the spinal centres for the lower limbs. We shall find that similar contraction, usually of a "tonic" kind, is frequently a late result of lesions of the brain in cases of hemiplegia; and less frequently the paralysed limbs become rigid soon after the paralytic stroke. In the cases we are now concerned with, the tonic contractions ("contractures") and rigidity are late, not early. They come on slowly after paraplegia, and they are associated with increased reflex actions both superficial and deep, and with the clonic contractions known as ankle-clonus. The pathology of these cases is still far from certain.

Sir William Gull has recorded one case of chronic spinal meningitis in which there was rigid flexion of the legs; but I do not find any evidence that this symptom is particularly common when the membranes are diseased.

In the forms of disease of which I have been speaking, the contraction of the limbs is obviously secondary to paralysis of them, for it generally does not show itself until the patient has already lost power over his legs for a considerable time. These cases of *secondary* rigidity and spasm of muscles which have already lost more often voluntary power are probably of the same pathology, whether paraplegic, hemiplegic, or monoplegic in distribution. They are believed to depend upon descending sclerosis affecting the motor tract, either from a focus in the encephalon, as a clot of blood, or from one in the cord, as a segmental lesion from compression.

There are, however, other cases in which rigidity is present from the first, and, indeed, constitutes the most marked symptom, so that it may seem to be the only thing that interferes with the patient's standing or walking. Thus a "tabes dorsalis spasmodica" is described by Charcot as an independent spinal affection, and Erb gives to the same complaint the name of "spastic spinal paralysis." These writers agree in thinking that its proper lesion is in all probability a symmetrical *primary* sclerosis of the lateral columns, the crossed pyramidal tract—or, perhaps, of the hinder parts of them,—leaving the anterior grey cornua intact. The most satisfactory evidence on this point is afforded by a case of Dr Morgan's, of Manchester, in which Dr Dreschfeld found sclerosis of the crossed pyramidal tracts and no other lesion, spinal or cerebral. It was published in the 'Transactions of the International Medical Congress of 1881;' and also in the 'British Medical Journal,' of the same year, vol. i, p. 407. Spastic paraplegia would

* *Synonyms.*—Tabes dorsalis spasmodica (Charcot)—Spastic spinal paralysis (Erb)—Spasmodic paraplegia (primary and secondary)—Primary and secondary lateral sclerosis.

therefore be a systemic, or "columnar" disease anatomically, the sclerotic process keeping to a definite tract of fibres.

Symptoms.—The lower limbs generally assume a position of rigid extension and adduction. The contraction may vary indefinitely in degree. At first it is only occasionally present, and is generally less marked when the patient is lying down than when he stands upright. One of the earliest symptoms of the complaint is a peculiar gait. In walking the foot is lifted with difficulty; the toes are scraped along the floor and catch against every inequality in the surface; the step is short and hesitating; sometimes there is a peculiar hopping movement, the body being raised upon the toes at each step. Very often the attempt to walk causes a tremor in the foot and leg, which may extend to the trunk also,—a true clonus. The back may be arched, and the head thrown backwards, or the body may be bent forwards over the toes by similar tonic contraction of the muscles of the trunk, so that there is danger of falling, especially in descending stairs. The gait is rolling or waddling: its peculiar character is carefully described by Dr Ross. The legs feel weak and heavy and are easily tired. Sometimes, if the patient sits upon a sofa, his lower limbs stick out before him, the feet not touching the ground. In more extreme cases he is confined to bed, and is perfectly helpless. His knees are tightly pressed together by spasm of the adductors, and cannot be bent from spasm of the quadriceps. Yet there is generally no impairment of sensibility, and pain is entirely absent. The bladder and rectum perform their functions naturally. Erb says that the galvanic and faradic contractility of the muscles is slightly lowered.

There is greatly exalted susceptibility to "tendon-reflexes." Not only are the common movements induced by percussion of the ligamentum patellæ exaggerated, but movements can be excited through such tendons as those of the tibialis anticus and posticus, the biceps femoris, and even through various aponeurotic structures. Ankle-clonus is present. There is not necessarily any corresponding increase of the superficial reflex movements which are excited by cutaneous impressions.

The complaint is said to occur chiefly in persons between thirty and fifty years of age, and in men more often than in women. Of Dr Morgan's seven cases only one occurred in a woman. Erb speaks of it as "very frequent." In this country primary spastic paraplegia is certainly rare, while the secondary form of the affection is common. Its causes are unknown. Syphilis and lead have been suspected and also poisoning by *Lathyrus cicera* in three cases reported from Italy. In one of Dr Morgan's patients the disease was apparently hereditary.

Its *course* is progressive, but very slow, occupying a period of eight, ten, or fifteen years; sometimes it remains stationary at the same point for a length of time. It may never extend above the hips, or it may at length affect the lumbar and the abdominal muscles or the upper limbs. The abdomen is then prominent and hard, and it is separated from the lower part of the chest by a furrow more or less deep. The fingers are from time to time, or even permanently, clenched within the palm; there is extension of the wrist and elbow-joints, with pronation of the forearm; the arms may be fixed rigidly by the side of the trunk. One lower limb is often affected before the other, and the contraction may then spread to the arm of the same side before it involves the opposite leg. Sometimes the upper limbs are the first to be attacked. Erb

says that it rarely happens—and only at the latest periods of the disease—that the lower limbs are rigidly flexed, instead of being extended.

According to Charcot and Erb the only real difficulty in *diagnosis* is from disseminated sclerosis in that form in which the symptoms of affection of the higher centres happen to be wanting. Charcot adds that in one of the cases with which he used to illustrate his lecture on "spasmodic tabes" scattered patches of sclerosis were found at the autopsy as high as the crura cerebri.

The *prognosis* is said to be favourable, in so far as the complaint has no tendency to destroy life, except through the supervention of phthisis or some other intercurrent disease.

As to *treatment*, Erb has seen two cases almost cured, and several much relieved by galvanic currents applied to the spine. He also speaks favourably of the "cold-water cure," and of the gaseous saline baths ("Sool baths") of Rehme and Nauheim. He has found the nitrate of silver sometimes useful as an internal medicine, and Charcot recommends the bromides. Strychnia is likely to do harm rather than good. According to Dr Ross, "the galvanic current is by far the most trustworthy remedy." According to Dr Gowers, faradism is harmful, and galvanism has no influence in lessening the spasm or improving the strength. As he well puts it: in primary lateral sclerosis the chance of recovery and the danger to life are both small.

Spasmodic paralysis in children.—Dr Gee has recorded in the thirteenth and sixteenth volumes of the 'St Bartholomew's Hospital Reports' a series of cases which he has called "spastic paraplegia." They all occurred in children, and the complaint was either congenital or began in early infancy. There were eight cases, six girls and two boys. The legs were always affected and sometimes the arms. The contractions corresponded in character very closely with Erb's and Charcot's descriptions. Dr Gee lays stress on the fact that handling the limbs increased the rigidity. Chloroform relaxed it in all cases but one; in that instance the muscles of one calf were a little wasted, the disease having lasted eleven years. To one patient the juice of conium was given, to another the extract of belladonna, but without any good results.

Seeligmüller, Erb, and Gowers describe spastic paraplegia in children. In some of these cases it was of the variety described by Charcot as "sclérose latérale amyotrophique," in which wasting of the affected muscles is present, and the anterior cornua as well as the lateral columns are affected. In other cases the symptoms were referred to a cerebral rather than a spinal lesion.

Cases at Guy's Hospital.—On looking through the reports of cases that have been under observation at Guy's Hospital during the last few years scarcely any answer well to the accounts given by these writers of primary spastic paraplegia. There have been eight or nine instances in which contraction of the lower limbs has been apparently the primary disease; but the knees and thighs have, without exception, been drawn up in a state of flexure instead of being stretched out. Any attempt to straighten the limbs has generally been attended with great suffering; but in one case —that of a man aged thirty-three, under Dr Wilks's care—the suspension of a weight of fourteen pounds to the right foot led to a marked diminution of the pain; the limb was bent, so that the heel almost touched the buttock, and it could not be put straight, even when chloroform had been inhaled.

But the point to which I am most anxious to draw attention is that the

extract of *Calabar bean* has in three or four instances appeared to remove the rigidity altogether.

The first case in which I observed this satisfactory result was that of a boy, aged six, who was admitted on August 19th, 1874. He had been well until three weeks previously; he was then found crying with pain, and it was thought that he must have had an injury, of which, however, there was no other evidence. He lay on his right side, with his legs drawn up, and unable to move them. They could be forcibly extended, but then became rigid. The expression of his face was indicative of pain, and he had complained of pain in the back of the head and over the spine. Sensation was unimpaired, but the fæces and urine were often passed into the bed. He could close his hands, but with a very feeble grasp, and sometimes they were noticed to be stiff. The diagnosis was chronic meningitis, and hydrarg. c. creta, iodide of potassium, bromide of potassium, and other medicines were given without any result up to the 8th of October. At that time he began to take the extract of physostigma in doses of one sixth of a grain, gradually increased to half a grain, three times daily. Improvement quickly set in; the limbs became less rigid, and he regained the power of moving them. On November 21st cod-liver oil and steel wine were substituted for the Calabar bean. On January 9th, 1875, he left the hospital, being then able to walk pretty well without help.

In 1876 I was asked to see a boy, aged fourteen, who was under Mr Cooper Forster's care, with contraction of the thighs and legs, which had been coming on for six months. He appeared to suffer intensely from pains in the affected limbs, and screamed when they were touched or when he made any movement, so that he allowed no rest to the other patients. The legs and thighs were much wasted. Remembering the former case, I prescribed the Calabar bean in doses at first moderate, but quickly increased. The symptoms at once began to subside. He was after a time transferred to my charge, and left the hospital cured.

On October 31st, 1876, a woman, aged forty-one, was admitted under my care with a spinal affection which had come on suddenly two months before. She first complained of pains in the leg-muscles and then of stiffness in the joints. This was followed by great weakness and wasting of the lower limbs; and then they slowly began to contract. She lay in bed with her legs strongly flexed; when they were forcibly straightened it gave her great pain. Faradic contractility was found to be much diminished, and she had partial loss of sensation; she could not pass her water. She took at first half a grain of extract of physostigma three times daily; after a week this dose was doubled, and four days later it was trebled. By this time she could already extend her legs to some extent of her own accord. The medicine was continued for a month and then some tincture of iron was prescribed. By December 23rd she could straighten her lower limbs perfectly well. In the month of February, 1877, she became able to stand and to walk without help, and on March 1st she was discharged cured.

In 1878 a sailor, aged twenty-eight, came into the Clinical Ward under my care on May 29th. Fourteen months previously he had been in a river in South America loading timber, and had repeatedly got wet through and allowed his clothes to dry on him; he said he was well used to getting wet with salt water but not with fresh water. Four or five days after he left off this work, he one morning on waking found that his left leg was stiff and painful in the ham. As the rigidity gradually got worse, he went into

hospital at New Orleans, and after a time it passed off. He returned to work, but two months later, the weather having been bad, he again began to suffer from stiffness and pain in the left leg. Presently the right one also was attacked. They both became weak, and for six weeks before admission he could not walk without a stick. At times he had short attacks of convulsive twitchings in the left leg. I found the muscles of the thigh decidedly rigid on both sides, especially the left. After leaving him for a few days without treatment, I prescribed the extract of Calabar bean in doses of a quarter of a grain, afterwards increased to one grain. Improvement was not at first very striking, but at the end of thirty-four days there was hardly any rigidity left.

The only case that I know of in which the extract of physostigma produced any unpleasant effects is one recorded in vol. xviii of the 'Guy's Hosp. Rep.' A boy, aged ten, had been suffering for some months with paraplegia before rigidity of the legs set in. A quarter of a grain of the extract was ordered, and after a week it was increased to half a grain three times daily. By mistake he took a grain at one dose, and an hour afterwards he was blue in the face but perfectly conscious, perspiring profusely, a clear froth coming from the mouth, the pupils of natural size, the hands cold, numb, and almost powerless. He had an emetic, and in three hours he was as well as before.

The last case seems to me to have been one of myelitis, apart from the tonic spasm, which was a mere complication; but the other cases just related seem to me to prove that there is a spinal affection, of which the principal symptom is a rigidly contracted state of the legs, but which yet fails to correspond with the rather narrow definitions of Charcot and of Erb. And I would propose that for the present at any rate they should all be placed in the same category under the name of *spastic paraplegia*. Whether a sclerosis of the lateral column occurs in these cases may be left doubtful. The fact that recovery takes place under medical treatment is perhaps evidence to the contrary, although not amounting to proof. What weighs more with me is the circumstance that there is a wide class of neuroses, of which I shall hereafter have to speak—all of which are attended with spasms of one kind or another—while none of them are as yet traceable to anatomical lesions. So long as they are regarded as due to functional disturbance of the nervous centres, I should be inclined to associate spastic paraplegia with them so far as its pathology is concerned.

Later cases.—A well-marked case of the more common secondary form, has been for more than eighteen months under the care of the editor in Guy's Hospital. The patient, a boy of eleven, when first admitted suffered from caries of the cervical and of the lower dorsal vertebræ which has since produced both lumbar and psoas abscesses. These have now long healed and there is only slight paresis of the arms and legs without anæsthesia or serious disturbance of the bladder or rectum, or bedsores or pain. The muscles have not atrophied except from want of use. When first admitted there was tonic contraction of both arms and both legs, the former flexed, with *main en griffe*, the latter extended in the cadaveric position. Knee-jerks and plantar reflexes were exaggerated and ankle-clonus was readily elicited. Faradic contractility was unaffected. Physostigma was pushed without benefit. But after many months, as the result apparently of improvement of the vertebral disease by surgical treatment, the power of his arms has become almost normal and contraction has nearly disappeared; he

can move his legs well, although clonus and exaggerated reflexes remain, and his general condition is greatly improved.

Of *primary* spastic paraplegia I have only seen two cases. Both patients happened to be clergymen. The one, a remarkably healthy well-developed man of about forty, complained of gradually increasing difficulty in walking owing to "stiffness" of the legs. He could not bend his knees and found it almost impossible to ascend a broad and shallow flight of stairs. When lying down, his legs were rigidly extended. Ankle-clonus was present and the knee-jerks were increased. There was no pain except during occasional cramps or spasms in the legs. The pelvic organs were unaffected. There was no true paraplegia, for the lower limbs could be moved readily until the check of stiffness came, and the skin was normally sensitive. The muscles were of good size. I prescribed him physostigma and he thought there was some improvement when I saw him again after several months, but if any it was slight; and afterwards I heard that he had put himself under a "rubber."

The other case occurred in an older man—one who had been an Alpine climber and enjoyed robust health. The loss of power also in his case only accompanied stiffness of the legs. The knees were bent, the body was bowed as if with the "chronic rheumatism" of an aged countryman, and it was almost impossible for him to walk or to stand upright. The pelvic organs were unaffected, knee-jerks were readily produced and clonus also. As in the previous patient, the upper limbs were free. In this case also physostigma was fully tried with only doubtful benefit, but under repeated courses of nitrate of silver very decided improvement has taken place, whether in consequence or not I do not pretend to say.

It appears that the four cases on p. 491 cited in the first edition of this book belong to a different clinical type from those described by Charcot, and have possibly a different pathology. Contraction of the legs in a *flexed* condition apart from primary disease of the joints may supervene in the last stage of chronic diffused myelitis, of compression-paraplegia, of infantile atrophic paralysis, and even of primary lateral sclerosis itself. It may also occur in a case of hysterical paraplegia. But the "cadaveric" position with extended hips, knees, and ankles and adducted feet is the rule in cases of presumed secondary lateral sclerosis. Moreover, in the latter cases there is no pain, and the bladder is unaffected. It is in them, whether secondary to cerebral hæmorrhage or to curvature of the spine, that we have good evidence of sclerosis of the lateral columns; and when primary, like the two cases just recorded, it is probable that the lesion is the same as in Dr Morgan's patient.

Diffused Affections of the Spinal Membranes.—This appears to be the most convenient place for a brief mention of certain diseases which ought perhaps in strictness to have been enumerated among the conditions which may give rise to "compression-paraplegia," but which differ from the rest in not being limited to any one part of the cord. They are not segmental, but indiscriminate or diffuse in localisation.

Spinal meningeal hæmorrhage.—Except as the result of severe injuries, which come under the care of the surgeon, this is a very rare lesion. The causes which I find assigned to it by authors are strong bodily efforts, violent emotions, and the suppression of a hæmorrhoidal or a menstrual flux. If a diagnosis is to be made during life, it must be based upon the sudden development of the symptoms, and upon the admixture of the effects of irritation of the cord and spinal nerves with those of compression of these

structures. Certain cases, in which there have been severe pains in the back and limbs, rigidity of the spine, and partial paraplegia have been attributed to the effusion of blood into the spinal membranes; and this explanation has not been regarded as invalidated by the circumstance that recovery has slowly taken place. But it would seem that in other cases in which meningeal hæmorrhage has proved fatal, so that it has been demonstrated in the deadhouse, the symptoms have not been equally characteristic.

Acute spinal meningitis.—I have already had occasion to mention the fact that secondary inflammation of the spinal membranes is not a very uncommon consequence of the formation of a deep bedsore over the sacrum. In some cases the vertebral canal is actually opened by sloughing of the fibrous membrane which closes its lower end; in others, perhaps, the morbid process extends inwards along the foramina for the posterior sacral nerves. At least four instances of this kind have been observed in the *post-mortem* room of Guy's Hospital; and probably several have been overlooked. I do not find that any distinctive symptoms have been noted in the cases in question. Pus has generally been diffused among the nerves of the cauda equina, and beneath the spinal arachnoid, up to the base of the brain. In one case the inflammatory products lay outside the theca vertebralis.

Other forms of acute spinal meningitis accompany the various kinds of inflammation of the membranes of the brain, which will be fully described hereafter. A "hæmorrhagic spinal pachymeningitis" is occasionally found in association with a similar affection of the cerebral dura mater. Of a primary acute inflammation of the spinal membranes, limited to them, and not involving the encephalic structures, I have no knowledge.

Chronic spinal meningitis.—Certain local forms of meningitis, occurring chiefly in association with syphilitic gummata, or with caries of the vertebræ, have been already mentioned. But it sometimes happens that the whole of the spinal membranes are found matted together and thickened round a cord which appears to be perfectly healthy. I once made an autopsy on a case of this kind, in an old woman of seventy, who had for about a year been suffering from a slowly advancing paraplegia. So far as I could learn, she had had no spasms of the legs, nor had they been rigid; she lay helpless in her bed, but the lower limbs still retained some power of sensation, so that she could distinguish heat from cold. She complained of pains in the legs (especially in the left one), and in the lower part of the abdomen, but it did not appear that they were severe. The visceral arachnoid, when it had been stripped off the inner surface of the dura mater, was found to be as thick as a sheet of writing paper. In the lower part of the dorsal region the adhesion of the membranes to the cord was particularly close for about an inch and a half of its length. The thickening extended upwards around the pons, and to the under surface of the cerebellum, so that the cerebro-spinal aperture was closed. I thought at the time that the absence of any marked irritative symptoms might perhaps be due to the fact that the cord itself was also extensively affected; but Dr Frederick Taylor, under whose care the case was during life, has since told me that he could detect in it no morbid change, except that the fibrous septa entering it from the surface appeared thicker than usual.

A similar case was recorded by Gull in his well-known paper in the 'Guy's Hospital Reports' for 1856 (Case 7). In that instance rigid flexion of the legs was a marked symptom, and they were also affected with frequent spasms. But it is not clear to me that the affection could have been satis-

factorily diagnosed from a transverse myelitis in the upper dorsal region, with "exalted susceptibility" (or descending lateral sclerosis) of the cord below.

Charcot describes a form of primary inflammation of the spinal dura mater, producing pain and partial paralysis in one or the other arm—Pachymeningitis cervicalis hypertrophica. The origin of the ulnar nerve seems most often to suffer. This affection will be described below (p. 514).

Some modern German writers describe chronic diffused spinal meningitis as of frequent occurrence, and as being often recognised during life without much difficulty. It is characterised, says Erb, at first by pain and stiffness in the back, a troublesome "girdle sensation," a feeling of weight in the legs, numbness, tingling, tearing, or dragging pains in them, and cutaneous hyperæsthesia. After a time partial paraplegia develops itself, which varies in degree from day to day. Erb supposes that such fluctuations depend upon alterations in the quantity of fluid effusion within the spinal canal, or perhaps in the amount of blood in the vessels about the cord. There is generally but little impairment of sensibility; and, according to Braun, the sphincters retain their functions. Erb speaks of the morbid change in the membranes as being usually but little marked; they are more or less thickened, opaque, and adherent to one another; and there is an excess of fluid round the cord. Cold is said to be the chief exciting cause of this affection; it has often been observed in officers after severe fatigue and exposure during a campaign. It then commonly begins acutely with fever, and passes into a chronic form afterwards. In many cases it becomes complicated with myelitis, and the peripheral part of the cord beneath the pia mater sometimes undergoes sclerosis in an annular form.

How far one is justified in maintaining an anatomical diagnosis of cases of this kind is, I think, very doubtful at present. But the clinical differentiation of them appears to be somewhat important, as regards prognosis and treatment. Erb says that a good many prove fatal by bedsores, cystitis, or exhaustion; but he admits that others recover more or less completely; and Braun declares that the prognosis is very favourable, if the acute stage, in spite of the increasing paralysis, be treated at first with local bleeding and local cold applications, and then with warm baths. Afterwards, when paralytic symptoms alone remain, the patient should begin a steady course of thermal baths, the brine baths of Rehme or Nauheim being preferable, for which places the English equivalent appears to be Droitwich. Erb endorses Braun's statements, and declares that when there is no myelitis, baths at high temperatures (98°—108° Fahr.) are well borne, and that the reputation of curing spinal paralysis in general which is possessed by hot springs is really derived from their success in this particular class of cases. The cold-water system is also said to be useful; and Hitzig and Erb have found benefit from passing galvanic currents along the spine. Iodide of potassium is the chief medicine which is given; but good food, wine, and tonics are important auxiliaries in the treatment.

SPINAL PARALYSIS ATTENDED WITH WASTING OF MUSCLES

General characters of this group of diseases of the cord.
SUDDEN ATROPHIC SPINAL PARALYSIS—*In infants—Its course, sequelæ, anatomy, and treatment—In adults.*
SUBACUTE ATROPHIC SPINAL PARALYSIS—*Its course and anatomy.*
LEAD PALSY—*Distribution—Diagnosis—Tremors—Treatment.*
CHRONIC ATROPHIC SPINAL PARALYSIS—PROGRESSIVE MUSCULAR ATROPHY—*Symptoms—Pathology—Primary and secondary forms—Diagnosis—Treatment.*
Bulbar Paralysis—Progressive muscular atrophy in children—The hereditary form.
PSEUDO-HYPERTROPHIC PARALYSIS—*Primary (myopathic) muscular atrophy.*

General character of this group of diseases of the cord.—The affections discussed in the last chaper are all accompanied by paraplegia; and when their anatomical seat is ascertained, it is (for all but one) a lesion of the cord which is "indiscriminate," *i. e.* whether it affects the whole organ, as "diffuse" myelitis, or one or more segments only, as transverse myelitis and paraplegia from compression, or the surface, as meningitis, or the deepest parts, as peri-ependymal myelitis—in all these varieties of disease, the parts are affected indiscriminately throughout the diseased segments.

The only exception was spastic paraplegia, where we found reason to believe that the lesion is in the most typical cases limited to the crossed pyramidal tracts of the cord.

There are several other spinal diseases which agree in this particular. They affect particular tracts of the cord, and as a rule do not transgress these anatomical limits. They are chronic in their course; they are much less dangerous than acute myelitis or paraplegia from compression; and the anatomical process which presumably causes their symptoms is identical with chronic interstitial myelitis, *i. e.* grey degeneration or sclerosis. They are called by German pathologists "system-diseases," an uncouth and obscure phrase in English. *Longitudinal*, as opposed to *transverse*, myelitis would express their range. Perhaps the term "columnar" sclerosis may serve better to denote the characteristic manner in which the same degenerative process is limited to the longitudinal columns or tracts of the cord.

The longitudinal, columnar or systemic scleroses which have been as yet observed are the following:*

1. Degeneration of the lateral columns (crossed pyramidal tract) is pri-

* The recognition of the physiological division of the white substance of the cord into distinct tracts of ascending and descending fibres is due to Türck, of Vienna, who in 1851 and 1853, described them as regions of secondary degeneration after lesions of the brain and cord. Flechsig subsequently showed that the same distinction is observable in development, the fibres of the anterior column and anterior root-zone acquiring their myelin first, those of Goll's tract and the direct cerebellar tract next, and the crossed pyramidal tracts last, not indeed until some weeks after birth. (See Dr Bramwell's excellent plates, 19, 20, 21.)

mary, or more frequently secondary; and descending from a primary lesion of the motor tract in the brain or cord. The corresponding disease is that described in the last chapter as Spastic Paraplegia.

2. Of the antero-median column of Türck. This is always secondary and descending. It does not correspond to any known clinical group of symptoms.

3. Of the posterior columns of Burdach (*fasciculi cuneati*). This is usually primary, and corresponds with the clinical malady which will be described in the following chapter as Tabes Dorsalis.

4. Of the postero-median columns of Goll. This is almost always a secondary lesion, often combined with the last in tabes, sometimes appearing as an ascending degeneration from a transverse lesion. No constant symptoms are known to answer to it.

5. Of the direct cerebellar tract. This is always secondary and ascending. It also does not give rise to any ascertained malady.*

6. Of the large motor and trophic ganglion-cells of the anterior cornua. This is sometimes an acute, sometimes a chronic process, and is often confined to a single segment or to some part of the anterior cornu in a single segment.†

In the present chapter we deal with affections for the most part identified with the last of these "systemic" scleroses. But they are also united by a common symptom, that of marked and rapid atrophy of the paralysed muscles, as well as negatively by the absence of anæsthesia, trophic changes in the skin, or affections of the bladder and other pelvic organs.

They may for the present be called in common Atrophic Spinal Paralyses.

Acute Atrophic Paralysis.‡—In the last century this affection was described by Underwood in his celebrated treatise on the 'Diseases of Children,' and Jacob von Heine published a monograph on it in 1840. Both this writer and afterwards Duchenne, of Boulogne, expressed the opinion that the cause of infantile paralysis was a lesion of the spinal cord. But as autopsies failed to reveal any morbid change, MM. Rilliet and Barthez proposed the name of "paralysie essentielle." Since 1863, however, more refined histological methods have shown that the anterior cornua of the cord are diseased. Accordingly German writers now designate the affection as "poliomyelitis§ anterior acuta." We cannot employ the old name of "infantile paralysis," because it is now ascertained that the same form of disease occasionally occurs in adults.

Onset and course.—A child who is about to suffer from acute atrophic paralysis is sometimes apparently well until the loss of power appears. But more often he falls ill with some slight febrile disturbance, oppression, or drowsiness. Sometimes, however, the earliest symptom is an epileptiform convulsion, or there may be spasmodic twitchings of the face or limbs,

* Secondary sclerosis of the "mixed tract" outside the anterior root-zone (antero-lateral column) has been observed by Dr Gowers and by Dr Tooth. It occurred as an ascending degeneration after fracture of the spine. Its pathological significance is as yet unknown ('St Barth. Hosp. Reports,' vol. xxi).

† The cells which form the posterior vesicular column of Lockhart Clarke appear also to be subject to a destructive process which affects them alone.

‡ *Syn.*—Acute amyotrophic spinal paralysis—Poliomyelitis anterior acutissima (Kussmaul)—Anterior cornual myelitis (Gowers)—Infantile Paralysis—Wasting palsy of children.

§ *i.e.* Myelitis affecting the *grey* matter (πολιός). Charcot proposed Tephromyelitis, τεφρός (ash-coloured, cinereous), and Vulpian, Spodiomyelitis (σπόδιος, also ash-coloured, like the last, but an epithet only used in poetry).

grinding of the teeth, and rolling of the eyes. The fever usually lasts a day or two, but it may pass off in a few hours, or may continue for a week or longer. Convulsions may be repeated during twenty-four or forty-eight hours.

When the child seems to be getting better, and is being washed or dressed —or perhaps when it first attempts to stand—the mother or the nurse finds to her horror that one or more of the limbs is powerless. It may have gone to sleep at night with full use of its arms and legs, and in the morning one or more of them may hang flaccid and motionless. Most often the affection may be a monoplegia, the paralysed limb being most often the right leg, but sometimes an arm; or it may be a paraplegia, or a hemiplegia, or a crossed paralysis of one arm and the opposite leg. Occasionally there is loss of power of the trunk or of all four limbs, and sometimes they are attacked in succession at intervals of a few hours.

Occasionally the initial fever is very slight; it may perhaps be absent, and the paralysis be the first symptom that the child is unwell. But, as might be anticipated, there is no thermometric evidence of this.

It is characteristic of acute atrophic paralysis that the affected parts are completely relaxed. No reflex movements can be elicited, whether by stimulations of the skin or of the tendons. On testing the muscles with faradic currents, one finds that their contractility becomes markedly diminished within the first four or five days, and that it is entirely extinguished at the end of a week or a fortnight, but the susceptibility to galvanic currents is increased. In other words, the reaction of degeneration (see p. 393) is present. Rapid and early wasting of the muscles takes place. Until recently they were said to undergo fatty degeneration, but in reality the histological changes in them are of a different kind, corresponding exactly with those which I have already described at p. 395 as following some lesions of the nerve-trunks. The surface of the paralysed limbs is cold, and they are pale or of a livid bluish tint; the volume of the arterial pulse in them is said by Volkmann to be diminished, and Charcot states that after death the main blood-vessels are found remarkably reduced in size.

As a rule, no impairment of sensation can be detected: the child cries lustily as soon as the poles of a galvanic battery are applied. The functions of the bladder and rectum are performed normally or nearly so, and bed-sores never appear.

Unlike almost every other form of paralysis, that now under consideration shows no tendency whatever to gradual advance. The limbs are often attacked one after another, but the loss of power in each of them, although not absolutely sudden, becomes complete within a few hours. Erb says that the whole development of the disease, in successive outbreaks, may be protracted over a week; it generally occupies much less time.

In fact, no long period elapses before recovery begins to take place; sometimes this is observed within a few days, more often in the course of two or three weeks. Even muscles which have to some extent undergone wasting regain their size and strength. If the arms and the upper part of the trunk are affected they are particularly likely to get quickly well. In the course of a few months the disease may entirely disappear, especially when it was all along confined to a single limb. Some writers have disputed the claim of such a "temporary paralysis" to be classed with these forms in which there is permanent loss of power; but I agree with Volkmann in thinking that it is the same disease. These favourable cases are, however,

very exceptional. Almost always the recovery is incomplete. It goes on for a month or eight weeks, so that the parents of the child cherish the most pleasing hopes; but after this the progress becomes slower, and at the end of six or nine months it ceases for ever.

The condition of the little patient at this time is very variable. One limb or two may be powerless and shrunken throughout, or the affection may have become limited to certain segments of an arm or of a leg. As a rule, the distal parts are more apt to suffer than those nearer the trunk. Volkmann, however, speaks of one patient as having a muscular forearm attached to a humerus like a stick, and Erb says that the deltoid sometimes suffers alone. Nay, the acromial part may escape, while the rest goes. Certain muscles exhibit a peculiar independence, now by remaining paralysed when those near them get well, now by recovering while the rest are undergoing atrophy. The following usually escape: the *supinator longus* of the forearm, the *tensor fasciæ latæ* and *sartorius* of the thigh; while the *deltoid*, *serratus magnus*, *quadriceps extensor*, *gastrocnemius*, and *peronæi* are apt to suffer; or the last-mentioned may suffer alone, and the other extensors of the front of the leg escape.* When a muscle wastes, the destruction of its fibres is not necessarily made manifest by a corresponding diminution of its mass, for interstitial development of adipose tissue often takes place, which may entirely conceal it. Erb says that this is particularly apt to occur in young children, and that a muscle may actually appear bigger than in the opposite healthy limb, although it scarcely contains any contractile elements.

Another peculiarity of parts affected with this disease is that they do not grow at the natural rate. Hence a paralysed limb is commonly shortened to the extent of an inch or an inch and a half, and sometimes by as much as eight inches. Even the corresponding half of the pelvis may remain undeveloped. The bones are thinner and more spongy and their processes smaller than on the healthy side. Remembering Hilton's observations with regard to the arrest of growth in the feet of patients suffering from disuse of the hip or knee-joint, one might be disposed to refer all these conditions to disuse, but this is certainly not the case. The disused muscles of hemiplegia are small and flaccid, but they do not atrophy like these. Moreover, the muscles waste even when the paralysis is very partial, so that the child halts very little and is on its legs all day. Volkmann saw persistent failure of development in four or five cases of "temporary paralysis," in which all the muscles recovered.

But the most important remote effects of acute atrophic paralysis in children are the contractions and deformities which are so commonly observed. I remember, twenty years ago, hearing Dr Wilks insist on the fact that club-foot, when not congenital, is the result of paralysis, and not primarily a spasmodic affection; and this view, which was opposed by "orthopædic" surgeons, is now generally accepted by pathologists. But it was reserved for Volkmann to show how the deformities are produced. Until recently, the explanation which was accepted was that contraction was generally the result of the unopposed action of the antagonists of the muscles paralysed. But Hüter and Volkmann have shown that when there is loss of power in several groups of muscles, it not infrequently happens that those on which the paralysis is most marked lie *within* the open angle formed by the dis-

* On this curious distribution of paralysis, seen also in the chronic form of atrophic paralysis and in lead palsy, light is thrown by the interesting experimental evidence, obtained by Professors Ferrier and Yeo, on the motor roots of the nerves of the extremities in the ape ('Proc. Royal Soc.,' March, 1881).

placement of the bones at a joint. For example, a "genu recurvatum" of which the concavity is directed forwards, may appear when the *quadriceps extensor* is powerless, or a "talipes equino-varus," when no contractions can be obtained by galvanising the calf of the leg. Volkmann explains these facts by showing that they are in part due to the influence of gravitation; thus the weight of the foot causes the toe to point downwards, when the limb hangs in the air. But another important factor is the gradual strain on a joint, in efforts made by the patient to employ the weight of the body as a propelling force. Volkmann points out that when a person whose thigh-muscles are paralysed learns to walk without crutches he swings the trunk forwards so as to extend the knee as fully as possible. The result is that after a time the ligaments behind the joint yield, and the knee becomes bent the wrong way. The articulations in such paralysed limbs often become exceedingly loose. Lastly, a subordinate element in the causation of deformities is the contraction of the new connective tissue developed in the interstices of the wasted muscles, a result which has been compared to "sclerosis" of the cord and "cirrhosis" of the liver.

Distribution.—Putting together sixty-two cases collected by M. Duchenne, fils, thirty-two of Dr West's patients, and sixteen of Dr Goodhart's, we find that of the total 110 patients, the right leg only was paralysed in thirty-six (a third), the left only in twelve, both legs in nineteen (paraplegia), one or other arm in seventeen (making sixty-five cases of monoplegia, or more than half), hemiplegia occurred (right five, left five, crossed two) in twelve, facial palsy in seven (all observed by Dr West); while in five cases, three of the limbs, all four, or the muscles of the trunk were paralysed.

Morbid Anatomy.—Within the last twenty years the morbid anatomy of the disease has been very carefully studied, and probably all the most essential points have been ascertained. Cornil, in 1863, was the first to record an autopsy in which changes were found in the cord; but the body was that of a woman who had been paralysed when a child, nearly half a century before.

The earliest autopsy after the attack which has hitherto been made was by Royer and Damaschino, in the case of a boy who died of scarlet fever within two months of the onset of the paralysis. The results of this, and of a few other observations made within two years, are summed up by Erb as follows:—To the naked eye there is generally no perceptible alteration. Sometimes the cord seems rather tough, and its antero-lateral columns are a little shrunken at the level of the lumbar or of the cervical enlargement. Its structure may be slightly blurred. The fore part of the grey matter may be whitish or pink and softened, or even reduced in size, the corresponding motor nerve-roots may be atrophic, grey, and translucent. The microscope reveals an extensive area of morbid change, perhaps even one inch or more in length, in this region. It is sometimes confined to one anterior grey cornu, sometimes it involves both of them, according to the distribution of the paralytic symptoms during life. Granule-masses are generally present in abundance; there is nuclear overgrowth, the vessels are dilated and full of blood, in other words, there is an *inflammatory softening.* But the most striking change is a more or less complete disappearance of the large multipolar nerve-cells, many of them that still remain being in various stages of atrophy. The nerve-fibres also have undergone destruction, even to their axis-cylinders. At the periphery of the affected area the nuclei are often massed together in large numbers, so

as to form a kind of capsule, but slight diffused lesions are discoverable throughout the grey matter of a large part of the cord, generally in the whole length of the dorsal region. In the anterior and lateral white columns there is little change to be seen; a slight degree of sclerosis sometimes, thickening of the trabeculæ, atrophy of a few of the nerve-fibres, granule-masses, scattered sparingly. The anterior roots display all the appearances of "degenerative atrophy."

When many years have elapsed since the onset of the paralysis the appearances are far more marked. The shrinking and atrophy of the anterior part of the cord is now evident on the cut surface, especially when the affection is one-sided, in which case the symmetry of the two halves is lost. The anterior and lateral columns may be obviously grey in tint, and translucent as compared with the posterior columns. The anterior cornu is less deeply coloured by carmine because the protoplasm and nuclei of the motor cells are gone, but the adjacent white substance is more so because the myelin is gone, and Deiters' cells and even fibrous tissue stain better. Under the microscope the diseased area in the lumbar, or the cervical, enlargement is seen to consist almost entirely of a delicate connective tissue, containing an immense number of corpora amylacea, but now no granule-masses. No normal ganglion-cells or nerve-fibres are at this stage to be seen; any remnants of cells are shrunken and pigmented. The anterior cornu often contain the small-branched spider-cells known as Deiters'.

That a local affection of the spinal grey matter should set up such violent fever as we have seen to be often present in this disease is surprising; but we seem to have no alternative but to regard it as symptomatic. There may, indeed, be a question as to whether the myelitis is not at first diffused, and whether it does not clear up and subside throughout the greater part of the cord, while one particular region, to which it becomes limited, undergoes entire destruction.

There have been many discussions as to the starting-point of the affection, whether it is in the multipolar cells or in the neuroglia of the anterior cornua. Charcot insists that the fact of its strict limitation to certain regions of the cord is strongly in favour of the former view; he also says that at some points the cells may be found diseased while the connective tissue remains all but normal. Erb thinks that perhaps both tissues become simultaneously diseased. The muscles are diminished in volume, and their fibres are replaced by fatty or by fibrous tissue.

Ætiology.—With regard to the *causes* of acute atrophic paralysis very little is as yet known. It is far more common in children between one and three years of age than in those who are older, but it not rarely develops itself in infants from six to twelve months old, and Duchenne has recorded cases occurring at the ages of twelve days and one month respectively. It may occur at any age up to puberty, so that it is not separated by any absolute line of age from the atrophic spinal paralysis of adults.

The two sexes appear to be equally liable to it during childhood. Whether teething plays any part in its ætiology is very doubtful. Wharton Sinkler, an American observer, found that it arose very much more often between the months of May and September than during the rest of the year, the proportion being forty-seven cases to ten, and this is confirmed by Ross, Gowers, and other observers. In July and August Dr Sinkler had seventy-seven cases out of 149, and in the same months Dr W. H. Barlow, of Manchester, forty-eight out of 111. The so-called neuropathic family tendency

seems seldom to be present. Duchenne says that he has never seen this affection in two children belonging to the same family, and the few cases on the other side that have been published may probably be mere coincidences. Those who are attacked by it are commonly neither rachitic nor scrofulous, but robust and healthy. Sometimes it is directly set up a by chill (?), at least Erb says that this has been demonstrated beyond dispute. Sometimes it arises during convalescence from some acute disease, such as scarlet fever, mild smallpox, typhoid fever, or ague. This may also be the case in adults. Some years ago I saw a marked case in a young man, aged twenty-two, who was just getting better of pleurisy. In women it may occur at the puerperal period.

The *diagnosis* is seldom difficult, if one keeps in mind the clinical history and features of the disease. One must of course remember that other forms of paralysis may occur in childhood; perhaps that which is most likely to cause a mistake is a peripheral paralysis of the brachial plexus from sleeping on the side, or from pressure by a band fastened tightly round the arm. In the case of an infant brought by a person ignorant of the circumstances it may be impossible to come to a positive conclusion.

Prognosis.—So far as is known, anterior corneal myelitis is never fatal, whether in a child or in an adult; but complete recovery is rare. When a child first comes under observation at an advanced stage, with the muscles already wasted, one is apt to think that the issue would have been better had treatment been earlier. But if it is placed under treatment from the commencement there is great difficulty in saying how far any improvement that occurs is attributable to the remedial measures that may have been adopted.

Treatment.—Next to keeping the palsied limbs warm and promoting their circulation, the application of galvanism to the spine seems to be the most important thing. The poles must be large, and a current of moderate strength may be passed for one or two minutes at a time. Afterwards the paralysed muscles may be galvanised in turn. Faradisation may also sometimes be useful if the muscles have not lost their susceptibility to induced currents. Even when several years have elapsed one must never hastily conclude that the electrical treatment will necessarily be fruitless. It now and then happens that a very few applications produce extraordinary effects, and very often, by persevering for several weeks or even for months, one at length attains considerable success. In one case—of which I unfortunately have no notes—the internal administration of strychnia was, beyond dispute, the means of restoring to a child the power of standing. Shampooing and friction with stimulating liniments may sometimes be of service. Dr Angel Money observes that massage well carried out is far more useful than electricity. Nourishing food, cod-liver oil, and tonics are matters of course. For the avoidance of the different forms of contracture recourse must be had to mechanical apparatus. Volkmann says that drawing up of the limb may be prevented by fastening the feet every night on a splint with a flannel roller, and carrying a strip of plaster across to the leg.

Acute atrophic paralysis in an adult develops itself in most respects as in infants. Meyer, of Berlin, is said to have been the first who, in 1868, recorded two cases of this kind after measles. Duchenne, Erb, and many others have since written upon the subject. The disease seems never to set in with epileptiform convulsions; but there is fever of greater or less intensity, with headache, drowsiness, and occasionally even delirium. After

a single night, or at the end of a few days, one or more of the limbs becomes paralysed; sometimes there is a transitory failure of the bladder; there is not the slightest impairment of sensibility, the muscles are perfectly relaxed and their susceptibility to reflex stimuli is lost. Their electrical reactions are like those of the same disease in children. The ætiology is equally unknown, but it is much more rare in women than in men. Recovery may either be complete or partial; in the latter case the affected parts become contracted and deformed, but not to the same extent as at an early period of life. Of course, there cannot afterwards be any difference in the length of the limbs on the two sides of the body. In the only marked case under my care in Guy's Hospital, the patient, a healthy railway servant, aged about twenty-five, the paraplegia gradually but completely disappeared, and he is now well.

Chronic and Subacute Diffused Atrophic Paralysis.*—Within the last few years writers on spinal diseases have recognised as a distinct affection a form of paralysis which was first described by Duchenne in 1849 and 1853 under the name of "*paralysie générale spinale antérieure subaigue.*" He suggested that it was probably due to an atrophy of the cells of the anterior grey cornua; and as recent observations have shown that there is a chronic inflammatory change in this part of the cord, Erb proposes to call the disease "*poliomyelitis anterior subacuta et chronica.*" It seems to me that the most suitable designation is "chronic diffused atrophic paralysis;" this at once distinguishes it from the "*acute* atrophic paralysis" already described, and also from the *limited* forms of paralysis which are usually known by the title of "progressive muscular atrophy."

Course.—It generally begins in the lower limbs. The patient first experiences a sense of weakness in one or both of his legs, especially in going upstairs, or if he attempts to walk far. At the end of a few days, or in the course of some weeks, this develops itself into actual paralysis, so that he is obliged to keep his bed. Before long the upper limbs are affected, especially the fingers and wrists. The muscles of the trunk also become powerless, so that he cannot sit up, and has difficulty in coughing or sneezing, and even in passing his fæces.

The paralysed muscles are absolutely lax and flaccid. No reflex movements can be excited in them by irritation, either of the skin or of the tendons. They very rapidly waste, so that with a tape one can follow the loss of substance in the calves or in the thighs from week to week, until the bones seem to be covered only by the integuments. When electrical tests are applied to them they are found to present the reaction of degeneration. Duchenne long ago discovered that their susceptibility to faradic currents is extinguished. Others have shown that they react well to interrupted galvanism, that A.C.C. is more marked than C.C.C., and that the contractions are slow and tonic in character.

So far there is nothing in the symptoms of chronic diffused atrophic paralysis which is distinctive of this affection rather than of myelitis, spreading upwards through the whole substance of the cord from its lower end. What shows that the lesion is limited to the anterior cornua is the absence of anæsthesia, the retention of power over the bladder and rectum, and the fact that there is no tendency to the formation of bedsores. Erb, indeed, says that pains in the back and slight paræsthesia in the limbs are not un-

* Chronic myelopathic atrophic paralysis of adults—Poliomyelitis anterior chronica.

commonly present, and may even be complained of before the paralysis has developed itself; but on testing the cutaneous sensibility one finds it absolutely perfect, or if blunted at all it is so only in the smallest possible degree. At the commencement there may be a little febrile disturbance, with headache and digestive disorder, but these quickly pass off and the patient afterwards feels quite well, and eats, drinks, and sleeps as usual.

The further progress of the disease varies in different cases. Generally it remains for a certain length of time stationary; that is, it ceases to spread upwards, or to extend to fresh sets of muscles, although those already affected go on wasting. Erb says that sometimes at this period the joints become slightly contracted. But at last, after several weeks, a gradual improvement begins to be observed; the upper limbs regain some of their movements, and afterwards the lower limbs some of theirs. All this takes place very slowly; months pass before the patient can feed himself, or write, or walk. Sometimes, however, he ultimately becomes as muscular and as active as before, but sometimes some particular muscles remain wasted for the rest of his life; according to Erb this is especially apt to be the case with the peronei. As recovery goes on, the electric contractility of the muscles gradually reappears.

On the other hand, there are cases in which no change for the better occurs, but in which the paralysis extends upwards to affect the face and the tongue and to interfere with deglutition and with respiration. In this way the disease may terminate fatally from one to four years after its commencement. It does not appear that there are any special indications which might enable a correct prognosis to be given at an early period of the case.

Histology.—Up to the present time only two autopsies appear to have been made in which the cord was examined by the modern methods. One was recorded by MM. Cornil and Lépine, the other by Dr Webber.* In each instance the multipolar nerve-cells in the anterior grey cornua had undergone a more or less complete destruction; there were evidences of inflammatory changes in the presence of compound granule-masses, the proliferation of the cells of the neuroglia, the accumulation of nuclei round the blood-vessels, and the like. In the antero-lateral columns there was a certain degree of sclerosis or degeneration of nerve-fibres. The motor nerve-roots and the muscles were in a state of extreme atrophy. In other words, the morbid anatomy of the disease appears to correspond exactly with what might have been anticipated on theoretical grounds.

With regard to the *ætiology* of chronic diffused atrophic paralysis nothing is as yet known. It is a rare affection, most apt to occur in persons between the ages of thirty and fifty, but in this, while differing from acute atrophic paralysis, it resembles several other spinal affections. It has sometimes been ascribed to cold or to an injury of the back, sometimes to intemperance or to sexual excesses.

The *diagnosis* is, in well-marked cases, easy. It is separated from ascending myelitis by the absence of anæsthesia and of pelvic symptoms; from acute atrophic paralysis of adults by its gradual instead of sudden advent; from progressive muscular atrophy by the paralysis preceding instead of resulting from the muscular atrophy, and by its better prognosis, from multiple peripheral neuritis by the absence of pain and of the peculiar

* 'Trans. American Neurological Association,' vol. i, p. 45.

distribution of that affection; and lastly, from lead palsy by the absence of the blue line and other proof of the presence of the poison.* There is no question that this chronic diffused paralysis closely resembles the diffused form of paralysis which occurs in persons poisoned by lead. Indeed, it is a question whether this is not dependent on the very same change in the anterior grey cornua of the cord; whether, in fact, we ought not rather to enumerate chronic plumbism among the causes of the disease now under consideration. I believe that the only distinctions are the presence of the characteristic line upon the gums, and of other effects of the metal, such as colic, sallowness of complexion, and gout. Erb says that he has seen two cases of paralysis limited to the upper limbs, which, in the absence of all evidence of lead-poisoning, he was obliged to regard as instances of a primary "polio-myelitis anterior," limited to the cervical part of the cord.

The *treatment* of the disease appears to be that of chronic myelitis in general; the chief remedies being iodide of potassium, ergot, and the application of galvanism to the spine. The paralysis is not always progressive, and recovery has been observed.

Paralysis from Chronic Plumbism.—Beside causing a form of colic and inducing gout, lead may also affect the nervous centres and give rise to a variety of symptoms.

Of these the most common is a form of paralysis affecting the upper limbs, which from its peculiar characters is often called "the dropped wrist." I believe that this always attacks certain groups of muscles much more than others. Generally the extensors of the hand and the supinators are the chief ones to suffer; the patient's hand hangs powerless from his wrist and his fingers are more or less forcibly flexed. When the forearm is laid prone upon a table he is unable to turn it round so as to bring the palm uppermost. The muscles which form the ball of the thumb are affected very frequently and sometimes before any others; those of the little finger also as well as the lumbricales and interossei. In some cases the deltoid muscles are first attacked, and with them the lower part of the trapezii† and the muscles which cover the dorsal surface of the scapulæ. The affected muscles always become greatly wasted. Thus the ball of the thumb, instead of being rounded, is sunken; the loss of substance in the interossei and lumbricales causes the flexor tendons to be visible on the palm of the hand; the neighbourhood of the external condyle of the humerus and the back of the forearm are hollowed out and flaccid. If the shoulder is attacked it loses its roundness, and the outlines of the bones can be felt much more plainly than is natural. In some cases the whole upper limb may be weak and all its muscles more or less wasted. But I have never seen a case in which the biceps and triceps in the arm and the flexor muscles in the forearm were affected to the same extent as the extensors.

Most frequently both upper limbs are attacked, but one much more than the other. Sometimes the paralysis is limited to one arm and hand. Certain writers have supposed that this is because they alone happened to absorb

* In this paragraph I follow an excellent article by Dr E. Remak in Eulenburg's 'Real-encyclopädie der gesammten Heilkunde,' but I think it must be admitted that diagnosis is often difficult; sometimes impossible, or even perhaps unreal.

† We shall see that this curious immunity of the upper part of the trapezius also obtains in progressive muscular atrophy. Compare the muscles most often affected in that disease (*infra*, pp. 507, 508) and in infantile palsy (*supra*, p. 449).

the poison, as, for instance, when a painter makes his colours with one hand, and little or not at all with the other. But such a view is altogether untenable, and I believe that any difference between the two upper extremities in this respect is to be attributed to the fact that the arm most affected is employed more than the other in the patient's daily work.

It is, however, a question whether lead paralysis is really due to an affection of the nervous centres, or whether it may not be caused by the local action of lead circulating in the blood upon the muscles themselves. John Hunter long ago took advantage of a fatal accident which had happened to a painter to examine the tissues of his hand and arm, which had been paralysed. He found that the muscles were cream coloured. Some years ago a man died in Guy's Hospital who had been attacked with lead paralysis seven years before, and had never completely recovered from it. Dr Moxon found that the affected muscles—especially the deep extensors of the forearm —were represented only by loose watery connective tissue.

The paralysis which occurs in chronic plumbism is not always limited to the upper limbs. I have seen two instances in which the whole body was affected. The patient could not raise himself in bed, nor use any of his limbs.

Sometimes, however, the diagnosis between lead paralysis and progressive muscular atrophy is so uncertain that the results of treatment have to be called in evidence upon it, and I am myself inclined to think that the latter affection, in its most typical form, is sometimes really the result of plumbism. I have met with at least one well-marked instance in which a paralysis of the forearm, presenting all the characters of progressive muscular atrophy, including its resistance to treatment, occurred in a man who had worked with lead for a length of time, and who had a well-marked blue lead line. The hypothesis that lead palsy is not central at all but due to peripheral neuritis has been already referred to (*supra*, p. 432).

Diagnosis.—We have already seen the close relation between lead palsy and chronic atrophic spinal paralysis in their symptoms. The only other disease for which lead paralysis is likely to be mistaken is progressive muscular atrophy. One distinction between them—on which Sir William Gull used to lay stress—is that in chronic plumbism the patient complains of pain when one grasps the affected muscles with one's hand, but I do not find that this is constantly, or even generally, the case. On the other hand, it is not attended with the fibrillary tremors which accompany progressive muscular atrophy. But the most remarkable distinction is afforded by the application of galvanism to the skin over the affected muscles. In lead paralysis faradism causes but slight contraction or none at all, whereas interrupted galvanism gives rise to movements more readily than in health, that is, a smaller number of cells is required to excite the muscles to contract, and the other peculiarities of R.D. are well marked. This is absent or ill-developed in progressive muscular atrophy. Whether the palsy is caused by lead or no, we can always ascertain the fact of plumbism.

The general condition of the patient must be carefully inquired into in all cases in which any doubt exists as to the cause of a paralytic affection of the upper limbs attended with wasting. The gums must be examined for the "blue line which is almost always present and practically pathognomic." One must ask whether the patient has suffered from any painful affection of the abdomen which could possibly be "lead colic;" but it is to be borne in mind that when a person has been slowly absorbing the metal in

minute quantities for a length of time, paralysis often occurs without having been preceded by any pain in the bowels, and these are precisely the cases which are difficult of diagnosis. Sometimes the recognition of chronic plumbism is materially facilitated by the aspect of the patient, the countenance being in such cases peculiarly sallow and anæmic.

Another nervous affection which is said to be an occasional result of lead-poisoning is a *tremor*, resembling that caused by mercury. It has hitherto only been observed, by Brockmann, among the miners on the Hartz. It is generally limited to the arms and hands, but it very often attacks also the lips and the angles of the mouth. Very rarely it attacks the legs and the muscles of the head and of the trunk.

Treatment.—The medicine most useful in lead paralysis is iodide of potassium. Dr Anstie lays stress on the importance of a nutritious diet, and on the value of cod-liver oil in such cases. Sulphur baths have been supposed to be serviceable. I believe that there is no doubt that they lead to the excretion of some of the lead which has been accumulated in the body; a blackish discolouration of the skin, and still more of the nails, is observed which does not at once rub off, and which evidently is due to the conversion of some of the lead present on the body into a sulphide. Locally the application of a continuous current, just sufficiently powerful to excite contraction of the affected muscles, is very useful. As soon as they will react to faradism, that form of stimulation may be substituted.

PROGRESSIVE MUSCULAR ATROPHY.*—In 1850 a French physician, Aran, described under the name of "*atrophie musculaire progressive*," an affection which he believed to have been altogether overlooked by previous writers, and of which indeed only a few instances had before been recorded, among others by Abercrombie, Sir Charles Bell, and Romberg. Three years later Cruveilhier wrote upon the same subject. Sir Wm. Roberts, of Manchester, published a monograph in 1858. Next to infantile palsy it is the most common and best known of all the diseases described in this chapter, but even this is comparatively rare.

The chief symptom is *muscular atrophy*, and this is the more conspicuous because it often affects a single fleshy mass, or even an isolated muscle or a part of a muscle, while others close by remain as well nourished as ever. In the large majority of instances the upper limbs are first attacked. Aran, whose statement is generally taken as fairly representing the truth, found that this was the case in nine among eleven patients, and that in seven of them the right arm was that in which the disease began. The proportion cannot yet be fixed with absolute accuracy because it is still doubtful whether all the cases in which the wasting is first observed in the legs really belong to the same category. Thus whereas Duchenne says that he met with only two such cases out of 159, Hammond, of New York, speaks of having had eight in a total of twenty-five. Duchenne himself saw one patient in whom the disease began in the *sacro-lumbales*, and one in whom the *pectorales*, the *trapezii*, and *latissimi dorsi* disappeared before the arms became involved. The apparently capricious way in which the atrophy is sometimes distributed is shown by another of this writer's cases, in which, even at an advanced stage, no muscles of the upper limbs except the *supinatores longi* were attacked. Conversely, Trousseau mentions a patient

* *Synonyms.*—Cruveilhier's paralysis—Wasting palsy—Amyotrophie essentielle—Poliomyelitis anterior progressiva longissima—Chronic spinal muscular atrophy.

of Bretonneau's, an old lady, who retained power over none of her muscles save those of the right index finger. She could not speak, but with her finger she picked out of a heap of letters those which she required to form words and sentences, and in this way she made her will.

As a rule, however, progressive muscular atrophy begins in the muscles of one particular region, namely, in the small muscles of the hand, and especially in those which constitute the ball of the thumb. Before passing to the forearm it commonly spreads from the thenar to the hypothenar muscles and also to the *lubricales* and *interossei*. Or, sometimes, it appears first of all in the *deltoid* and other scapular muscles. A curious circumstance is that when it attacks the *trapezius* it always leaves the anterior part of the muscle uninjured; and another peculiarity is that the *triceps* usually escapes, even when all the other fleshy masses in the upper limb have wasted away. The patient's arm then hangs helplessly by his side, he cannot raise it by any muscular effort; if he wishes to lay hold of anything he has to swing it forward with a jerk until his fingers are brought into contact with it. His hand often presents the "griffin" or "bird-claw" deformity. His power of prehension is greatly diminished, especially when the *opponens* is atrophied. Instead of being able to grasp an object between the fingers and the thumb, he has to fix it as well as he can within the hook-like concavity formed by the fingers alone.

Whatever may be the starting-point of the disease, it in most cases soon afterwards shows itself in the corresponding muscles on the other side of the body. But sometimes the symmetry of its distribution is very incomplete; so that, for instance, one forearm and the opposite upper arm may be the regions principally affected by it. Having spread more or less generally over the upper limbs it at length passes to the neck and to the trunk; the lower limbs almost always remain intact until a very late period. But cases are on record of the atrophy beginning in the legs. The face is seldom or never affected.

The loss of substance in the muscles is commonly conspicuously visible through the integuments, and all the more because usually the subcutaneous fat disappears, and the skin itself becomes thin. The ball of the thumb is flat, and there is an obvious hollowing of the metacarpal spaces, especially of that which lies between the thumb and the forefinger; or the shoulder has lost its fulness, the bones forming sharp angular prominences; or the normal rounded curves of the forearm are replaced by shallow depressions. But in stout persons the wasting is not always thus easily to be detected by the eye. It is not merely concealed by the overlying fat; there is often an actual new growth of adipose tissue in the interstices of the affected muscles as in infantile paralysis; and since at the same time a tough fibrous material is present, the part sometimes feels quite as fleshy and firm as in the natural state. Thus Trousseau relates a case in which he could not account for a loss of power in the arms and elsewhere, until Duchenne pointed out to him what he had altogether overlooked, that the deltoid and several other muscles had undergone atrophy. The application of a faradic current may at once clear up all doubt, if the disease is in a sufficiently advanced stage; or a little portion of muscular tissue may be removed for microscopical examination by Middeldorpff's "harpoon," or by Duchenne's "emporte-pièce."

The functional weakness in the affected muscles is increased by fatigue, and to a remarkable extent by cold. More than one patient has told me

that when warm in bed after a night's rest, his hands felt as strong as ever. During the day the hands and fingers often look pale and blue, and they are cold to the touch. Jaccoud and others have taken thermometric observations, from which it appears that the temperature is notably lower on the side on which the muscles are the more wasted; it is said that a difference can sometimes be detected even between the two axillæ. Sometimes there is an excessive secretion of sweat. All these symptoms are referred by Leyden to disturbance of the vaso-motor nerves. In several instances a contracted state of the pupils is recorded as having been noticed.

A symptom on which some writers have laid great stress is the occurrence of transient "fibrillary tremors," or slight quivering movements limited to particular fasciculi of the muscles involved in the disease. They may either be spontaneous, occurring when the part is perfectly at rest, or one may elicit them by giving the skin a smart tap with the finger. All observers now admit that so far from being pathognomonic of any one affection, they may be seen under various circumstances, and even in healthy persons, if carefully looked for. Those which follow taps upon the surface of the muscle are particularly well marked in patients who are emaciated by pulmonary phthisis or any similar disease. But it would appear that in progressive muscular atrophy the spontaneous fibrillary contractions sometimes far exceed those which occur under other conditions, producing perceptible movements of the limbs and attracting the notice of the patient, although they are attended with no pain. It is said, too, that they sometimes afford the earliest indication of the extension of the disease to a fresh group of muscles. Duchenne, however, found them absent in about a fifth of the cases that came under his observation.

The cutaneous sensibility is altogether intact; there is usually no pain in the affected parts; nor are any subjective sensations of numbness experienced. This, at least, is what Charcot says of that form of the disease which he terms "protopathic," and which alone is under consideration now. Leyden and other writers, while pointing out that impairment of sensation is comparatively little marked in proportion to the muscular wasting, have yet described a partial anæsthesia as of rather frequent occurrence. But they doubtless included certain other affections which Charcot has recently shown to be both clinically and pathologically distinct, and of which I shall give some account further on. Occasionally severe shooting pains are felt, especially in the early stages of the disease.

It is not in every case that progressive muscular atrophy manifests a tendency to spread from one group of muscles to another. On the contrary, it may remain for many years limited to those muscles which are first attacked by it. But, as we have seen, it often extends from the limbs to the trunk; and then it is very apt to destroy life by interfering with the respiratory movements. One should always carefully observe the condition of the intercostals, and also that of the diaphragm. If the former muscles fail to contract, the upper two thirds of the chest cease to expand, and the act of expiration is proportionately shortened. If the latter is atrophied the epigastric and hypochondriac regions are drawn in during inspiration. In either case the patient has frequently to stop between his words to take breath. He has difficulty in crying out and in singing, and the attempt to cough or to sneeze causes him great distress. An attack of bronchitis is almost inevitably fatal, so that the greatest care must be taken to protect him from exposure to cold. Not infrequently the intercostal muscles and

the diaphragm fail simultaneously; and then, as the disease advances, suffocation becomes inevitable. Sometimes, on the other hand, the disease proves fatal by spreading to the muscles of the throat and of the pharynx; the symptoms which are then observed will be presently described under the head of "Progressive Bulbar Paralysis." In not a few instances death occurs by phthisis or pneumonia, or an intercurrent attack of enteric fever. Its duration is from two or three to as many as twenty years.

Ætiology.—With regard to the causes of progressive muscular atrophy there is much uncertainty. It occurs chiefly in young adults, seldom or never in children (cf. *infra*, p. 524); according to Leyden, the mean age is about thirty-two years. It is far more common in males than in females. Friedreich found only thirty-three women among 209 patients, and Dr Wm. Roberts only fifteen among ninety-nine. This fact, however, may be regarded from two points of view. Charcot and many other writers think that the disease is frequently transmitted by inheritance, and especially in the male line. That such transmission sometimes occurs is indisputable; but it would seem that the majority of the supposed examples have really belonged to a different affection, which will presently be described under the name of pseudo-hypertrophic paralysis. On the other hand, over-use of the muscles, leading to exhaustion (of them or of the corresponding spinal centres), is often made chargeable with the disease, as when it occurs in a bank clerk, or in a cobbler, or in a saddler; and this would naturally be more common in men, although washerwomen are mentioned as furnishing examples of it among persons of the weaker sex. But here, again, confusion may have arisen from the admixture of cases of "writers' cramp" with those of the affection now under consideration, since until recently the two diseases were often included under the vague designation of "scriveners' palsy," at present fallen into disuse. I have often seen progressive muscular atrophy in persons who had in no way overfatigued their muscles, and I doubt whether the relation between the supposed cause and its effect is not merely one of accidental coincidence. Dr William Roberts and others speak of cold and of blows on the back of the neck as sometimes setting up the disease; but Charcot seems to be right in thinking that the cases in point which have hitherto been recorded have not really been instances of progressive muscular atrophy in a strict sense of the term. A certain number of cases follow closely upon measles and other acute diseases, and others again after a longer interval upon syphilis.

Pathology and histology.—The true nature of this affection has only lately been placed on a satisfactory footing. Both Aran and Cruveilhier originally supposed that it was primarily a disease of the muscles, but the latter modified his opinions when in 1853 he found, in the celebrated case of a rope dancer named Lecomte, that the anterior roots of the spinal nerves were grey and atrophied. The preparation from this case is, I believe, one which I some years afterwards saw preserved in the Musée Dupuytren. However, the occurrence of such a morbid change in a number of isolated nerve-roots, independently of any other lesion, would be scarcely conceivable, whereas the relatively small number of motor fibres in the peripheral trunks renders it easy to understand why in their case a similar affection should escape notice, supposing it to be present. And I believe that those who, since the time of Cruveilhier, have doubted the "myopathic" origin of the disease, have always thought that it would ultimately be proved to have its seat in the cord. This was first demonstrated by Lys, Lockhart, and

Clark. So lately as 1873, Friedreich, of Heidelberg, devoted a quarto volume to the support of the former view, and proposed to term the affection "Polymyositis chronica progressiva." His chief arguments were that the spinal lesions which had in certain cases been found, were very variable in their nature and in their exact position, and that they might fairly be attributed to the extension of an inflammatory process upwards along the nerves. But about the same time Charcot devoted five of his lectures to this subject, and he has shown beyond dispute not only that the most typical form of progressive muscular atrophy in one or both upper limbs is due to a particular change in the corresponding anterior grey horn or horns, but also that other clinical varieties of the complaint can be assigned to separate and distinct affections of the spinal cord.

It seems to me that one thing which has introduced a large amount of obscurity and confusion into the study of this disease has been the adoption by subsequent writers of a phrase of Duchenne's, according to which it is characterised specially by *atrophy without paralysis*. The great electro-therapeutist ultimately abandoned the myopathic theory of its origin which he had at first supported, but he nevertheless continued to maintain that there was no failure of "nervous *motor* action." He thought that the multipolar cells of the anterior cornua naturally possessed two independent functions, the one motor, the other trophic, and he supposed that the *trophic* function alone was interrupted by the lesion of progressive muscular atrophy. He asserted that the loss of power was always proportionate to the degree of wasting which was present, and that any muscular fibres remaining at a particular time would always obey the will, even though they might not succeed in overcoming the resistance opposed to them, so as to effect a visible movement. In proof of this doctrine he appealed to the fact that faradisation of the affected muscles would cause contractions in them even when their substance had undergone the most extreme wasting, provided that some fibres were still undestroyed. Now, it is certain that the maintenance of a normal electro-muscular contractility—which Duchenne formerly maintained to be the condition in progressive muscular atrophy—would be in flat contradiction with what is observed in all other affections of the multipolar cells of the anterior cornua. But I think that the way out of the difficulty is not far to seek. Erb has recently investigated very carefully the action of galvanic as well as of faradic currents, and he says that he has always been able to detect in some of the affected muscles a modified form of the "reaction of degeneration:" the fibres contracted slowly under a powerful galvanic current when they were no longer sensitive to the faradic current, and A.C.C. was greater than C.C.C. The impossibility of verifying this change in other muscles, and the fact that it is easily overlooked, are attributed by him to the partial and gradual character of the wasting process. The fibres which have undergone degeneration have their true nature concealed by the still healthy fibres which lie side by side with them. So far as theory can go, this seems perfectly satisfactory, but Erb himself has overlooked the corollary that it does away with the necessity for supposing that paralysis is not present. It seems to me that those parts of the muscles which are affected by the disease are really as much paralysed as in any other spinal affection whatever. Thus, the principal distinctive feature of progressive muscular atrophy appears to be the way in which the individual fibres of the muscles are one by one attacked and destroyed.

Histologically the morbid process in them is far from being as simple as it was at one time supposed to be. By the earlier writers on the subject they were described as being in a state of "fatty degeneration," having lost their transverse striation more or less completely, and being full of granules and small globules of fat. The accuracy of this statement was soon disputed by Robin, who pointed out that the granules were often dissolved by acetic acid, and therefore could not be of a fatty nature; and it is now known that many of the fibres become merely reduced in size and thickness while retaining their striation, that others pass into the condition known as cloudy swelling, and that others, again, undergo a "waxy" degeneration, exactly like that which we have seen to occur during enteric fever (p. 176). Another part of the process consists in an increase in the number of the sarcolemma-nuclei, and at the same time the interstices between the muscular fibres become the seat of a cell-growth, from which are developed the adipose and connective tissues that sometimes give to the nuclei a deceptive appearance of being still well nourished and substantial. The fibres themselves undergo destruction, not simultaneously, but in succession. Thus there are all possible degrees of wasting, down to a point at which the microscope reveals not a vestige of the normal striation, nor even a single empty sarcolemma sheath. Muscles so affected, instead of red, are pale grey or yellowish, and at length become converted into mere bands of white fibrous tissue, or shapeless masses of fat, passing across from the bones to the tendons.

The histological condition of the cord in progressive muscular atrophy is essentially the same as we have seen to exist in the acute atrophic paralysis of children and in the acute or chronic atrophic paralysis of adults. The large motor ganglion-cells of the anterior cornua are found shrunken or have disappeared, and Deiters' cells or corpora amylacea take their place with fibrous tissue made up of neuroglia, atrophied nerve-fibres, and obsolete blood-vessels, and often considerable pigmentation. The pathological process differs from that of other forms of cornual myelitis chiefly by its exceeding chronicity. It is right to add that the lesion is often found also in other parts of the grey substance, and the anterior horns are said to have been found unaffected in certain cases.

I have already alluded to the observations of Charcot, from which it appears that, under the designation of progressive muscular atrophy, there have been included several distinct lesions of the spinal cord. I must now briefly cite his description of these, and it will be convenient to mention, at the same time, those peculiarities in their symptoms and in their course by which he thinks that they can be clinically distinguished from one another.

1. *Primary progressive muscular atrophy.*—In the typical affection of Aran and Cruveilhier—Charcot proposes to call it *amyotrophie progressive protopathique*—the primary morbid change is an atrophy of the multipolar cells of one or both of the anterior grey cornua. They may either become pigmented or shrivelled without any change of colour. At the same time the neuroglia undergoes proliferation, granule-cells are formed in it, its vessels become enlarged and thickened, and in extreme cases the whole anterior cornu may be found shrunken in all its diameters. The resulting impairment of muscular power presents those characters of sharp and apparently capricious limitation which have already been fully described, and is accompanied neither by disturbances of sensation nor by spasmodic contractions of any muscles. Charcot says that his account of the pathology of this form

of the disease is based upon six or seven autopsies. It is only in thin sections of the hardened cord that it can be recognised with any certainty.

2. *Progressive muscular atrophy combined with spastic paraplegia.*—Contrasting with primary muscular atrophy there are other cases which Charcot terms *amyotrophies deutéropathiques*, because in them the change in the anterior cornua is secondary to a lesion of some other part of the cord. Of these he describes the commonest as *sclérose latérale amyotrophique*, consisting in a chronic inflammatory process which begins in the lateral white columns (occupying them symmetrically on each side), and then spreads into the grey matter, principally in the cervical region. Türck, in 1856, appears to have been the first to notice such a morbid change occurring independently of any disease in the brain. Charcot says that it is characterised clinically by the presence of paralysis with rigidity of the lower limbs, in addition to the atrophy of muscles in the upper limbs. This of course accords perfectly with the modern French doctrine about the effect of sclerosis of the lateral columns in general. The spasm in the legs is at first transitory, but afterwards permanent; they generally assume a position of flexion; they lose but little of their substance. In the arms and hands the muscles undergo wasting *en masse*, and are not picked out one by one, as in the typical variety of progressive muscular atrophy; the elbow becomes somewhat fixed in a position of semiflexion and pronation; the wrist is semiflexed; the fingers are bent into the palm of the hand. Charcot further says that the morbid change in the muscles presents inflammatory or irritative characters in a more marked degree than in the "protopathic" form; the connective tissue between the fibres undergoes a still more decided overgrowth, and the nuclei with which it is infiltrated are more numerous. Another peculiarity is that the course of the disease is more rapid; all four limbs become quickly involved; the patient is confined to his bed in a few months and does not live more than from one to three years, the usual cause of death being an extension of the morbid process to the medulla oblongata, with the symptoms of "bulbar paralysis." This form of progressive muscular atrophy is said by Charcot to be always incurable; those affected by it have been from twenty-six to fifty years old; exposure to cold and damp has sometimes been assigned as its cause. At the Salpêtrière five cases have occurred in which autopsies have been made.

There is no doubt of the accuracy of these clinical observations of Charcot, and lateral sclerosis affecting the crossed pyramidal tracts in the dorso-lumbar region has been discovered after death. But there is good reason to believe that this is a descending secondary sclerosis, and that the primary lesion is the poliomyelitis of the cervical enlargement which produces the atrophic palsy of the arms.*

3. *Pachyméningite cervicale hypertrophique.*—This is the name given by Charcot to a remarkable affection which he regards as another form of *amyotrophie deutéropathique*. As its name implies, it is a chronic thickening of the dura mater, which presents a number of concentric layers, and may fill up the whole vertebral canal. It and the arachnoid adhere firmly to the cord, so as to compress and flatten it; and necessarily surround and press upon those nerve-roots which come off at the level of

* This view was taken by Leyden and is supported by Dr Gowers. See also cases by Dr Ferrier in the 'Lancet' for 1881, p. 822. Erb recognises the validity of Charcot's striking account of the affection and places it provisionally with other forms of spastic paralysis.

the lesion, generally belonging to the brachial plexus on each side. This affection, like that last mentioned, is attended with progressive wasting of the muscles of the upper limbs, and with rigidity of the lower limbs. Charcot, indeed, says that it is distinguished by the circumstance that the ulnar and median nerves are especially involved (the musculo-spiral nerve escaping), so that the wrist assumes a position of extension instead of being flexed. He also remarks that parts of the cutaneous surface often become anæsthetic, not only in the arms, but even in the upper part of the trunk; and that when the lower limbs become rigid they do not seem to waste. But what is especially characteristic of cervical pachymeningitis is the occurrence of an early stage, lasting two or three months, and accompanied by severe pains in the neck and back of the head, by a sort of rigidity of the cervical muscles, by sensations of numbness and tingling in the upper limbs, and sometimes by bullous eruptions. All of these symptoms are of course due to irritation of the nerve-roots at the seat of the disease. The affection evidently stands to the other diseases that are now being described in the same relation as "compression-paraplegia" to the common forms of myelitis. Perhaps the most important point of all is that Charcot describes it as not always incurable. A woman under his care, after an illness which lasted five or six years, and during which she was for a long time perfectly helpless and confined to her bed, ultimately became able to walk, and could also to some extent make use of her hands. The lesion does not tend to spread to the medulla oblongata, so that the symptoms are not apt to become complicated by the supervention of bulbar paralysis. On the other hand, there may at last be loss of power over the bladder and the rectum, and bedsores may develop themselves, symptoms which are not observed in amyotrophic lateral sclerosis.

In 1868 I recorded in the 'Practitioner' a series of cases of Progressive Muscular Atrophy, one of which would seem to have been an example of hypertrophic cervical pachymeningitis, if reliance can be placed on the facts that rigidity of the lower limbs constituted a prominent symptom and that the patient, a woman, aged thirty-two, recovered. At that time I spoke of the affection as altogether exceptional. It is interesting now to refer to the clinical report, and to find that at an early stage she had complained of severe pains about the elbows, which were doubtless due to irritation of nerve-roots. Moreover, three years previously, she had for a time lost power in the second finger of the right hand. I have also reason to believe that a second example of the same affection is afforded by another of my cases, that of G. P. H—, aged twenty-seven, who is recorded as having recovered from a well-marked form of progressive muscular atrophy under treatment by the continuous current. This man was readmitted into the hospital in 1869 with severe cramps and numbness in the arms, tonic extension of the legs, rigidity of the recti muscles of the abdomen, and a sensation of tightness round the waist, as if constricted by a cord. On referring to the notes taken in 1867, I find that at that time he had tingling sensations in the arms and weakness of the lower limbs, so that even then the case deviated to some extent from the typical "Cruveilhier's paralysis."

4. *Hydromyelia.*—Again, in some instances of what must still be called a deuteropathic form of the disease there is a very obvious lesion, in the form of a hollow space occupying the centre of the cord. The first case in which an atrophic paralysis of the upper limbs was observed in connection

with such a lesion was recorded by Gull in the 'Guy's Hospital Reports for 1862. The patient, a journeyman tailor, aged forty-four, had suffered for thirteen months from loss of power in the little and ring fingers of the right hand, and for a shorter time from a similar affection of the corresponding fingers of the left hand. The muscles of the hands had undergone extreme wasting. He caught typhus in the hospital, and died of that disease. In the cervical and upper dorsal regions of the cord the place of the grey matter was taken by a large quadrilateral cavity, which was supposed to be a dilated part of the central canal. But a *hydromyelus* having sometimes been met with in persons who had been altogether free from spinal symptoms, the doubt might fairly be entertained whether its presence was not after all a mere accident. In children suffering from spina bifida, at any rate, such an affection is of common occurrence. Since the publication of Gull's case, however, similar ones, attended with like symptoms, have been met with by Schüppel and Hallopeau and Westphal; and it now seems impossible to explain them away. Th. Simon has shown that in reality the cavity is often altogether unconnected with the central canal, which lies in front of it and is no larger than natural. He proposes to apply to such cases, instead of hydromyelus, the name of *syringomyelus*, originally invented by Ollivier, but not with the object of conveying this distinction. I have already mentioned that the development of a solid new growth in the cord is often attended with the formation of a cyst in its interior (p. 467); but the cases now under observation do not seem to come under that head. Clinically, the progressive muscular atrophy due to syringomyelus does not appear to differ from the ordinary protopathic form of the disease.*

5. Lastly, deuteropathic progressive muscular atrophy may occur as a complication in cases of *locomotor ataxy;* and also in those of *multiple sclerosis*, if the morbid change should happen to involve the anterior cornua and to destroy extensively the multipolar cells. A *tumour* growing in the substance of the cord, or in the membranes, may sometimes be attended with symptoms of a similar kind. Erb remarks that it is not always possible to distinguish between a meningeal new growth and cervical pachymeningitis. I had in 1878 a case which illustrated this very point. A woman was admitted with atrophic paralysis of certain muscles in the left hand; and she also was liable to exceedingly violent paroxysms of pain in the right shoulder and arm, attended with sudden redness and swelling of the tender parts, evidently due to disturbance of the vaso-motor nerves. She had had iritis, and on the whole I inclined to the diagnosis of a syphilitic gumma, growing from the membranes and pressing on the cord in the cervical region. She improved under iodide of potassium before she left the hospital.

Diagnosis.—From considering how the various spinal lesions that may give rise to progressive muscular atrophy are to be distinguished we pass on to the diagnosis between this and other diseases attended with wasting of muscles.

First among them should perhaps be mentioned various forms of *peripheral paralysis* due to neuritis of the several branches of the brachial plexus, such as have been described at p. 429. There is no doubt that cases of this kind have often been mistaken for those of Cruveilhier's palsy; but a careful observer will generally recognise the limitation of the affection to the area of distribution of particular nerves; and all doubt will be removed by the

* See the remarks by Dr F. Taylor on his ten cases reported in the 'Pathological Transactions,' vols. xxix, p. 21, and xxxv, p. 36.

application of galvanic and faradic currents, which will reveal a far more marked perversion of the normal electrical reactions than is ever seen in progressive muscular atrophy.

The same tests will at once distinguish ordinary cases of *lead paralysis*, even when the history of the patient, the line on the gums, and the other poisonous effects of the metal are not of themselves sufficient to prevent any hesitation as to the diagnosis. For the peculiarities of R.D. are well marked; whereas in progressive muscular atrophy the muscles respond to faradism as well as to galvanism so long as there is any muscle left to contract; since the motor nerves, trophic cells, and muscle-fibres with their end plates all undergo very slow and equal atrophy, and healthy muscle is not left with damaged anterior cornua and degenerated nerves. I have seen a few instances in which, although it was known that the poison had been absorbed to some extent, opinions yet differed as to whether this fact would satisfactorily account for the symptoms, which seemed to be too severe, or too little amenable to treatment, to be caused by lead. Two such cases I published in the 'Practitioner,' under the name of progressive muscular atrophy, although with some hesitation. I am bound to say that now, on reading them over, I think that the view then taken was wrong: they were cases of plumbism.

The diagnosis from *chronic atrophic spinal paralysis* apart from lead (p. 503) rests on the fact that, in the latter, paralysis precedes atrophy, in progressive muscular atrophy it goes with it, and instead of R.D. occurring only in the "middle" qualilative form, or only towards the close of the disease, or not at all, it is present in its typical form as pronounced as in infantile palsy.

Another possible error is to mistake for progressive muscular atrophy the diffused wasting of the muscles, attended with great helplessness and loss of power which is a result of protracted *osteo-arthritis*. The hollowing of the interosseous spaces and of the ball of the thumb may be very striking in cases of this kind.*

The *prognosis* of progressive muscular atrophy is a very grave one. Its tendency is slowly but surely to spread from one muscle to another until those of respiration are involved. Occasionally its course is arrested without apparent cause, and the atrophied muscles remain like those of a withered limb after infantile palsy.

Treatment.—It only remains for us to consider to what extent cure is possible, and I think that the universal experience has been that drugs are altogether useless. Neither arsenic, nitrate of silver, phosphorus, nor iodide of potassium can restore the wasted muscles, or prevent further extension of the morbid process.

There is a large amount of testimony to the value of electricity. By Duchenne faradisation of the affected parts was strongly recommended. He advised the application of currents of moderate intensity, with not too frequent intermissions, and for a few minutes only at a time, so as not to fatigue the fibres remaining undestroyed; he particularly insisted on the importance of including in the treatment any important muscles, such as the diaphragm, the intercostals, and the deltoids when they are first threatened

* French writers describe, under the name of *marasme essentiel*, an affection which they say occurs in hypochondriacal patients, who gradually assume the appearance of living skeletons. It corresponds with the remarkable case of emaciation in a man who was cured by Weir Mitchell's massage treatment, published by Drs Playfair and Brunton.

by the disease, and before it actually invades them. In the case of a man named Bonnard, who had lost many muscles of his trunk, and who was beginning to suffer from dyspnœa, so that he could scarcely walk a few steps without stopping to take breath, faradisation of the phrenic nerves, repeated three or four times a week, was of great service, enabling him to walk considerable distances and to go upstairs without fatigue. A similar treatment, applied to certain muscles of the arms which were wasted, restored their functions, so that at the end of six months he was again able to support his family by his exertions. And he went on for some years without the disease advancing further.

On the other hand, Remak advocated the use of the galvanic current. His method was to place the positive pole in front of one mastoid process, and the negative pole on the opposite side of the neck, near the spinous processes of the vertebræ, not higher than the fifth cervical. This, he found, would often produce contractions, which he termed diplegic, in the fingers or other paralysed parts.

All observers admit that in not a few cases each of these plans of treatment fails utterly. They should, however, always be tried in succession, or at the same time. And it is worth while to persevere with them, even when they seem at first to be doing no good, because it has sometimes happened after several months that favourable results have been attained.

At the commencement of the disease the affected parts should be rested as much as possible. In one of my cases, the forearms were wrapped in cotton-wool, and placed in splints, and after a week the patient was found to have much more power in his hands. But in the more advanced stages, methodical exercise of the wasted muscles appears to be sometimes useful.

Dr Ross agrees that all drugs are useless, and that galvanism is the most efficient remedy for progressive muscular atrophy. But Dr Gowers finds that "the most sedulous and skilful use of electricity, voltaic or faradic, fails, as a rule, to produce any effect on the course of the disease." He is equally incredulous of the asserted benefits of *massage*, which are "usually inappreciable," and of *balneotherapie*. Even when the disease follows syphilis he finds mercury useless. "The disease is one in which it is not easy to do good and not difficult to do harm."

Progressive Bulbar Paralysis.*—In 1860, Duchenne gave a clinical description of a form of progressive paralysis of the tongue, palate, and lips, which had previously received no systematic recognition from writers in medicine, although Trousseau, as far back as 1841, had noted the peculiar symptoms presented by a well-marked case occurring in the Prince de M. The disease has now become well known in England, and is often termed labio-glosso-laryngeal paralysis, after Trousseau and Duchenne; but a shorter and better name is that of "progressive bulbar paralysis," which was originally suggested by Wachsmuth in 1864. As Kussmaul has remarked, it might with still greater accuracy be called "progressive paralysis of the bulbar nuclei;" for the morbid change does not involve that part of the bulb (*i. e.* medulla oblongata) which transmits the motor and sensory strands for the limbs and the body generally, and is confined to the grey centres for certain of the cranial nerves on the floor of the fourth ventricle.

* *Synonyms.*—Labio-glosso-laryngeal paralysis—Progressive muscular atrophy of the muscles of the mouth and throat.

Course and symptoms.—As a rule, bulbar paralysis begins very insidiously. The earliest symptoms are commonly subjective,—a feeling of pressure, or even of pain, at the back of the neck and head, a little giddiness, a sense of constriction round the throat or chest, a slight discomfort in talking, as if the tongue were heavy and its movements laboured, or a tired feeling after speaking for some time without intermission. Krishaber has stated that in two cases he discovered a loss of reflex irritability in the pharynx and larynx some months before any signs of paralysis made their appearance. Sometimes the palate is affected before the tongue, or the disease begins in the lips; in the former case the speech acquires a "nasal" quality, and swallowing seems to require an unusual effort; in the latter the expression of the face about the mouth becomes altered, or there may be a little difficulty in the utterance of certain letters, as though (to use a phrase of Duchenne's) the lips were half paralysed by cold.

Sometimes, however, the commencement of the disease appears to be sudden. Kussmaul, in one of the clinical lectures published by Volkmann, relates that a patient of his, a Catholic priest, found one day, while preaching, that his mouth was distorted and that he had a difficulty in speaking. He was able to finish his sermon, but from that time there remained a heaviness of the tongue. For a week previously he had suffered from pains in the back, but he had had no giddiness. Dysphagia soon set in, and within six months the case became one of confirmed bulbar paralysis, with loss of power in the arms and wasting of the small muscles of the hands. It was ascertained at the autopsy that there was no hæmorrhage into the pons or the medulla oblongata; and this, as well as the progressive character of the complaint, distinguishes it from certain cases of Wilks, of hæmorrhage in the bulb producing symptoms of paralysis, to which I shall presently refer (p. 522). In a woman whose case is recorded by Leyden the first symptom was a sudden attack of dyspnœa, lasting five minutes; a few days afterwards she naticed a difficulty in moving the tongue when she spoke or ate.

In fully-developed cases, the tongue is generally the part in which the loss of power is most obvious. It lies flaccid in the floor of the mouth; the patient can neither bend it laterally, nor raise it against the palate, nor hollow its centre; he may even be unable to protrude it beyond the teeth. Scarcely less marked is the paralysis of the lips. The mouth remains open, and with its angles drawn wide apart; the naso-labial furrows are deepened, the lower lip hangs away from the gum. The patient cannot whistle or blow out a candle, or kiss. What is most remarkable is that although fascial muscles supplied by the lower branches of the *portio dura* are thus affected on both sides in every case, those to which the upper branches of the same nerve are distributed as constantly escape. The orbicularis palpebrarum and the occipito-frontalis act as well as ever. The countenance thus acquires a curious expression: the expression about the eyes is full of life, that of the mouth is fixed, sad, and gloomy.

On the other hand, the paralysis of the palate is not indicated by any obvious change in its form. The uvula hangs in its ordinary place; and Duchenne says that he always found that irritation of it caused the usual reflex movements. With the laryngoscope the vocal cords may in advanced cases be seen to be more or less completely paralysed.

Several important functions are impaired in ways which are somewhat complicated, and demand careful study. Thus the defect of speech appears

to vary with the part which becomes earliest paralysed. According to Kussmaul, if the lips suffer first, O and U are the vowels which the patient experiences most difficulty in uttering; if the tongue, I is sooner lost. A is always retained longer than any of the others.* Among consonants, loss of power in the tongue renders the patient first unable to utter R, and SH; next S, L, K, G, T; afterwards D and N. Paralysis of the lips prevents the formation of P and F, then of B and M, ultimately of V. Paralysis of the palate gives to the speech a nasal twang, and it specially prevents the formation of the lip-sounds B and P, because it allows so much of the air to escape through the nose; the proof of this being, as Duchenne pointed out, that closing the nostrils may enable these letters to be sounded. So far, the affection of speech is one which merely concerns articulation, and may, as has been proposed, be called "alalia" or "anarthria," in distinction from the "aphasia" that depends upon lesions in the left side of the brain, and the "aphonia" that is caused by loss of power in the larynx. But in bulbar paralysis, after a time, the vocal cords themselves lose their functions; the voice then becomes altogether extinguished, and the patient is able to utter nothing beyond a meaningless grunt.

Another set of movements which are interfered with in progressive bulbar paralysis are those which are concerned in the reception of food into the mouth, and its transmission backwards into the œsophagus. During mastication it collects inside the cheek, not only when the buccinator is paralysed, but (even apart from this) because the tongue cannot properly dislodge it. Very often the patient helps himself with the fingers of both hands, supporting the floor of the mouth or the cheeks, or pushing the half-chewed food into the proper position. Then, again, the tongue cannot roll up the softened pulpy material into morsels for swallowing, nor carry them into the pharynx. Loose fragments are constantly dropping out of the open mouth into the plate or upon the patient's clothes. Other pieces collect about the root of the tongue, or in the grooves by the side of the epiglottis. The attempts to swallow them succeed very imperfectly—some pass up into the pharynx or through the nose, others enter the larynx and set up a choking cough; they may even accumulate in the fauces to such an extent as to interfere with the passage of air and to produce sudden death. It depends upon circumstances whether the patient finds most difficulty in dealing with solids or with liquids. If the principal defect is a weakness in the tongue and in the muscles of mastication, he requires to have all his food reduced to a semi-fluid state; but when there is paralysis of the parts concerned in closing the larynx during deglutition, he can often dispose of solid masses better than of liquids, being unable to prevent the latter from trickling down into the air-passages. The inability to swallow gives to the disease another peculiarity in the circumstance that the saliva keeps running out of the mouth; such patients keep a handkerchief constantly held below the chin, and, as Wilks remarks, this often at the first glance enables one to guess what is the matter with them. The secretion itself is sometimes viscid, sometimes watery. It seems so abundant as naturally to suggest

* These vowels must be pronounced in the Italian way: U=OO (in *fool*), I=E (in *feel*), A=Ah (in *far*). To complete the list of the chief sounds which occur in English, those represented by the letters Th would have to be added; probably they are among the first to disappear in cases in which the tongue is affected. I may remark that Kussmaul's statements correspond closely with what might have been anticipated from theoretical considerations, of which an admirable account will be found in a paper by Dr Bristowe in the first volume of the 'St Thomas's Hospital Reports.'

that it is formed in excess, and in one instance Schultz is said to have estimated that there was six or eight times as much of it as would have been poured out under normal conditions. Kussmaul, however, found no such increase in a case in which he determined its quantity. Sometimes the masticatory muscles become at length involved in the paralysis; the patient is then unable to move the lower jaw from side to side, nor can he close the mouth firmly.

Generally the respiration is not obviously affected at an early stage of the disease. Later on dyspnœa often becomes a marked symptom, and on stripping the patient one may find that the respiratory movements of the chest walls are very shallow. In some cases, however, distress of breathing and a constant craving for air are said to have been present, although the diaphragm and the thoracic muscles were still vigorous. Kussmaul cites the written statement of a woman under his care who was in great dread of gaping, because "she was obliged to groan and strain in order to get rid of the air and then breathe more freely." He follows Duchenne in attributing such symptoms to paralysis of the bronchial muscles. There is generally an inability to sneeze, to cough, to hawk up phlegm, and to blow the nose; the patient sometimes complains bitterly that he is no longer able to smoke. Towards the last, paroxysms often occur, attended with a rapid pulse; these are supposed to depend upon paralysis of the vagi.

Among the symptoms which have been noticed in some exceptional cases may be mentioned slight deafness, noises in the ears, ptosis, paralytic affections of the ocular muscles, numbness and anæsthesia of the face or tongue. Physiological considerations have led some observers to search for sugar and for albumen in the urine; but I believe that the results have always been negative.

The higher cerebral functions remain undisturbed, the patient sleesp well, his intelligence and memory are perfect. The movements of the body and limbs are generally free and active, except towards the last, when emaciation from want of food often causes extreme weakness.

Pathology.—Although, as just stated, the great motor paths through the bulb and the pons escape, it nevertheless frequently happens that there is associated with bulbar paralysis an affection of the upper (or more rarely of the lower) limbs, which agrees in all respects with what has already been described as Progressive Muscular Atrophy. Sometimes one, sometimes the other of these two diseases is the first to develop itself. The fact that this connection exists is of considerable importance, for Duchenne drew a sharp distinction between them. In labio-glosso-laryngeal paralysis, he said there was paralysis without atrophy; in progressive muscular atrophy there was atrophy without paralysis, and certain cases in which he found the tongue wasted were regarded by him as examples of a separate malady, a true progressive muscular atrophy involving that organ. But as I have already observed, the tendency of recent investigations into "Cruveilhier's palsy" has been to show that this disease is a true spinal paralysis. Moreover, there seems to be no satisfactory proof that, after death from progressive bulbar paralysis, the muscles have ever been examined microscopically and found to be all of them in a healthy state. And, inasmuch as Charcot once discovered a tolerably advanced degenerative atrophy in a tongue which during life had appeared smooth and of natural size, it is clear that nothing but pathological evidence can be conclusive. In many cases the fleshy substance of the tongue, and even that of the lips and palate, is

obviously pale, of a yellow or greyish-red colour; and it is streaked with fat or more or less completely converted into a mass of fatty connective tissue.

Thus at the present time the opinion of all the most competent authorities is that progressive bulbar paralysis is pathologically identical with progressive muscular atrophy, differing merely in the circumstance that it affects a particular set of nerve-nuclei in the bulb instead of those in the cord. It is admitted that in the earlier stages the tongue is often not obviously reduced in size, but in advanced cases it is described as being soft, small, wrinkled on the surface, and incessantly agitated by a fibrillary tremor. The lips also become thin and sharp edged; their muscular substance quivers, and the skin over them is marked with minute furrows. In the palate, as might be anticipated, wasting of the muscles is not discoverable, at least during life. It is allowed that there is no absolute correspondence between the degree of the paralysis and that of the atrophy.

The results which have been attained by testing the affected muscles with electricity appear to accord with these statements. The earlier investigators described the faradic contractility as normal; but several German observers have since found it lowered or even extinguished; and with galvanic currents, Erb (as well as Kussmaul) has recently detected the "reaction of degeneration" in a perfectly characteristic form. The mistake seems to have arisen from the stimulus having been applied to the motor nerves rather than to the muscles themselves. Lastly, atrophy of the motor cells which form the nuclei of the hypoglossal and other motor nerves in the bulb has now been repeatedly observed after death from bulbar paralysis; and this agrees histologically with the lesions of the anterior cornua seen in Cruveilhier's palsy. The bulb is the *medulla spinalis oblongata*, and the grey matter of the floor of the fourth ventricle is formed by the anterior cornua laid open and separated, so that the seat of each disease is serially homologous.

Histology.—Progressive bulbar paralysis is one of those diseases of which it has been possible to work out the morbid anatomy satisfactorily only within the last few years, since we have been in possession of the new methods of investigating the nervous tissues. Kussmaul cites eight cases, in each of which a complete microscopical examination was made. In general no marked change is seen by the naked eye in the fresh medulla oblongata; but it is said that sometimes a little want of symmetry in the two halves of the floor of the fourth ventricle has been detected, or a slight shrinking; or that this part has appeared discoloured and reddish grey; or that its texture, when cut into, has looked blurred; or that its consistency has been greater or less than natural. One thing, however, is very obvious, even to the naked eye, namely, an extreme degree of atrophy of the nerve-roots arising from this part of the cerebro-spinal axis. It is especially conspicuous in the hypoglossal and the facial nerves; but it is generally plainly visible also in the three divisions of the eighth nerve on each side, and sometimes in the sixth, and in the motor portion of the fifth. The roots in question are grey and transparent, and exceedingly reduced in size.

In stained sections of the hardened medulla oblongata, the multipolar cells in certain nuclei are uniformly found to have undergone degenerative changes. Their colour is darker than natural, being deep yellow or yellowish brown; they are often shrunken; their prolongations may be indistinct or may have quite disappeared. They seem to be reduced in number. In one case Charcot and Joffroy are said to have come to the conclusion

that the hypoglossal nucleus contained only one tenth or one twelfth part of the cells which would have been present in it in a healthy bulb. French histologists appear not to have recognised any marked change in the neuroglia, but in Germany both Leyden and Maier have found it increased in quantity and filamentous, containing stellate cells; in other words, presenting appearances like those which characterise myelitis going on to sclerosis. The nerve-fibres undergo atrophy. The structures in which these parts are most constantly seen are the nuclei of the hypoglossal, the vagus, and the facial nerves. As regards the last-mentioned nerve, indeed, the symptoms of bulbar paralysis point to the conclusion that only a part of its nucleus is affected; and this accords well with Lockhart Clarke's statement that its roots arise in two separate masses of grey matter; but it does not appear that the obvious pathological application of his observations has been yet traced out in detail. The nucleus of the glosso-pharyngeal nerve escaped the morbid process in a case recorded by Duchenne and Joffroy. The sensory nucleus of the fifth and that of the auditory nerve have been constantly found intact. The olivary bodies have sometimes presented degenerative changes, but more often they have been in a normal state.

Ætiology.—With regard to the causes of bulbar paralysis nothing is certainly known. It scarcely ever occurs in persons under thirty, and appears absolutely to increase in frequency as age advances up to the seventieth year. It is rather more common in men than in women.

Erb speaks confidently of its being sometimes caused by cold, and Kussmaul relates a case in which the patient traced it to a cold caught while he was at work in the fields; he was attacked with headache and pain in the neck, and a week later he was conscious of difficulty in swallowing and in speaking. Among other conditions which have been supposed to give rise to it are excessive smoking, over-exertion in playing wind-instruments, syphilis, and falls producing concussion of the medulla oblongata. In one of Trousseau's cases it began during convalescence from a febrile attack.

Diagnosis.—This at the commencement of the disease requires considerable acumen; one might easily make light of the early symptoms, and so justly forfeit the confidence of the patient. Even when they are fully developed a hasty conclusion is dangerous. It is to be remembered that the lips and the tongue may be paralysed by various affections, beside that which constitutes the disease described by Duchenne. What characterises them is the presence of additional symptoms which do not belong to it.

Most of the cases that have been recorded as occurring in young persons have presented peculiar features, and their pathology must be regarded as still undetermined (p. 524).

Wilks has pointed out that a condition precisely like that which belongs to progressive bulbar paralysis may be suddenly developed as the result of a circumscribed effusion of blood into the lower part of the pons. He relates in the 'Guy's Hospital Reports' two cases, in one of which an old brownish cyst was found at that spot after death. So also, tumours growing near the bulb may cause similar symptoms. The same author has seen the disease simulated by hysteria; and other observers have recorded cases due to syphilis which have been cured by iodide of potassium. All such cases must, for the present, be regarded as distinct.

Prognosis.—It seems probable that all of the few recorded instances in which recovery has taken place from "bulbar paralysis" have been essentially different from the disease now under observation. Such, for

example, is a remarkable case, observed by Dr Dowse, of a young man who, after a series of epileptic seizures, was affected with paralysis of the whole body, with the exception of the muscles of the eyeballs. At the end of four years he had improved greatly, but the tongue still lay immoveable in the floor of the mouth; he was quite dumb, and had difficulty in deglutition. He was under observation for nearly twelve months, and was making no further progress when a few applications of a continuous current to the tongue restored the power of articulation, and at length his cure was complete.

Genuine cases which depend upon a progressive change in the bulbar nuclei appear always to end fatally. This was Trousseau's verdict, and all later experience confirms it. Their duration is generally from one to three years; but it may be as long as five years. Sometimes the patient is choked unexpectedly by a mass of food which cannot be propelled beyond the entrance into the larynx; of this an instance occurred at Guy's Hospital in 1865. Sometimes death is brought about by an attack of syncope, sometimes by a paroxysm of dyspnœa. Kussmaul remarks that such seizures are particularly apt to happen after exertion of some kind, but that they not infrequently occur at night when the patient is in bed. In other cases exhaustion and emaciation gradually lead to a fatal termination; or pulmonary phthisis may develop itself; or acute pneumonia, consequent upon the admission of food into the air-passages during the act of deglutition.

Treatment.—I do not think I have seen benefit result from any therapeutic measures. Kussmaul recommends dry cupping at the nape of the neck in early cases, and the use of shower baths. Among drugs he thinks the nitrate of silver most likely to be serviceable. He has seen transitory benefit from faradisation of the palate and tongue; and he mentions two cases in which striking results were for a time attained by the use of strong galvanic currents passed through the neck and spine. One patient, a priest, who had been unable to get his tongue out between his teeth, could, after being galvanised, protrude it beyond his lips; his speech became more distinct and his deglutition easier. After four or five weeks, however, the improvement ceased. Specially applicable to progressive bulbar paralysis is, perhaps, Schulze's method of inducing the act of deglutition by galvanism. It consists in fixing the positive pole upon the nape of the neck, and then rapidly moving the negative pole downwards over the side of the larynx. This procedure may be repeated at short intervals several times during four or five minutes. In the first instance a current from six to eight cells should be tried; but to produce the desired result a large number of elements are often required.

When the patient cannot swallow food at all, or seems likely to be choked in the attempt, a tube must be passed into the stomach; but this procedure often has to be abandoned on account of the irritation excited by it. Nutrient enemata then afford the only means of sustaining life for a time, unless recourse is had to gastrostomy. In one case the trachea was opened when danger was apprehended from frequent attacks of dyspnœa; and the result was that they ceased, although of course the other symptoms remained.

Acute Bulbar Paralysis.—This appears to be the most convenient place for a brief notice of an affection which on clinical grounds requires a separate description, although to the pathologist it is merely the representative

of a circumscribed "focal" myelitis, which happens to be seated in the bulb instead of in the spinal cord. Three examples of it are cited by Erb, all of which were observed by Leyden. In one case a patch of softening with numerous capillary extravasations of blood was plainly visible in the pyramids and in the olivary bodies at the autopsy; in the others no lesion was discovered until the parts had been hardened, when the microscope revealed inflammatory changes of the most marked kind. Each patient had been taken ill rather suddenly, and had died in from four to ten days. The symptoms varied considerably, being of course dependent upon the exact seat of the morbid process. Chief among those which were present in one case or another were headache, giddiness, vomiting or severe hiccup, difficulty of deglutition, or even a complete inability to swallow, a more or less profound impairment of speech, irregularity or great rapidity, or occasional interruption of the breathing, a quick and feeble and irregular intermittent pulse, a partial or complete paralysis of the tongue, of some or all of the limbs, and perhaps of the face, formication and pains in the limbs. There was no failure of consciousness, nothing at all resembling an apoplectic seizure. Depression and collapse quickly set in, and the fatal issue was brought about by paralysis of the respiratory muscles, with distress of breathing and lividity.

As may be supposed, the diagnosis of the seat of the disease was not difficult; but it was not easy to exclude the possibility of embolism of the basilar artery or of the vertebral arteries, or their branches, or of a minute spot of hæmorrhage, or even of a rapidly-developed compression of the medulla oblongata by some disease in its neighbourhood. The most important consideration of all is that perhaps such cases need not invariably terminate fatally, since ordinary myelitis is not infrequently recovered from.

A case which seems to have been of this kind occurred in Guy's Hospital in 1874, in the person of a man aged forty-five. It proved fatal in about ten days. The central part of the pons appeared softened; but after preparation in chromic acid no decided morbid changes were made out.

Progressive Muscular Atrophy of Youth.—*a. The infantile form of Duchenne.*—According to Duchenne, progressive muscular atrophy is not so rare as is supposed in children, and when it occurs it presents certain peculiarities in its symptoms and course. It begins in the lips, which become thick and hanging, and cannot be brought together. If the child smiles, the angles of the mouth are drawn far apart, and the cheeks are flattened by the action of the buccinator muscles. The articulation of labial letters and of the vowel *o* is impaired. It is almost always between the fifth and the seventh year that this form of the affection first appears. But at that time it often attracts very little notice from the parents, although the reality of the morbid change is said to be at once made apparent if the orbicularis oris and the other muscles are tested by faradic currents, when they are found to have lost their contractility. About the eleventh or twelfth year, however, the muscles of the shoulders and arms begin to waste capriciously and irregularly; and it is now that medical advice is first sought. Later still the muscles of the trunk and those of the lower limbs are attacked in their turn. I have no recollection of having ever seen a case of this kind; but Duchenne, whose first cases were recorded in 1855, said in 1872 that he had observed no fewer than twenty. One thing which would rather tend to show that the progressive muscular atrophy of childhood has no very close

relation to the most common forms of wasting paralysis in adults is that it very often occurred in two or more brothers and sisters; but a curious circumstance is that the father of the first two children in whom Duchenne recognised its peculiar characters was afterwards, at the age of forty-eight, attacked with typical "Cruveilhier's paralysis," affecting first the shoulders and arms, but ultimately the lower limbs; he also stated that his father had died of a similar complaint. Whether this was anything more than an accidental coincidence it is difficult to say.

Duchenne's description was written in 1855, but no one appears to have observed similar cases until Landouzy and Déjerine published several in 1874 and one, with an autopsy, in 1885. Westphal, E. Remak, and other writers in France and Germany, have published a few cases since. The cord and bulb appear to be unaffected, and the seat of the disease to be the nerves, so that it would appear to be pathologically a form of Peripheral Neuritis affecting the facial and other motor nerves.

β. *The juvenile hereditary form.*—A clinically distinct "juvenile form" of progressive muscular atrophy has been described by Erb ('Deutsches Archiv f. klin. Med.,' 1884). It begins usually at puberty. The muscles of the shoulder are more affected than those of the hands, and the legs are not unfrequently attacked. The cases observed are also marked by a hereditary character. There are no fibrillary contractions, no R.D. even of the "middle" or imperfect kind, and *post mortem* the anterior cornua have been found intact in two cases by Friedreich.

Dr Tooth has recently (1886) collected the previously published cases of this form of paralysis, and added four new ones. Of 30 in all, he finds that 8 occurred between one and five years of age, 10 between five and ten, and 18 between ten and twenty, while only 3 patients were older—twenty-five, thirty-seven, and forty-six; 28 were males and 16 females, so that the disproportion was much less than in typical muscular atrophy. Grouped in families, there were 17 instances of more than one case in a family, 12 of the disease being isolated. The lower extremities were first affected, and particularly the peroneal muscles. The extensors and gastrocnemii shared in the paralysis, and the hands and forearms followed. Fibrillary tremors and marked R.D. were usually present.

It appears from the clinical features, as well as from the anatomical results of Friedreich's two cases, that the lesion is not cerebral, but a Peripheral Neuritis. In Virchow's case, this also was found along with sclerosis of the posterior cornua and root-zones.

Dr Meryon's remarkable cases of hereditary muscular atrophy, described in 1852, may not improbably belong to this same clinical group. They have, however, been generally appropriated as examples of the following disease, and will be again referred to (p. 528).

It is probable that progressive paralysis of the muscles of the orbit, described as Ophthalmoplegia Externa at p. 412, is, like Bulbar Paralysis, a local form of the same pathological process as that which causes myelopathic progressive muscular atrophy.

Pseudo-Hypertrophic Paralysis.*—In 1861, Duchenne recorded in the second edition of his 'Electrisation Localisée' the case of a boy who, with legs so weak that he could scarcely walk or even stand, had the

* *Synonyms.*—Myopathic atrophy with fatty overgrowth—Myosclerosis—Progressive muscular sclerosis—Lipomatosis musculorum luxurians.

muscles of his calves and of his hips as large as those of an athlete. Seven years later the same fertile author wrote a detailed paper on the disease in question, to which further investigation has added little. In the meantime cases had been noticed by a few German observers. I saw one in 1863 in Oppolzer's wards at Vienna; and another, which came under Griesinger's notice in 1864, gave Billroth the opportunity of excising a piece of the deltoid muscle and of proving that there was no real but only an apparent hypertrophy; it was found to consist almost entirely of adipose and fibrous tissue, separating from one another the muscular fibres, which themselves were unaltered. Duchenne, in 1865, made similar observations in one of his cases with the aid of his "emporte-pièce." He therefore proposed to term the disease *paralysie myosclérosique*. Dr Gowers' monograph published in 1879 contains all the information obtained up to that date.

Histology.—The substance of the enlarged muscles has a whitish-yellow colour, with perhaps a faint reddish tint. There is still a linear arrangement of the fibres, and in extreme cases this may be the only thing which distinguishes it from the subcutaneous adipose tissue. The fat sometimes extends into the tendons, giving them during life an appearance of having been encroached upon by the fleshy bellies of the muscles. On the other hand, there has occasionally been found no fat, but fibrous tissue only, between the muscular fibres; and in the earlier stages of the disease, this new tissue has been found full of nuclei or spindle-cells. The muscular fibres seem to become greatly reduced in number by a process of simple atrophy, which at last leaves only the collapsed sarcolemma-sheaths. Those fibres which remain are not always completely unaltered, as described by Griesinger. Duchenne speaks of their transverse striation as unusually faint. Other observers have seen some which were striated longitudinally, some which were translucent or waxy, and some which were in a state of granular or fatty degeneration. Some fibres again are truly hypertrophied, having two or three times their normal thickness, as was first observed by Cohnheim.

Course.—The enlargement of the muscles is not present at all stages of the affection. There is an early period during which the only symptom is an impairment of power in the lower limbs. The child—for pseudo-hypertrophic paralysis almost always begins in childhood—is noticed to totter in walking, and to be apt to fall; he has difficulty in getting on to his feet, he is particularly awkward in going upstairs; and when he tries to sit down he falls into the chair. He may previously have been able to run about like other children. But in many cases the commencement of the disease occurs before the little patient has learned to walk; the proper age is passed without this being accomplished. At first the parents think that the child is only backward, as so many rachitic children are, but at length they see that there is something more seriously wrong.

Even when, at the end of a few months or a year from the beginning of the paralysis, the change in the muscles is discoverable, its extent and degree vary widely in different cases. The calves are often affected alone; next in liability to undergo enlargement are the masses of the *erector spinæ* in the loins, the *glutæi*, the *deltoids*, and the *infra spinati*. The glutæus medius usually escapes. The muscles of the thigh, the *latissimus dorsi*, the lower part of the *pectoralis* and *serratus magnus* are more often reduced in size, so as to afford a strange contrast. But sometimes the whole of the trunk and all parts of the limbs display an exaggeration of contour which leaves far

behind that of the Farnese Hercules, as Duchenne shows by comparing a sketch of that ancient statue with drawings of a patient of his, a boy ten years old. The enlarged muscles are generally firm and elastic, and when they are brought into action they harden so that it is difficult to believe that they do not consist wholly of contractile elements. Leyden, indeed, speaks of them as doughy, and even as feeling semifluid when they contract; but I do not think this is the case in the majority of instances.

When a child affected with pseudo-hypertrophic paralysis stands up, its attitude is very peculiar. The abdomen is pushed forwards, and the hollow of the lumbar vertebræ is greatly exaggerated. The nates project behind; but the shoulders are thrown further backwards still, so that a line carried downwards from the upper dorsal spines falls behind the sacrum. The legs are separated widely from one another. If an attempt is made to straighten the back the child at once falls down. When he walks, he balances the body from side to side at every step, with a "waddling" gait. He cannot rise from the sitting posture without the use of his hands, and the way in which he uses them is very characteristic. He places both hands upon his knees, and gradually pushes up his body into the erect posture by moving his hands from his knees to his groin. This manœuvre, which has been not inaptly called "climbing up the thighs," is well represented by sketches in Dr Gowers' work on 'Diseases of the Cord' (p. 391), and by photographs in Dr Ross's work (vol. i, p. 996).

The susceptibility of the affected muscles to faradic currents is sometimes normal, but in other cases it is considerably lowered. This appears to depend solely on the degree of atrophy of muscular fibres. The R.D. is absent. As with all the atrophic disorders of muscles described in this chapter, pelvic symptoms are absent, and the sphincters act naturally throughout the whole course of the disease. The knee-jerk gradually disappears as in other atrophic paralyses. Fibrillary tremors, such as are seen in progressive muscular atrophy, are not generally to be noticed; but they have been observed in a few instances. The cutaneous sensibility is altogether unimpaired. The legs and feet are often cold and damp and bluish. The heels are usually drawn up to a greater or less extent, by contraction of the *tendo Achillis* on each side; and there may be a well-marked club-foot. Sometimes the knees are rigidly flexed. The intelligence may be perfect; but it is not rarely very defective, and in some of the cases recorded the patients have been idiots.

When the disease has developed itself to a certain point it is described by Duchenne as remaining stationary for two or three years, or even longer. But at length a further advance takes place. If the legs only were affected, the arms are now involved. The muscles, however, which at this time lose their functions, never show even an apparent hypertrophy, but are always reduced in size, although the histological processes in them are of exactly the same kind as in those which were earlier affected. Even those muscles which were at first enlarged ultimately shrink, until they too are obviously atrophied. The patient, who has generally now reached adolescence, becomes altogether unable to stand, or even to sit up; he is a prisoner upon the couch or the bed. Ultimately he dies of exhaustion, or is carried off by phthisis or some other intercurrent complaint. Friedreich, however, remarks that a large majority of the cases hitherto recorded have been lost sight of, so that the end of them is not known.

Ætiology.—Very little has been learnt as to the cause of pseudo-hyper-

trophic paralysis. The most conspicuous point in its history is its tendency to appear in succession in two, or three, or even four children of the same family, especially in the boys, and at the same age in all of them. As might be expected, the parents themselves have always been free from it; but its hereditary origin has often been traceable by its having occurred in brothers or other relations of the father or mother. Transmission is almost always through mothers to sons.* Males are altogether far more subject to it than females, the proportion, among seventy-seven cases collected by Friedreich, being sixty-four to thirteen. Of Duchenne's original fifteen cases only two were girls, of forty-one collected by Webber of Boston, only five (quoted by Ross), of thirty-three by Gowers ten. In a solitary series of cases recorded by Lutz it appeared in two successive generations in the female line only, affecting five individuals. On the other hand, it has often attacked several sons of the same parents, and spared all the daughters.

Out of 75 cases in which the date of its commencement was ascertained, Friedreich found 45 in which the children were under five years of age, 17 between six and ten, 8 between eleven and sixteen.† In the remaining 5 cases the patients were adults; and 2 of them are said to have been aged forty and forty-one respectively when the disease began. As occasional exciting causes are mentioned unhealthy conditions of life, chills, over-exertion of the muscles, and the occurrence of convulsions or of an exanthem such as measles.

Diagnosis.—This is very easy in well-marked cases. One morbid state, which must be thought of in connection with it, is that which Brodie and Hilton both believed to depend on the hip-joints being congenitally placed too far backwards; the drawings given in Hilton's work show a compensatory curvature of the spine very like that which is seen in pseudo-hypertrophic paralysis, so that one is inclined to wonder whether his cases could possibly, after all, have been examples of that disease in a slight form. Dr Leech's "harpoon" enables one to examine the muscular tissue during life, and is said to be more useful than Duchenne's. That enlargement of the muscles is sometimes not a conspicuous symptom is, I think, certain; unless it should prove that Duchenne and Friedreich were wrong in including under the present head the cases described by Dr Meryon in 1852 under the name of "granular and fatty degeneration of the voluntary muscles," which occurred at about the right age in several boys belonging to two families. Meryon found a different morbid change in the muscles; but it must be remembered that his investigations were made after death had occurred at a very advanced period of the disease, and not upon portions of muscle removed during life. Charcot and William Roberts, however, regard the cases in question as examples of progressive muscular atrophy. And it is to be noted that Friedreich himself relates as instances of the latter disease several cases which seem to belong to the same class as Meryon's (cf. p. 525). They occurred in three families residing in or near Heidelberg; and what is very curious is that Hemptenbacher, a pupil of Friedreich's, succeeded in tracing all three families to a single pair of ancestors a century and a half back. A similar complaint is said by Eichhorst to have appeared in six successive generations in a family at Königsberg.

Pathology.—Charcot and most other writers at the present time hold

* In this character of chiefly affecting males but being chiefly transmitted through females, this curious disease resembles Hæmophilia.

† There is now in Guy's Hospital under Dr F. Taylor, a youth of 18, in whom this disease has developed within the last few months only.

that this disease is primarily an affection of the muscles themselves, and thus that it differs absolutely from progressive muscular atrophy, which has been traced to a lesion in the grey matter of the cord. Now, we have seen that in progressive muscular atrophy, and even in the atrophic paralysis of children, the wasted muscles often have their bulk made up to the natural standard by an interstitial development of adipose and fibrous tissues. If, on the other hand, an apparent overgrowth of the muscles is not an essential feature of pseudo-hypertrophic paralysis, one cannot help suspecting that its relation to the other diseases must, after all, be very intimate. Indeed, although Pierret and Charcot studied with great care the histology of the cord in a fatal case of M. Bergeron's, and detected no morbid change in it, Dr Lockhart Clarke and Dr Gowers have since recorded a case in which they discovered extensive areas of disintegration in various parts, in some of which the nerve-cells were in a state of atrophy. Further observations are very much needed; but I strongly incline to the opinion that pseudo-hypertrophic paralysis will ultimately be found to be a spinal affection.

Since the last sentence was written in the first edition of this work, Dr Ross and Dr Bramwell have each found changes in the cord. The former observer, however, has in a second case found the cord and nerves of the paralysed muscles unaffected, and the lesions recorded by the latter were either congenital or secondary and recent. Accordingly Dr Ross has, in the second edition of his work, abandoned the theory of a spinal origin, and come to the conclusion that pseudo-hypertrophic paralysis is a primary and idiopathic atrophy of the muscles. Dr Gowers also holds that "the essential lesion is the growth of connective tissue by which the muscular fibres are damaged, whether fatty tissue is found or not."

Treatment.—There is unfortunately very little to be said on this point. Duchenne believed that he cured two cases (the details of which are given in the 'Archives Générales' for 1868) by faradisation of the affected muscles, with the aid of hydrotherapeutics and massage; they were both in an early stage, with but slight enlargement of the gastrocnemii. At a later period of the disease he found treatment of no use. Benedikt has recorded three cases in which he obtained results by "galvanising the sympathetic." But in the hands of Erb this procedure has since signally failed. Meryon considered that the administration of arsenic retarded the progress of his cases. When contractures have occurred, so as to produce talipes, division of the tendo Achillis has sometimes been useful, by enabling the patient to walk until the muscular atrophy has gone further still.

Primary multiple muscular atrophy is a condition closely allied to the preceding, but differing in the fact that there is no stage of apparent hypertrophy. Such cases will be found mentioned in Dr Poore's edition of Duchenne's great work, and in the treatises of Leyden and of Gowers. They occur in families, and sometimes reappear for several generations. Thus Barsickow, in a thesis on the subject, quoted by Gowers, found twenty-four cases in five generations. The affection is not much, if at all, more frequent in men than in women, and usually appears, not in childhood, but in early adult life. It often begins in the muscles of the face, giving what has been called the "myopathic expression," or rather want of expression. When fully developed, the condition is much like that of advanced progressive muscular atrophy, and probably some of the cases would be counted among the "juvenile" type of that disease, referred to above (p. 525).

TABES DORSALIS, OR LOCOMOTOR ATAXY*

History and nomenclature—Symptoms affecting movement and sensation—Lightning-pains—Visceral pains—Disorders of vision—Atrophy of joints—Perforating ulcer—Loss of knee-jerk—Complications—Course and event—Histology and anatomical seat of tabes—Discussion of the relation between the ataxy, dysæsthesia, and other symptoms—Ætiology—Diagnosis—Prognosis and treatment.
Hereditary tabes: Friedreich's disease.

History and nomenclature.—Among the chronic affections of the spinal cord, there is one which is characterised by an impairment of the power of coordinating or combining the actions of muscles in the execution of movements. That it is not an ordinary paralysis is evident from the fact that in some cases the contractile force of the muscles is undiminished. In England it is commonly known as "locomotor ataxy," the name being derived from that of *ataxie locomotrice progressive*, given to it in 1858 by M. Duchenne, of Boulogne. But in Germany the most usual designation for it appears to be *tabes dorsalis*, and this title, being as old as Hippocrates, would be far preferable, provided that its claim to represent the disease in question could be satisfactorily established. Now, there is no doubt that Romberg and others had described under the title *tabes dorsalis* most of the principal symptoms of ataxy, long before Duchenne wrote on the subject; and the French observer seems to have somewhat underrated the value of the work of those who had preceded him, although it is incorrect to say that he altogether ignored it. But, on the other hand, it is certain that *tabes dorsalis* had generally been used in a vague way for almost any very chronic and incomplete form of spinal paralysis, especially if it seemed to have been caused by sexual excesses and was attended with wasting of the muscles. Indeed, I cannot find that in this country the term had ever a precise signification. Moreover, it is only since the publication of Duchenne's observations that the attention of the profession has been kept fixed upon the disease, and its peculiar features were depicted by him far more accurately than by any previous writer. Consequently, although locomotor ataxy is a very clumsy name, it is still used. It is worthy of notice that Todd—who in 1847 ('Cyclop. of Anat. and Phys.,' iii, p. 721) pointed out the distinction between paralysis and loss of the power of coordinating movements, and who mentioned the difficulty of walking and the tottering, uncertain gait, while considerable voluntary power remained—made no attempt to give any name to the latter affection. In one respect his knowledge went beyond the point reached by Duchenne, namely, in assigning the seat of the complaint to the posterior columns of the cord. It might be thought that if we could find a convenient English equivalent for

* *Synonyms.*—Tabes dorsalis *v.* dorsualis—Spinal tabes—Locomotor ataxia—Ataxie locomotrice progressive (Duchenne)—Asynergie locomotrice (Trousseau)—Posterior spinal sclerosis—Chronic posterior leucomyelitis.

the German *Hinterstrangsclerose*, there would be an advantage in adopting it; but such a change of name would, in reality, be unfortunate, for, as we shall presently see, the lesion in question is far from being so distinctive of locomotor ataxy as it was at one time supposed to be.*

Clinical characters.—The onset of tabes is always insidious and its progress gradual. Ataxic symptoms not unfrequently appear only after months or years which have been marked by pains, by loss of knee-jerk, by contracted pupils, or other warnings. Nevertheless they remain the most constant and characteristic, and therefore may properly be first considered.

Motor symptoms.—These begin almost invariably in the lower limbs. In walking the patient lifts his foot high in the air and throws it outwards, and then he brings the heel to the ground with a stamp. If the case is a slight one, it is at first only in starting that he experiences a difficulty. Wilks relates how a gentleman whom he knew, if he stopped to look in at a shop-window, had to ask someone near to give him a push before he could set off again. Once fairly started, he did pretty well, but found himself unable to turn round sharply without stumbling. Another patient, when he had once walked some distance along a road, wanted to go back, and was obliged to guide himself up against the bank, when he fell down. A third, an out-patient, one day apologised for being late at the hospital by saying that his friends had sent him off in a wrong direction, and that he went on until he fortunately met an acquaintance who turned him round.

Most writers describe ataxic patients as rolling and staggering like persons who are intoxicated, but Wilks says that the gait rather resembles that of a man walking on a ledge who is anxiously balancing himself. It is, however, certain that many persons in the early stage of the disease have been condemned by their neighbours as drunkards. The attempt to run, or to hop, or even to ascend stairs, makes the patient's incapacity still more manifest, and he may be unable to stand on one foot, or with the two feet close together, without tottering and falling down. If told to walk in a straight line—as along one particular board in a floor—his course is most sinuous and irregular. The use of a stick, or leaning upon the arm of a friend, has an extraordinary effect in steadying his movements. In more advanced cases, however, as soon as he attempts to stand, his legs are violently jerked in all directions, and this, even though he may be held up on each side by an attendant. When lying on a couch he cannot carry the foot straight

* Since the above paragraph was written, the terms "Tabes dorsalis" and "Tabes" without a qualifying local adjective have become more common in England. The original meaning of Tabes dorsalis was of course purely clinical, and had reference to the weakness and pains in the back, the malnutrition and other disorders which follow exeessive sexual indulgence. In fact it nearly answered to what is now called Neurasthenia spinalis. Tabes dorsalis from this cause is regarded by Cullen in his nosology as a species of atrophy, distinguished from that of phthisis, of mesenteric tabes, and chronic suppuration, only by the absence of fever.

Now, however, the word *tabes* is not used, any more than its Greek equivalent *phthisis*, to denote generally wasting, atrophy, or marasmus.

It persists only in two combinations, "Tabes dorsalis" and "Tabes mesenterica," or tubercular enteritis and peritonitis, with swelling (probably always secondary) of the mesenteric lymph-glands. The latter disease is so different in its clinical features and associations, that confusion can scarcely arise with the former. Moreover, increasing knowledge has shown that locomotor ataxia is far from being the most prominent or the earliest symptom. There is therefore no reason against Tabes (or, when needful for distinction from Tabes mesenterica, Tabes dorsalis) being used. It conforms to the criteria stated elsewhere (p. 386) of a good name. It is short, classical, unmeaning, distinctive, and capable of forming an adjective. This adjective is ready made: *tabidus*—not *tabeticus*.

towards an object so as to touch it, nor raise the leg up in the air with an even movement.

The complaint may last for years without extending beyond the lower limbs, but in many cases it at length affects the arms. The patient then becomes incapacitated for actions requiring delicate manipulation, such as writing or playing the piano; he cannot fasten his nectie or button his clothes. If he is told to bring the two forefingers into contact from a distance, they are carried about in the most irregular manner before they meet. He cannot touch his own nose or rub his own eyes with certainty. He finds a difficulty in taking hold of anything held up before him, and when he attempts to draw or write in the air with his finger the figures are zigzagged in all directions.

Sensory symptoms.—Feeling is more or less impaired in the majority of cases of tabes. Subjective feelings of numbness, or of formication, are very often complained of. The patient says that when he stands his feet seem to be covered with thick woollen stockings, or to be treading upon a water-bed, or upon india rubber. The cutaneous sensibility may be found diminished in the feet, the legs, or even the thighs, when carefully tested as described at p. 397; but it rarely happens that there is any approach to complete anæsthesia. Ability to feel pain is often absent (*analgesia*), while tactile impressions are readily perceived. On the other hand, French writers point out that patients generally retain the power of appreciating differences of temperature long after they have lost common sensation. That curious symptom, the retarded transmission of impressions, is almost peculiar to locomotor ataxy. It especially concerns the sense of pain, but Hertzberg believed that tactile impressions and those of heat and cold may also be delayed. If there is no anæsthesia, the prick of a needle may first be only felt as a touch, and then, after an interval, a sensation of pain may follow. Erb says that the interval may be one of some minutes; Cruveilhier, who seems to have been the first to draw attention to the symptom in question, noted it at twenty or thirty seconds. Another point is that the pain produced by a slight injury may last longer than under normal circumstances and gradually culminate, and, as a consequence, that the patient becomes unable to count a series of similar impressions if they succeed one another at all quickly. The paræsthesiæ which accompany locomotor ataxy are by no means limited to the lower limbs. Several writers insist on the frequency with which sensations of numbness are felt in those parts of the hands supplied by the ulnar nerves, even at the commencement of the disease, and Trousseau has pointed out that the mucous membrane of the mouth may be anæsthetic, the patient not being able to feel the food between his lips, nor to appreciate its temperature, nor with his teeth to distinguish hard substances from soft.

A symptom on which Trousseau laid great stress, as indicative of locomotor ataxy, is the failure to execute movements as well with the eyes shut as when they are open. And there is no doubt that this symptom is often exceedingly well marked. Many a patient who can stand quite steadily so long as he is looking down at his feet totters and falls when he is made to close his eyes. It may even be the first indication that anything is amiss, as in the case recorded by the late Dr Bazire, of a man who seems to have believed himself to be quite well until he noticed that he could not wash his face in the morning unless he was supported by another person, or could lean against a wall; for as soon as he shut his eyes he lost his balance, and stag-

gered. According to Jaccoud, the effect of keeping the eyes open is in part due to the influence of light; for he says that a person who has ataxy cannot walk so well when they are closed as when they are open, even though something may be held in front of his chest in such a way as to prevent his seeing his feet. But, as Dr Bazire has remarked, this is no doubt due to the feeling of confidence inspired by looking about one, for nervousness has a marked tendency to augment the symptoms of the disease, so that a patient who is conscious of being observed by others cannot walk nearly so well as when he supposes himself to be unnoticed. A similar explanation must, I imagine, be offered of the fact, noticed by Benedikt and Friedreich, that some ataxic patients who are perfectly blind nevertheless totter more when they are made to shut their eyes.

At present, however, it is the opinion of those best qualified to form a judgment in the matter, that the inability to execute movements accurately with the eyes closed is, strictly speaking, a sign, not of ataxy, but of defective sensation in the lower limbs. Erb says that he has found this symptom wanting in certain cases, those, namely, in which sensation is absolutely unimpaired. On the other hand, it is invariably present when there is much anæsthesia. He admits, indeed, that it may often be noticed to some extent in cases in which there is no objective evidence of loss of sensation in the skin. But he accounts for this by supposing that it is the earliest indication of a failure of the so-called muscular sense. The obvious conclusion is that a patient suffering from any spinal disease may be expected to totter in attempting to stand with his eyes shut, if he has imperfect sensation in his legs and feet. There is no doubt that the want of stability is exaggerated and made more manifest by a defect of coordinating power. But it must be clearly understood that, however valuable this symptom may be in the diagnosis of tabes from rheumatism, neuralgia, or other peripheral affections of the limbs, it does not afford a criterion by which any one lesion of the spinal cord can be distinguished from the rest.

Paraplegic symptoms.—The fact is that the boundary-line between paraplegia and ataxy requires very careful consideration. I have stated that in some instances of tabes the contractile power of the muscles is undiminished. This was proved by Duchenne, who invented the dynamometer for the purpose. In the case of certain muscles, a rough estimate of their strength can easily be made. For example, one can pretty accurately measure that of the quadriceps extensor muscle, by getting an assistant to fix the lower part of the thigh, and then telling the patient (who should be lying on the opposite side) to keep the knee straight, while by grasping the malleoli one forcibly flexes it. Or one can test the force of the psoas and iliacus muscles by placing the man in a chair in the sitting posture, and making him lift his knee towards his chest, while at the same time one resists this movement with the hands placed over the lower end of the femur. Or, following Trousseau's plan, one may determine what weight the patient is able to bear upon his shoulders while standing with the support of a friend's arm or leaning against the wall. A young man under his care could support a hundred and sixty pounds in this way, and another patient was able to carry on his back a medical man who had imagined him to be paraplegic. But, while the theoretical importance of these observations is very great, as showing that locomotor ataxy may be altogether independent of paralysis, it is a great mistake to suppose that all, or nearly all, of the patients who exhibit a want of muscular coordination have their muscular

power undiminished. On the contrary, one often finds it much enfeebled, and some of the earliest symptoms in these cases are commonly an inability to continue standing for any length of time, a sense of fatigue in walking even a short distance, and the like. It is true that the suggestion has been made that such sensations are really muscular paræsthesia, and do not prove a loss of motor power; and Duchenne records their presence in his patients in whom he demonstrated that the force of the muscles was unimpaired. But my experience is quite in accordance with that of Erb, who found an evident paresis in at least one half of those cases in which the characteristic symptoms of locomotor ataxy existed.

A view which may be taken of such cases is that they are examples of incomplete paraplegia, with a certain amount of failure of coordinating power, as a complication. This, I think, is the opinion held by Dr Wilks. If one is right in supposing that a chronic inflammatory process in a particular region of the cord is the cause of tabes, it seems reasonable to admit that this may coincide with a similar process occurring in other regions; and it seems that mixed cases may in this way be accounted for. For most ataxic patients exhibit other symptoms which belong to chronic diffuse myelitis, beside those already mentioned.

One of them is the curious "girdle-feeling," described at p. 451. Trousseau speaks of some of his patients as feeling as if the chest or the legs were compressed by an india-rubber cuirass or stocking. Or the sensation may be that of a belt constricting the abdomen, of a garter tied below the knee, or of a tight shoe pressing on the foot.

Again, if we inquire how the functions of the pelvic viscera are carried on in ataxy we shall find but little to distinguish it from the partial forms of paraplegia. Thus the bladder is apt to be irritable, and its muscular power may to some extent be diminished, so that the urine no longer flows in a good stream, or is from time to time passed into the bed. Erb notes that the fæces are sometimes discharged without the patient's knowledge, and attributes this to a loss of sensibility in the mucous membrane of the sphincter. The sexual functions are almost always impaired. At an early stage the genital organs are often extremely irritable. It is said that this condition may be accompanied with an increased virile power, but much more frequently there is marked weakness. As the disease advances the patient generally becomes altogether impotent. There seems, however, to be no doubt that some persons have been able to beget children after having been for years the victims of tabes.

The reflex excitability of the cord to cutaneous stimuli is almost always present, although in very variable degrees—as indeed is the case in healthy ndividuals. On the other hand, recent observations by Westphal and Erb appear to show that the susceptibility to tendon-reflexes is from the earliest period of the disease invariably extinguished, which is far from being the case in paraplegia. The electrical irritability of the muscles is often perfect, but sometimes slightly augmented or impaired. They remain well nourished until the disease has reached an advanced period in its course; but this is likewise the case in paraplegia, unless the anterior cornua of the lumbar cord are affected. Ultimately many tabid patients pass into a condition of complete paralysis with atrophy of the muscles, bedsores, absolute failure of the bladder and cystitis.

The leading French and German observers, however, seem to think that in the mixed cases of ataxy and partial paraplegia the disorder of coordina-

tion is the fundamental and essential affection, while the impairment of muscular force is secondary and accidental. Their reasons appear to be partly based upon morbid anatomy, partly upon clinical observation.*

Pains.—A clinical feature of the disease, which goes far towards establishing its claim to be admitted in its entirety as an independent member of the nosology, is the frequency with which it is attended with certain remarkable early symptoms, warnings, or *prodroma*, which belong to no other spinal affection. Chief among these are sudden pains in the lower limbs or elsewhere. These may either be of a stabbing or boring character, as though a sharp instrument were thrust into the tissues at the spot, which is generally near a joint; or they may be like flashes of lightning or electric shocks shooting down along the course of a nerve. Charcot says that they are worse at night. They last but an instant and return again and again duriug a period of from four to eight days, after which they may disappear altogether for a fortnight, or even for some months. Their occurrence seems to be favoured by changes of weather. Erb remarks that he has often been struck by the unanimity with which different patients have complained of them on some particular day when there has been much wind, or a fall of snow or of rain. It is also said that they are especially liable to return at spring and autumn, and that they may be brought on by over-exertion of body, mental emotions, or the excitement of coitus. They are sometimes of the most agonising character, sometimes so slight that careful inquiries are needed to elicit the fact of their presence. They are commonly set down as neuralgic or rheumatic. They frequently spread from the legs to the trunk, much more rarely to the arms. They may go on for five, ten, or fifteen years, before any other symptoms show themselves. They often persist throughout the whole course of the disease. As to the frequency of their occurrence, Topinard found that they were wanting in only 22 cases out of 104, Erb in only 8 out of 60, Cyon that among 203 patients there were 138 in whom they were said to have been present, but no more than 8 in whom they were expressly stated to have been absent.

Visceral pains.—Yet other early symptoms mentioned by Charcot are the following:—A pain in the bladder or in the urethra, with a constant desire to micturate, a sudden pain in the rectum, and certain strange attacks of stomach disorder, which he terms *crises gastriques*, and which he says were first noticed by Delamare in 1866. He describes them as consisting of lightning-like pains, which start from the groins and pass up both sides of the abdomen towards the shoulders. They are generally accompanied by palpitation of the heart and a quick pulse, by vomiting (of a liquid at first clear but afterwards stained by bile or blood), and by giddiness and malaise. They commonly last for two or three days at a time, and then pass off entirely. They must be carefully distingished from mere dyspepsia, to which tabid patients are as liable as other people.

Ocular disorders.—But most remarkable of all are what Charcot terms the *cephalic* symptoms of tabes, principally affections of some of the ocular muscles and of the eyesight. The functions of one or another of the branches of the third nerve on one side, or of the whole of the sixth nerve, may be more or less completely annulled, so as to cause diplopia, strabismus, ptosis, and various other disorders, such as have been described in a previous chapter

* Dr Gowers recognises Westphal's "Ataxic Paraplegia," with lateral and posterior sclerosis, as a separate affection ('Lancet,' July 3rd, 1886, and 'Dis. of Nervous System,' vol. i, p. 341).

(p. 407). Von Graefe is said to have remarked that in tabid patients, when they have double vision, there is particularly little tendency to fusion of the images, and that this points to a central origin. The paralysis may be quite transitory, or may last only a few days, or may persist for weeks or months; it may return again and again; and may end by becoming permanent. Another common symptom is an inequality of the pupils, one of them being constantly smaller than the other. Still more often both irides are symmetrically contracted to an extreme degree; this *myosis* is often among the earliest warnings. Another important symptom is that the pupils are insensible to the stimulus of light, but contract with accommodation for near objects. This condition is generally persistent. Its recognition is due to Dr Argyll Robertson, of Edinburgh.

With or without some of the symptoms just enumerated impairment of vision is frequently noticed. This is said to develop itself as a progressive narrowing of the visual field, generally from without inwards, until the only part of the retina that remains sensitive to light may be a small patch to the inner side of the blind spots. It is accompanied by achromatopsy; the perception of green being generally the first to disappear, then that of red, and lastly that of yellow and of blue. The defects of vision are commonly worse in bright light, so that the patient sees better after sunset. The pupils are often permanently contracted when there is nearly complete blindness. The ophthalmoscope reveals atrophy of the disc, which in advanced stages is perfectly white, with sharply outlined edge, and very small arteries, whereas at an earlier period it may be greyish in colour. The two retinæ generally suffer together; the affection is a progressive one, and commonly goes on until there is complete amaurosis.

As to the significance of those cephalic symptoms, the occurrence of which in a spinal disease has of course attracted unusual attention, I shall have something to say further on. Their frequency appears to be very considerable. Erb estimates that, if slight and transitory phenomena are reckoned up with the rest, affections of the ocular muscles are present in more than half of all the cases of ataxy; and that persistent affections exist in one third or one fifth. How often the optic discs undergo atrophy cannot yet be stated accurately; ophthalmologists are apt to rate it too high, physicians too low; Erb observed it only 8 times in about 70 cases. Topinard found visual disturbances in 49 of 102 cases; and Cyon, amblyopia or amaurosis in 60 of 203 cases. Optic neuritis often occurs at a very early period; according to Charcot it may precede all other symptoms of ataxy by ten years.

Arthropathy.—Another most curious early complication is a *joint-affection*, which has been specially studied by Charcot. Its most common seat is the knee, next in frequency comes the shoulder, and then the elbow, the hip, the wrist. It is not traceable to any blow or other injury. It sets in suddenly with extreme swelling; there being not only effusion into the synovial cavity, but also a brawny infiltration of the parts around. Yet there is generally neither pain, nor heat, nor redness. In the course of a few months this symptom may disappear, but it often ends in the destruction of the articular cartilages, with erosion of the ends of the bones and partial dislocation of their surfaces. Charcot says that it never occurs at an advanced period of the disease, except in the upper limbs, when they are beginning to be affected in their turn. He styles it "arthropathie ataxique" or "tabetique."

Other trophic lesions occasionally seen in the course of tabes are: pigmentation or leucodermia in the course of the nerves, like the effects

observed in ordinary neuralgia; herpetic eruptions like those of zona; painless ulceration about the fingers and toes which make the nails fall out; painless loss of teeth, of which we lately had a striking case in Philip Ward; and, most remarkable of all, ulceration of the sole of the foot, which still, without notable pain, gradually bores deeper and deeper until it makes a sinus through to the dorsum. This "perforating ulcer of the foot" has been long known to surgeons, and is now believed to be most frequently a symptom of tabes.

Loss of knee-jerk.—One of the most constant of all symptoms of tabes, first pointed out by Westphal, is complete loss of what is called the "deep knee-reflex" (*vide* p. 440). This begins early, and probably continues unchanged throughout the disease. The true (superficial) reflexes are usually and progressively diminished in proportion to the anæsthesia, but are never absent as in atrophic paraplegia; they are sometimes greatly exaggerated in the early stages, as much so as in many cases of spastic paralysis. Sexual power is said to be at first increased; it is usually found wanting. Of the visceral reflexes, that of the cremaster is usually impaired, the bladder is but slightly affected in uncomplicated cases, and the pupils almost always cease to contract to the stimulus of light.

Complications.—These are chiefly complete loss of motor power (secondary paraplegia) causing ataxic paraplegia, as described by Charcot, which Dr Gowers prefers to describe as a separate disease; secondly, muscular atrophy; thirdly, cerebral symptoms, such as epileptiform fits, and occasionally hemiplegic attacks; and fourthly, general paralysis with insanity. Cystitis, with other pelvic symptoms succeeding the slight affection of the urinary reflexes on the one hand, and optic neuritis with blindness succeeding the ordinary amblyopia, may be rather regarded as ingravescence of normal symptoms than as new complications. Tabid symptoms, including loss of knee-jerk, are exceedingly common in asylums.

Course and event.—Tabes is most insidious in its origin, most irregular, slow, and uncertain in its progress, and most intractable in its course. This is one of the greatest difficulties in tracing its natural history. Patients must be watched not for a few weeks, but for months and years. They are common in our great hospitals, and by their frequent reappearance in fresh places, and under fresh observers, they give an exaggerated impression of their numbers; but autopsies on cases of tabes are rare.

After lightning-pains, loss of knee-jerk, and myosis have lasted for several years, there may still be no ataxic symptoms. After marked locomotor ataxia is developed, there may be long delay before the advent of the paraplegic, pelvic, or cephalic symptoms which belong to the latest stage. And after apparently steady, and, by comparison, rapid progress, the disease may suddenly be checked and remain stationary for months or years. Still the natural tendency is to ingravescence.

Anatomy.—I have already mentioned that sclerosis of the posterior columns of the cord is found after death from tabes. The current description of this change is that it is visible as a grey or greyish-yellow streak on the surface on each side of the posterior median fissure; in transverse sections it is most marked in the upper lumbar and dorsal regions, and diminishes in extent, both above and below. Sometimes it reaches upwards as high as the restiform bodies. Its histological characters are those which have already been given of sclerosis generally. In the more advanced stages most

of the nerve-fibres have disappeared, their axis-cylinders being no longer discoverable ; but even in the oldest cases a not inconsiderable number of them are found with their structure unaltered, scattered through the dense connective tissue, and can be readily recognised, either negatively by carmine staining, or positively by help of osmic acid. The posterior roots are thin, grey, and atrophic. The pia mater at the back of the cord is generally opaque, thickened, and more or less adherent to the other membranes. The peripheral sensory nerves are atrophied, but not the trunks outside the posterior roots. The posterior cornua are usually found atrophied in advanced cases.

Romberg described as follows the posterior sclerosis in a typical case which was seen by himself and by Froriep :—"I was not a little surprised to find that the atrophy of the spinal cord (which, compared with the fresh cord of a man of the same age, fifty-two, was only two thirds of its size) was confined to the lower part of the posterior columns and the corresponding nerves. The medullary tissue (*i. e.* white substance) of the former had almost entirely disappeared, so that they were translucent and of a greyish-yellow colour. The posterior roots of the nerves were deprived of the nervous substance (*i. e.* myelin) and presented a watery appearance." He states that the lumbar and lower dorsal regions were alone affected, and that the anterior columns and nerve-roots were unaffected.*

There have been many speculations as to the mode of origin and significance of this lesion. Do the nervous elements first undergo degeneration, and is the growth of connective tissue the result of secondary reaction ? Or is the original morbid change chronic inflammation of the neuroglia, leading afterwards to an atrophy of the nerve-fibres, as cirrhosis of the liver leads to destruction of its secreting cells ? Or does the disease begin as a meningitis, and spread to the cord ? Or is its starting-point in the sensory nerve-roots ? How far do the admitted varieties in the course of the disease correspond to anatomical changes in the cord ? Is the lesion itself constant ?

Trousseau refers to a case, of twelve years' duration, which had been seen by Duchenne and accepted as typical, and in which Gubler and Luys and Duchenne examined the cord and its posterior roots, and could detect no alteration in them, whereas many of the nerve-fibres had disappeared from the anterior roots. Dr Alex. Hughes Bennett published in the 'Clinical Transactions,' vol. xviii, a case which presented the classical symptoms of tabes during life ; but the posterior column and nerve-roots were found healthy. The lesion was sarcoma of the bulb and recent cerebral mischief. He quotes two similar cases of M. Déjerine. The importance of such negative evidence is enhanced by the fact that the course of the disease is commonly so protracted as to give one scarcely a chance of tracing it to a fatal termination in ordinary hospital practice.

On the other hand, a precisely similar change in the posterior columns is frequently seen as the secondary result of local myelitis, without any symptoms of ataxy having been present. In cases of Pott's disease of the dorsal vertebræ, for example, "ascending degeneration" often exists in the cervical part of the cord, but the coordination of the movements of the arms and hands is in no way affected by it.

* We have already seen that Todd distinguished the symptoms of Locomotor Ataxy from Paraplegia, and placed the disease in the posterior column of the cord on physiological grounds. His hypothesis was confirmed in the cases he examined *post mortem* (1847). Mr Stanley, of St Bartholomew's Hospital, had described the same anatomical condition in a case of tabes (probably for the first time) in the 'Med.-Chir. Trans.' for 1840.

The solution of the second set of difficulties seems to have been discovered by Pierret. The secondary ascending sclerosis just described is limited to the internal fasciculi of the posterior columns, the columns of Goll. He finds that in cases of tabes these parts of the cord are affected in the cervical region, when the upper limbs had escaped, as much as when they were involved in the disease. On the other hand, a similar morbid change in the external fasciculi of the cervical posterior columns, the *bandelettes externes*, or *fasciculi cuneati*, Burdach's columns, occurs only when there have been irregular movements of the hands and arms, and it is more marked on that side of the cord corresponding to the limb which was the more disordered in its actions. The conclusion is obvious that in tabes, as in other diseases of the cord, sclerosis of the tracts of Goll is an "ascending degeneration," and has nothing to do with the symptoms of which the essential lesion has its seat in the outer parts of the posterior columns. Pierret relates a case in which sclerosis limited to the *fasciculi cuneati* was found in the dorso-lumbar region, the patient having died of hæmatemesis after displaying some of the early symptoms of ataxy.

In transverse sections the sclerosis is seen as two narrow grey streaks, one lying behind each of the posterior cornua of the cineritious matter, and parallel with it. It is not always obvious to the naked eye, nor until the cord has been hardened, so that one can understand how it has come to pass that some good observers should have declared the cord healthy, when locomotor ataxy had been present during life. The negative results, however, obtained by competent histologists with modern methods cannot be easily set aside.

Pathology of tabes.—This brings us to the interesting but difficult question of the physiological connection of the morbid process with the symptoms above described. Now, it has been shown that inability to stand or walk steadily with the eyes shut must no longer be counted a distinctive feature, since it is no more than an effect of anæsthesia of the lower limbs. A precisely similar doctrine, however, is held by some authorities with regard to the failure of coordination itself. Basing his views upon the acknowledged fact that guiding sensations contribute to the due execution of movements, Leyden maintains that ataxy is merely a consequence of interruption in the transmission of such sensations along the cord.

It must be admitted that some plausible arguments can be brought forward in favour of the doctrine in question. One is that the most obvious lesion is seated in a part of the cord which is (or was) believed by physiologists to convey afferent impulses. Another is the frequency with which a more or less considerable degree of anæsthesia is present, even at an early period. And if there are cases in which cutaneous sensibility appears to be perfect, it may be replied that in many of them the sensibility of the muscles and tendons and joints has not been tested, and that this is no less essential for the even and regular execution of muscular movements. The fact that it is impossible to elicit tendon-reflexes, even at the commencement of the disease, may be pressed into the same argument.

Nevertheless, it would seem that there are facts which make it impossible for us to accept Leyden's views. One is that some cases, at least, of ataxy are unattended with any discoverable anæsthesia or defect of muscular sensibility. Both Friedreich and Erb speak positively about this. Again, there is no close correspondence in individual patients between the degree of impairment of sensation and that of disorder of coordination. Lastly,

there may be absolute anæsthesia without the slightest irregularity in the movements of the limbs. A case in point was recorded by Späth in 1864, and was ten years later followed to an autopsy by Schüppel. The patient, whose name was Remigius Leins, had for more than twenty years lost sensation in his arms and hands, and for six years in his feet. He was quite unconscious of the position of his limbs; and when they were moved by another person he knew nothing of it. Yet he could walk steadily, without support, and tolerably fast. When asked to raise his foot to a certain height with his eyes shut, he did it with a perfectly even and regular movement. After death the whole length of the cord was found excavated in the centre; the posterior columns in the cervical region were completely destroyed; the anterior columns were everywhere normal. It is particularly to be noticed that in this instance the muscular sensibility was absent, as well as that of the skin.

If, then, ataxy is not due to any interruption of afferent or sensory impulses, how is it caused by a lesion situated in the posterior columns? Most writers seem to suppose that these columns may possess the special function of conveying impulses from a coordinating centre above, in the medulla oblongata or pons, to the motor nuclei in the cord. But I think it is clear that coordination, instead of being affected by a single centre, must require a very complicated apparatus, probably consisting of several parts situated in different regions of the cerebro-spinal axis. We have seen that the reflex movements which are performed by the spinal cord independently of the brain are never entirely un-coordinated, and that sometimes they are coordinated in a very complex way. One can hardly doubt that the machinery which is brought into operation under such circumstances is also made use of when similar movements are excited by the will. Again, I fail to conceive why there should be any channels for the conveyance of motor volitional impulses, apart from those which pass through, or are connected with, any centres for higher coordination that only exist in the medulla oblongata or elsewhere in the brain. To whatever extent the movements of the trunk and limbs are coordinated above the spinal cord, to that extent surely must the motor tracts of the cord itself transmit impulses themselves already coordinated.

Moreover, we must remember that what is observed in ataxy is by no means an entire absence of coordination, such as would occur if the volitional impulses passed straight down to an individual muscle or to a group of muscles, without relation to any others. On the contrary, it is probable that the failure is very often of the slightest possible kind. Scarcely appreciable irregularities in the force which the muscles severally exert, or even in the intervals of time at which they are increasingly brought into action, would account for all the phenomena presented by some well-marked instances of the disease. No wonder, then, that lesions of the proper motor tracts of the cord do not cause ataxy. We should rather expect it to be due to some affection just touching the outskirts of these tracts so as to injure them only very slightly. A sclerosis commencing in the fasciculi cuneati of the posterior columns seems to be just such a lesion as might produce this effect.

Again, it is certain that even in the earliest stage of the disease other parts besides the posterior columns must be involved. In no other way can one explain many of the prodroma. What is to be said as to the origin of the atrophy of the optic discs? Pathologically the morbid change in them is identical with that which is going on in the cord itself. But why should

it occur in cases of tabes, and not in those of diffused myelitis? And, further, why should amaurosis from this cause often precede all spinal symptoms by an interval of several years, so that Charcot believes that the majority of the female patients admitted into the Salpêtrière for simple atrophy of the discs become sooner or later the victims of ataxy? The affections of the ocular muscles are supposed by Erb to be due to slight lesions of the nerve-roots or of their nuclei in the pons and bulb, but he does not attempt to show how it is that only motor structures are attacked, nor why the nerves concerned in moving the eyeballs suffer more than any others. As regards the last point, indeed, we may perhaps see an explanation in the delicate way in which the muscles in question are naturally balanced against one another. This renders the slightest impairment of the power of any one of them at once obvious to both the physician and the patient. The same degree of paresis elsewhere would be altogether incapable of recognition. It is said, however, that actual paralysis of the facial muscles, or of those concerned in mastication, has sometimes been observed in tabid patients.

The joint-affection which is now and then seen among the prodroma of locomotor ataxy is believed to depend upon an atrophic change in the cells of the grey matter of the anterior cornua. Joffroy and Westphal are said to have made observations which directly confirmed this opinion.

On the other hand, the boring and flashing pains are doubtless dependent upon the morbid changes in the fasciculi cuneati, *i. e.* the posterior root-zones, and the nerve-roots immediately adjacent.

There is abundant proof that in the more advanced stages of the disease the morbid process may extend in all directions within the substance of the cord. Changes in the peripheral parts of the lateral columns were long ago noticed by German observers, and doubtless give rise to the paralytic symptoms which are so commonly present. Charcot says that the nerve-cells of the anterior cornua may be destroyed so as to cause atrophy in the corresponding muscles. When there has been an absolute loss of power in the lower limbs, almost the whole thickness of the lumbar cord may be wasted, tough, grey, and transparent looking.

The fact that even the prodroma of locomotor ataxy can be accounted for only on the supposition that changes exist in structures far distant from the posterior columns of the cord, makes it surprising that a definite boundary-line should be traceable between it and other spinal affections. As in Multiple Sclerosis (the disease to be described in the next chapter), it seems that the clinical features of the disease are far more uniform than would have been anticipated by the morbid anatomist. Charcot, indeed, admits that pains, which he says are precisely like those that occur in the early stage of ataxy, and which he believes to be really due to a sclerosis of the lateral columns, may be observed in disseminated sclerosis, in general paralysis, in chronic alcoholism, and even in diffuse myelitis or compression-paraplegia. Probably the lesions which give rise to even the partial forms of paraplegia are too coarse and too diffused to be capable of producing the slight and delicate disturbance of function which seems to be characteristic of tabes.

So difficult is it to connect the symptoms of tabes with its morbid anatomy, so few, after all, are the cases which are watched during life and satisfactorily investigated after death, and so complicated and various are both the clinical symptoms and the local distribution, that some experienced

physicians even now doubt the reality of the connection between locomotor ataxy and posterior sclerosis.

But an impartial study of the evidence seems to show that, while locomotor ataxy is not the invariable result of sclerosis of Burdach's columns, the combination of disturbed movements, impaired sensations, loss of knee-jerk, defects in the eye and its muscles, and peculiar neuralgic pains, make up a "complex of symptoms" which has a beginning, a middle, and an end and may therefore be called a disease.

Ætiology.—Among tabid patients the preponderance of males over females is very marked, but the proportion is variously stated by different observers at 8 to 1 (Erb), 3½ to 1 (Cyon), 2½ to 1 (Carré, Schulze). Eulenburg found in 149 cases, 128 men, including 2 youths under twenty and 5 men over fifty, *i. e.* 6 males to 1 female. Ataxy very rarely occurs in persons under twenty years old, or begins in those who have passed the age of fifty. The case of an old gentleman of eighty is mentioned by Trousseau; but it is not stated how long he had been a sufferer. This writer gives some instances in which there was a marked family neuropathic disposition. One patient had an uncle and an aunt insane, a brother ataxic, and a brother hemiplegic; another patient was the son of a man who committed suicide, and two of his sons laboured under certain peculiar nervous affections. Erb, however, says that he has seen many cases in which no predisposition to nervous diseases could be traced. Carré has recorded the direct transmission of ataxy by inheritance in a family of which the grandmother, the mother, her seven children, and eight others of her relations were all attacked. But such instances are very rare; and, as we shall presently see, it is possible that they may hereafter be found to constitute an independent malady.

With regard to the exciting causes there is much uncertainty. Most often none are discoverable. Sometimes tabes has followed an attack of fever, acute rheumatism, or pneumonia; sometimes it has taken the place of paraplegia in sequence of diphtheria, as in cases recorded by Jaccoud and Erb. Syphilis has been said by some writers to be a frequent precursor, and few now believe that this connection is accidental.* Tabes has occasionally followed injuries, not merely such as affect the spine, but a fracture of the thigh, or a fall on the abdomen. In some cases it appears to be pretty clearly traceable to a severe chill, as from falling into water, or sleeping on damp ground; to over-fatigue, as in soldiers after forced marching; to sexual excesses, or onanism; to prolonged mental anxiety or distress; possibly even to excessive indulgence in tobacco. The wars at the beginning of the present century seem to have given rise to numerous cases of ataxy among the French and German soldiery; it is difficult to say exactly how campaigning sets up the affection, but possibly by the combined operation of several of the causes already mentioned. The greater liability of men between the ages of thirty and fifty to these various morbid influences is doubtless the reason why male adults are especially apt to suffer from ataxy; as indeed from several other serious nervous diseases.

* Tabes was first definitely connected with syphilis by Fournier, who believed that among 51 tabid patients 37 had suffered from lues. Erb strongly supported this theory, and his statistics gave the proportion 27 in 44; Vulpian found 15 in 20, Buzzard 49 in 100, Gowers 53 in 100, and some writers made the proportion higher still. Thus Ross, among 20 cases of tabes, could only exclude syphilis as an antecedent in a single one. See on this point 'Trans. Internat. Med. Congr. 1881,' pp. 32—42.

Diagnosis.—From the account which has been given of the early symptoms of tabes, it must be evident that its diagnosis at that period of the disease is often a matter of extreme delicacy. Probably there is scarcely a case in which the premonitory pains are not for a length of time dismissed as neuralgic or rheumatic. Every medical man is sure to be consulted at one time or another for pains in the limbs by patients who are to become the victims of ataxy. In many cases of this kind a careful investigation will elicit other indications of it. There may be a slight squint, a contracted state of the pupils, or a failure of tendon-reflexes; or the patient may totter a little when told to walk, or to stand with his feet close together, while keeping his eyes shut. This last is a most valuable sign when the doubt is as to the presence of any spinal affection, notwithstanding that it is almost without significance as between tabes and other diseases of the cord. If no corroborative evidence can be discovered, one can only wait before giving an opinion as to the nature of the case.

When the complaint is fully developed, its diagnosis is generally easy. Pronounced paralytic symptoms, with wasting of muscles, may sometimes cause one to overlook the prior existence of an ataxy, and to concentrate one's attention on the lesions in the motor regions of the cord; but such a mistake can hardly be deemed very serious. Again, when there is "multiple sclerosis," and when the posterior columns are also attacked in a marked way, it may be impossible to assign the case to one affection rather than to the other. We have seen that tabes not infrequently occurs in association with "general paralysis of the insane," so as to cause confusion in the minds of those who happen to be more familiar with one than with the other of these two diseases.

The failure of coordination which accompanies disease of the cerebellum does not seem to be likely to be mistaken for the complaint with which we are now concerned. It may generally be distinguished by the absence of other spinal symptoms, and by the presence of headache, giddiness, vomiting, optic neuritis, epileptiform attacks, and the various other signs of an encephalic lesion. And, so far as I can gather from the statements which have been published, the so-called cerebellar ataxy is not attended with the peculiar mode of walking described, but merely with a reeling tumbling gait, exactly that of a drunken man.

Prognosis.—Notwithstanding what has been said above of the relentless progress of the disease, we must remember, first, that this progress is always slow, often exceedingly slow, and frequently interrupted for an indefinite period; secondly, that it does not lead to death unless one of the complications above noticed supervene. Probably most tabid patients die of phthisis, because that is the commonest of diseases; nearly as many perhaps die of latent pyelitis or consecutive Bright's disease from vesical complications; others again from the bedsores of paraplegia, from the effects of cerebral or mental disorders, or from accidental injuries. Great improvement often takes place under judicious non-specific treatment, and although patients are not cured of the disease, they are of its most distressing symptoms.

Erb says that the prognosis of tabes is not quite so bad as that of multiple sclerosis or of simple transverse myelitis; but this means little more than that the disease advances at a comparatively slow rate, for it very rarely subsides entirely. As a rule, its duration varies between six and twelve years, and it may last twenty or thirty years. Some writers

have described an acute form, and I remember one case which was so diagnosed in a woman who died in hospital six weeks after admission, and about ten months from the beginning of her illness. But in this instance there was softening of the anterior columns in the lumbar region, and it now seems probable that the marked change found in the posterior columns was only a secondary degeneration. The course of the disease is not always steadily progressive; it is often quiescent at a certain stage for a very long period. Generally it is better in the summer than in the winter. Sometimes it scarcely seems to shorten the patient's life.

Treatment.—When tabes is fully developed it rarely yields to treatment of any kind. Erb, however, mentions two cases, in each of which almost complete recovery took place. One patient afterwards held a Government appointment for several years; the other, who had been unable to get about without help, and had had incontinence of urine with cystitis, regained the power of walking for three or four hours at a time, and that of holding his water for five or six hours; he married and he took the command of an ironclad frigate. The treatment which Erb recommends most strongly consists in galvanism applied as a continuous current to the spine. He uses a moderate number of cells, for three to six minutes at a time, once daily. Of sixty-six cases thus treated, twenty-five received no benefit, forty-one were more or less improved. He also galvanises the peripheral nerves.

The drug which appears to be of most value is the nitrate of silver; it often fails entirely, but sometimes does great good. We generally order a quarter of a grain three times a day. Belladonna, gold, phosphorus, iodide and bromide of potassium all seem to be useless, and strychnine to be injurious. Calabar bean seems to do no harm. Arsenic (and ergot in early cases) may perhaps do good.

The general management of the patient is of great importance. He must be carefully protected from cold and damp by flannel underclothing. He must avoid all bodily and mental exertion. Dr Radcliffe thinks it advisable that crutches should be used in the early stages, so as to save the lower limbs as much as possible. It has been suggested that maintaining absolute rest in bed might favour the subsidence of the disease, and Dr Weir Mitchell brings positive evidence in favour of this plan. Dr Gowers recommends a sea voyage. Those who can afford it will find great benefit from spending the winter and spring in the South of Europe, or still better in the Azores or the West Indies. *Abstineto tabidus a vino et venere.*

Locomotor ataxy is among those affections for which persons resort to the various spas. Almost all writers are agreed that hot baths should be avoided, many believe that cold ones should be also. Erb says that any temperature above 58·5° F. may be injurious, and that the patient should bathe only once in two or three days, and remain in the water not above fifteen or twenty minutes. Nauheim and Rehme are the places which seem at present to have the highest reputation. Peat baths are recommended. But the figures given by Erb appear to show that the "cold-water" cure with packing, friction, and douches is quite as useful: of nineteen patients treated in this way, sixteen are said to have been benefited.

Among the *symptoms* of tabes, the pains most need relief. Sinapisms, blisters, liniments of chloroform or belladonna, veratria ointment, may be of some use; but often one cannot avoid frequent injection of morphia.

When tabes undoubtedly follows syphilis, we should certainly give the

patient a fair trial of antisyphilitic remedies. No doubt they often fail and in the latter stages of the disease probably do more harm than good, but the only cases of tabes seen by the editor which were "cured" were treated by mercury. One was a man about forty-five who was admitted into Guy's Hospital with marked ataxy, loss of knee-jerk and other symptoms, indeed myosis was the only important one absent. He had signs as well as history of syphilis several years before. Under a mercurial course he improved so much that he went out, walking well, free from pain and in fact cured, though the knee-jerk had not returned. He stated while in the ward that he had some years before suffered from similar symptoms (ten years or so after primary syphilis) and had been cured by the same treatment.

Another case is a patient who has been under my observation for five or six years. He had syphilis years ago, and still has occasional tertiary sores on one heel. Other symptoms of tabes have disappeared under mercury and iodide of potash, but he still has minutely contracted pupils and loss of knee-jerk. Neither case is conclusive, but it seems worth trying this method when we know that syphilis preceded the nervous symptoms.

Lastly, mechanical stretching of the nerves has been introduced for tabes. It was first performed by Langenbuch with apparent success; but when the patient died, the cord was found healthy. On the whole, the good effects seem to be doubtful and the drawbacks and dangers undoubted. See an interesting paper by Dr Cavafy, with eighteen cases, beside one of his own ('Brit. Med. Journ.,' Dec. 10th, 1881).

FRIEDREICH'S DISEASE.*—Friedreich first described in 1863, and more fully in 1876, a *hereditary* or rather family form of Tabes which presents several peculiar features. He recognised it in only three families; in each of them it attacked in succession several children of the same parents, in all nine patients, of whom seven were girls. It began unusually early, at or near the age of puberty, between the thirteenth and the eighteenth years. Among eleven cases Rütimeyer found seven boys and four girls, the ages being all under puberty (1883). Since then further observations have shown that this form of tabes occasionally occurs in two generations, much more often in several brothers and sisters; that it begins in childhood as early as four, at puberty, or in early adult life as late as twenty-four; and that girls are almost as often affected by it as boys.

The disease is attended with remarkably little disorder of sensibility, the prodromal pains in the limbs and the later girdle-sensation round the body are absent or very little marked, and there is little or no anæsthesia. The upper limbs show a loss of co-ordination at an early period. A peculiar form of nystagmus has been present in five cases; it is especially characterised by occurring only when the patient endeavours to fix his eyes upon an object before him; the movements are always bilateral, and are comparatively slow, being repeated about two or three times in a second. In every instance there has been a remarkable disorder of speech. The utterance is described as having been at first slightly lisping (Calloud), then as irregularly interrupted and stammering, and finally as almost unintelligible. It is altogether unlike the slow, accentuated articulation of a patient suffering from multiple sclerosis. The skin and joints are unaffected. Knee-jerk is absent. The senses and the mental faculties are perfect. Choreiform move-

* *Synonyms.*—Hereditary Ataxy—Juvenile Ataxia—Hereditary tabes in children—Hereditary ataxic paraplegia (Gowers)—Early tabes with nystagmus occurring in families.

ments of the face and limbs have been observed, so that the disease has been mistaken for chorea.

The duration of these cases has been very long, in one instance it was more than thirty years; a curious circumstance is that no fewer than five of the patients died of enteric fever.

Clinically it resembles Insular Sclerosis or functional spasmodic disorders as much as it does ordinary tabes.

Sclerosis of the posterior columns has been found in cases in which a *post-mortem* examination has been made, but it has been combined with sclerosis of the direct cerebellar and of the lateral columns. In two cases (Friedreich, and Kahler and Pick) the entire cord was remarkably small.

The author had once under observation a typical example of Friedreich's ataxy. The patient, a medical man, was first noticed to be uncertain in his gait when about sixteen years old. He was one day walking across a plank in a house that was being built when he tottered; afterwards he remembers that he had to be careful not to go near the edge of the path where it was raised above the road. But he came to the hospital as a student, passed his examinations, went into practice, and took a wife. At the age of about twenty-five he was obliged to give up his profession on account of an increasing inability to walk or ride; he became unable to write, and his speech became laboured and thick. In 1878, when he was thirty, he could just walk from one room to another with assistance; there was even then but little impairment of sensation in his feet; when he moved his lips in speaking a large number of the facial muscles were thrown into action, so that the mouth became drawn outwards into a meaningless smile; the words were tolerably distinct, but some syllables were slurred over, and others pronounced too strongly. The nystagmus was very obvious when he was told to direct his eyes to either side. He is a member of a highly neurotic family, and one sister suffers from some form of paralysis, but whether her case is like his I do not know.

We have since had three brothers in Guy's Hospital suffering from this juvenile family type of ataxia with nystagmus. Their cases will be reported in the 44th volume of our 'Reports' (1887), and are identical with three of the five cases in the same family recorded by Dr Gowers in the fourteenth volume of the Clinical Society's 'Transactions.' One of them died under the late Dr Moxon's care. Sections of the cord were made by Dr Pitt and are figured in Gowers's 'Diseases of the Nervous System' (vol. i, p. 354). They showed sclerosis of the posterior columns (both Burdach's and Goll's), with similar change in the crossed pyramidal and cerebellar tracts. The anterior columns, which also have been sometimes found affected, were free, and the grey substance normal. The lesion was most marked in the cervical and dorsal regions, but extended throughout the whole length of the cord.

Dr J. A. Ormerod has published a critical digest of the recorded cases of this remarkable form of disease in 'Brain' for April, 1884; and Dr Everett Smith (quoted by Gowers) has collected not less than fifty-seven cases in the 'Boston Med. and Surg. Journ.,' Oct. 15th, 1885.

INSULAR SCLEROSIS*

History—Anatomy—Sympioms—Ætiology—Diagnosis—Course—Prognosis and treatment.

In the great illustrated work of Cruveilhier, published between 1835 and 1842, there are figures taken from four different patients representing the spinal cord affected with what he termed "grey degeneration," in the form of patches scattered irregularly through its substance. In two of these cases he also gave the clinical features of the disease. Almost at the same time Carswell figured the same lesion in his 'Atlas of Pathological Anatomy' (1838), and Marshall Hall described the characteristic symptoms in his 'Diseases of the Nervous System' (1841). Afterwards various writers from time to time referred to similar cases, but it was not until about the year 1866 that a series of observations were made at the Salpêtrière, from which it results that the disease in question is characterised clinically by a remarkable and definite group of symptoms, so that it can often be diagnosed with certainty. In these researches Charcot took the principal part. The subject was soon taken up in Germany, but in England it seems to have been altogether neglected until in 1873 the late Dr Moxon published a case in the 'Lancet,' and in 1875 a paper containing eight cases in the 'Guy's Hospital Reports.'

Cruveilhier's drawings show that the bulb and the pons are affected as well as the cord, and the same morbid change has since been found in the hemispheres. Insular sclerosis might therefore with equal justice have been placed among diseases of the brain; indeed, several symptoms belong to it as a cerebral affection. But, on the whole, this appears to be the best place for it.

As a rule, the morbid change is at once obvious when the nervous centres are examined in the *post-mortem* room. Bourneville has, however, recorded one case in which they appeared to he healthy until the microscope was used. Some writers have wondered that so remarkable a disease so long remained unknown, but its slow and hopeless course renders it little likely to go on to its fatal termination within the wards of ordinary hospitals. I do not find a single case which can be assigned to disseminated sclerosis in our pathological records between 1854 and 1873.

Histology.—The patches of sclerosis are rounded or elliptical or very irregular in form. In the cord they come to the surface, and are sometimes seen through the pia mater. In the brain they scarcely ever penetrate into the cineritious substance of the cortex, so that they are visible only at the base. However, in the cord, the pons, and the basal ganglia, they show no tendency to spare the grey matter, or to affect one column or cornu more than another. Hence Insular Sclerosis is not a "systemic" disease in its distribu-

* *Synonyms.*—Disseminated Sclerosis—Sclérose en plaques (Vulpian)—Multiple inselförmige Sclerose (Leube)—Herdweise Sclerose—Sclérose en îlots multiples et disseminés (Liouville).

tion, nor focal, nor transverse, but essentially indiscriminate or scattered.* They are often scattered in large numbers throughout the white matter of the hemispheres. The cerebellum seldom contains many of them. A similar change may be present in the olfactory bulbs and in the roots of the various cranial and spinal nerves. In the peripheral trunks it has not yet been recognised: Dr Moxon found the brachial plexus in one case normal on each side.

The patches generally feel hard, but recent ones are said to be softer than the rest. These are also of a deeper grey tint, whereas the older ones are yellowish grey and less translucent. They become pinkish when exposed to the air. They may either project somewhat above the cut surface, or lie at the same level, or be slightly depressed. They vary from a microscopic minuteness up to the size of a hazel-nut or more. Histologically they present the characteristic appearances of chronic myelitis generally. Charcot says that there are fewer corpora amylacea, and that a distinction is afforded by the way in which naked axis-cylinders persist even when the neuroglia has become highly fibrillated, but the last point is contested by Erb. It is to be observed that in thin sections the affected parts are found not to be sharply defined at their edges as they appear to the naked eye; on the contrary, the sclerosis fades off very gradually into the healthy tissue. Dr Moxon remarks that granule-cells are more numerous at the circumference of the patches (and even in the apparently normal brain-substance beyond) than towards their centres. Deiters' cells and corpora amylacea are present.

Symptoms.—That the clinical features of disseminated sclerosis should vary in different cases is no more than we should expect from the irregular distribution of the patches, which would account for the utmost possible irregularity in the symptoms. Charcot, indeed, speaks of it as, *par excellence*, polymorphous. He and other French writers describe, in addition to the "cerebro-spinal" form of the disease, separate "spinal" and "cerebral" forms, but it is admitted that such distinctions are scarcely ever needed at the bedside. Moreover, notwithstanding individual variations, the symptoms present a degree of uniformity which seems to me to be very remarkable. This uniformity Dr Moxon regards as "a constant average result of the numerous points of disease." But the explanation leaves much unexplained.

We may refer disturbance of the special senses to sclerosis of their nerves, spasms to implication of the lateral columns, and mental symptoms to cerebral lesions; but neither in this disease nor in spastic paraplegia could we have predicted the symptoms from the anatomical lesions.

The tremors, the nystagmus, and the peculiar speech—the most constant symptoms—are quite unexplained.

Chief among the symptoms is a sort of *tremor*, which until recently was confounded with paralysis agitans. There are, in fact, records of two cases diagnosed as examples of that affection by Skoda and by Hasse respectively, in which patches of sclerosis in the nervous centres were discovered after death. Careful observation, however, has shown that in paralysis agitans the trembling movement is less extensive, more regular, more rhythmical,

* See an excellent plate in Dr Bramwell's work, which shows the distribution of the patches of sclerosis throughout the cord in a well-marked case, and the way in which they stain with carmine. This is an example of the larger-sized islands. In Dr Moxon's paper in the 'Guy's Reports' (1875) is a coloured drawing of the centrum ovale, with insular sclerotic patches, and a microscopic section of the cord.

and slower than in insular sclerosis. In the latter disease, when the hand is raised, the oscillations increase in amplitude as it gets further from the side, until they greatly embarrass and disturb the patient. If he is asked to carry a cup to his lips it becomes more and more violently shaken until at last it is dashed against the teeth, so as perhaps to spill its contents. In slight movements the tremor is not so marked; the handwriting, although shaky, long remains legible. Still more important is the fact that there is no agitation of the muscles as long as they are at rest and supported. When the patient is lying in bed, one could not tell that anything is the matter; but as soon as he is asked to sit up, the arms, and then the head and the neck, begin to oscillate. When he is resting in a chair the arms are quiet, but the head may still continue to show a slight tremulous movement. If he attempts to get on his feet the whole trunk and all the limbs become violently shaken, so that in severe cases standing is out of the question. Mental emotion or excitement aggravates the tremor considerably. According to Erb there are some cases in which it continues even when the patient is lying still, but this is altogether exceptional.

Charcot has suggested that this remarkable symptom depends upon the persistence of naked axis-cylinders in the parts affected with the sclerosis, and conceives that the transmission of volitional impulses may become interrupted and, as it were, jerking; but such a view appears unlikely. Ordenstein is disposed to think that the tremor depends in some way upon the presence of patches in the pons and still higher in the motor tracts, and Erb says that an analysis of twenty-two recent cases tends to support the notion of the connection of this symptom with abundant islets in the mesencephalon.

A second characteristic symptom of disseminated sclerosis is a peculiar *affection of the speech.* The pronunciation is slow, accented, laboured, as if the utterance of each syllable was a great effort. And yet it is not distinct; Charcot says that the letters L, P, G, are especially apt to be slurred over; and sometimes, though rarely, two or three syllables are run together. German writers describe the articulation as "scanning," each syllable being pronounced separately. In advanced stages speech may become quite unintelligible. Erb further remarks that the voice is pitched in a monotone; or, as in one case that came under his observation, that there may be a rhythmical alternation of notes at a definite musical interval. Leube found with the laryngoscope that the vocal cords, although capable of closing the glottis, were yet apt to relax, and liable to changes of tension ('Deutsches Archiv,' 1871).

Thirdly, *nystagmus* is commonly present. It, however, differs somewhat from the ceaseless oscillation of the eyeballs with which ophthalmic surgeons are familiar. As Dr Moxon pointed out, it is absent so long as the eyes are at rest, showing itself only when they are directed to an object. In other words, it is identical with the affection which is observed in Friedreich's hereditary ataxy (see p. 545); indeed, both Friedreich and Erb have suggested that the anatomical lesion may be the same; but rather as a matter of speculation than from direct observation.

In addition to these symptoms, there are others which belong to disseminated sclerosis in common with other diseases. Subjective sensations of *numbness* and other paræsthesiæ are not uncommon, although they were absent in Dr Moxon's eight cases; anæsthesia, or impairment of tactile sensibility, can seldom be made out. The reflex susceptibility is generally

normal, but sometimes it is much exalted. The gait at an early stage is often *spastic* in character. Knee-jerk is exaggerated, and clonus often present. Afterwards there is a marked tendency for the lower limbs to become rigidly extended and adducted; this condition being at first transitory, but afterwards permanent. Dr Moxon mentions that one of his patients was obliged to sit in an elbow-chair, so that when her legs stiffened she could hold on to the arms and save herself from sliding down on to the floor. Such early contractions are painless, but at a later period a painful flexion of the limbs is not infrequent. There is no reaction of degeneration, and the muscles react to both currents until they finally undergo atrophy. The characteristic symptoms of locomotor *ataxy* are now and then present in disseminated sclerosis; this is supposed to depend upon the existence of patches in the posterior columns of the cord. After a long time complete *paraplegia* usually develops itself. The functions of the pelvic organs are at first but little interfered with. Constipation is often complained of. Dr Moxon refers this in part to weakness of the abdominal muscles. The sexual organs retain their powers.

Transient *diplopia* is not infrequent at an early stage of the disease. Among other *cephalic* symptoms amblyopia often occurs, with narrowing of the field of vision and achromatopsy. Charcot insists on the fact that the impairment of sight rarely goes on to complete amaurosis, even when the optic nerves have been found sclerosed in their whole thickness; he refers this to the persistence of naked axis-cylinders in the midst of the diseased tissues. The optic discs are generally normal unless there is total blindness; in the latter case they have sometimes been found in a state of white atrophy. Headache, giddiness, and sleeplessness are not uncommonly present.

The spirits are often depressed, and the *memory* and *intelligence* become gradually impaired. The patient bursts into uncontrollable laughter, or sheds tears without any cause. Charcot insists on a peculiar *facies;* the expression is, he says, vague, uncertain; the lips droop and are half opened; the features have an air of dulness, even of stupidity. Sometimes there is an actual insanity. Charcot mentions two patients who exhibited the *délire des grandeurs* supposed to be characteristic of general paralysis; in others there has been melancholia, with refusal of food. A curious feature, which is observed in about one fifth of the cases of disseminated sclerosis, is the occurrence of attacks of stupor, followed by transitory hemiplegia. They are attended with flushing of the face, a rapid pulse, and a temperature raised to 104° or even 106°. The coma lasts for a day or two, and then passes off into sleep. Sometimes, however, a seizure of this kind is directly fatal; one of Dr Moxon's patients died so, in about twenty-four hours. Such an attack is said always to lead to an aggravation of the general symptoms of the disease, even when it is recovered from.

Lastly, in some exceptional cases atrophy of muscles is observed, affecting the upper or lower limbs, the face, or the trunk. At the same time their electrical excitability, which ordinarily remains normal, may be lowered; the natural reaction is doubtless replaced by the "reaction of degeneration." Charcot says that the characteristic symptoms of bulbar paralysis sometimes show themselves, and dysphagia or paroxysmal dyspnœa may then be the immediate cause of death.

It is obvious that many of these symptoms of insular sclerosis are due to the disease invading the posterior or lateral columns, the anterior cornu

and the nuclei in the bulb and mesencephalon, so as to produce the symptoms which belong to tabes, spastic or atrophic paralysis, and so on. The paraplegic symptoms again, with bedsores, retention of urine, and other symptoms, belong to the last stage of all chronic diseases of the cord.

Ætiology.—Disseminated sclerosis is now ascertained to be equally frequent in either sex, although Charcot found twenty-five females to nine males among the first thirty-four cases which he collected. The age at which it most frequently develops itself is said to be between twenty and twenty-five years, but it has been several times observed at puberty, and instances of its occurrence in childhood have been recorded. It has not hitherto been known to begin in persons over forty or forty-five years old. Now and then it has appeared to be hereditary or has occurred in two children of the same parents.

Like other chronic affections of the cord it has been ascribed to "chills," to falls and blows on the head, and to mental excitement. Bauwinkel observed a case in which the patient fell into water and allowed his clothes to dry upon him, three days before the first symptoms appeared. Charcot cites instances in which the disease began during convalescence from typhoid fever, cholera, or smallpox. We may therefore conclude that these several conditions do not protect a person from insular sclerosis.

Diagnosis.—This is not invariably, or even generally, an easy matter. Charcot says that trembling movements, precisely like those which are present in this disease, may occur not only in mercurial poisoning (when the history will in most cases remove all doubt), but also when the lesion is a "chronic cervical meningitis with cortical sclerosis," and even where it is a "primary or consecutive sclerosis of the lateral columns." The presence or absence of the other characteristic symptoms must guide the observer to a decision so far as these affections are concerned. The diagnosis from Paralysis agitans is not the most difficult, for that disease occurs in older patients, the tremors continue when the muscles are at rest, and affect the hands more than the feet, their amplitude is smaller and more uniform, and their rate quicker. Moreover, in paralysis agitans the shake precedes the palsy, whereas slight loss of power in walking is usually the earliest symptom of insular sclerosis.*

Diagnosis from tabes, from Friedreich's ataxia, from spastic paralysis and bulbar palsy, probably means only that we must recognise clinically mixed forms of disease in which the main characters of one type are complicated by some usually associated with another. Corresponding coincidences of posterior, lateral, anterior, and bulbar sclerosis with disseminated cerebro-spinal sclerosis have been found *post mortem.* But great caution is required to avoid mistaking Hysteria for disseminated sclerosis in women. Dr Moxon says that he has seen several patients about whom he has for a time been in doubt, but who have before long completely recovered. Moreover, the tremor may be absent, so that the case may appear to be an example of some other spinal affection. Charcot accounts for this by supposing that the symptom in question was present at an earlier period; but Erb refers to several cases in which it was wanting throughout the whole course of the disease. Thus he cites Westphal and Killian as having recorded instances in which the only symptoms were dementia and a more or less diffused paralysis with contractions of the limbs.

* In a typical case recorded in 1875 by Dr Goodhart in a woman of 38, with characteristic lesions after death ('Path. Trans.,' xxvii, p. 17).

Course, prognosis, and treatment.—This remarkable disease usually begins insidiously, with slight giddiness, affections of sight or hearing, weakness in the legs, and difficulty in walking. As an exception it begins more suddenly, perhaps with an epileptiform fit.

The course of disseminated sclerosis is slowly but irregularly progressive. Remissions in the symptoms may occur spontaneously or under treatment. The average duration of the disease is said to be from five to ten years, the most rapidly fatal termination being in one year from the commencement, while the latest has been at the end of seventeen years. Something, however, depends upon the nature of the symptoms. Charcot says that cases in which "spinal" symptoms are alone present may go on for twenty years or longer. If death does not occur in one of the ways already mentioned, it is generally by the supervention of some intercurrent disease, such as pneumonia, pleurisy, dysentery, œdema of the glottis, or (above all) phthisis.

Very few remedies have been found of any service. Charcot thinks that the administration of strychnia or of the nitrate of silver has sometimes influenced the tremor and the weakness of the lower limbs very favourably, but only for a time. Some observers have seen transitory benefit result from the cold-water treatment, from galvanism, or from the subcutaneous injection of arsenic. Some of the drugs given probably do harm. The only rational treatment is to alleviate individual symptoms and to make the patient as comfortable as he can be made.

DISEASES OF THE BRAIN

DUE TO LOCAL LESIONS OF THE CIRCULATION

Introductory remarks—Anatomical effects of obstructive disease of the arteries—Embolism, syphilis, &c.—Red and white softening—Effects of hæmorrhage—Renal disease, atheroma, miliary aneurysms.

HEMIPLEGIA—*Its characters—Diagnosis of the local lesion—Course and sequelæ.*

APHASIA—*Amnesia—Agraphia—Its characters, seat and physiology.*

APOPLEXY—*Symptoms, onset and course—Determination of locality—Its diagnosis from injury, poison, alcohol, pyæmia, urœmia, epilepsy—Diagnosis of the anatomical causes of apoplexy—Treatment of apoplexy and hemiplegia.*

THE brain differs from the spinal cord in its pathology in two important respects. On the one hand, its lateral halves, instead of being closely united and bound up so as to form a slender column within the firm sheath of the pia mater, are to a great extent isolated from one another, and are expanded each into a large independent mass. On the other hand, its arteries, instead of penetrating its substance as minute twigs which pass to both sides indifferently, are large trunks, with well-defined areas of distribution. Consequently, an important group of cerebral affections depend directly upon disease of the arteries of the brain, or upon an interruption of the supply of blood through some of these vessels. And in these very affections the symptoms are in a marked degree unilateral; any paralysis, or spasm, or loss of sensation in the body or limbs is confined to parts on one side of the mesial line. *Hemiplegia*, or loss of power in one arm and the corresponding leg, is generally present in such cases; unless, indeed, the lesion is so severe as to be attended with a rapid annihilation of all the chief functions of both sides of the brain,—a condition which has long been known as *Apoplexy*. Thus the affections in question contrast broadly in their features with those to which the cord is liable, and which, as we have seen, are attended with a paraplegic form of paralysis, and are commonly slow in endangering the vital processes.

The boundary-line is not, however, fixed at the foramen magnum; for the medulla oblongata,—which the physiologist recognises as the continuation upwards of the medulla spinalis,—are subject to at least one of its diseases, that which I have already described under the name of Bulbar paralysis. But the pons is also apt to be the seat of lesions which are due to changes in its arteries, and produce a very fatal form of apoplexy. In fact, one may say that this part of the encephalon refuses to recognise the boundary-line that separates the affections of the brain from those of the spinal cord and bulb. The paralyses which have their seat in it are not necessarily unilateral, but its pathology is to a great extent dominated by that of its blood-vessels, exactly as is the case with the higher cerebral centres.

To avoid the necessity of going over the same ground more than once, we will first give an account of the several lesions of the cerebral blood-vessels,

of their respective causes, and of their anatomical effects. Then we will proceed to discuss the symptoms to which they may give rise; and, finally, we will endeavour to point out how we can diagnose one of these lesions from the others, as well as how they are to be distinguished from affections of a different nature.

The lesions themselves fall into two groups: (1) Those in which there is an arrest of the flow of blood through one or more of the cerebral arteries, and (2) those in which an artery is ruptured, allowing the blood to escape into the brain or beneath its membranes. The former group includes chiefly *embolism*, *thrombosis*, and *syphilitic disease* of the cerebral arteries; the latter is made up of the various forms of *cerebral hæmorrhage*.

I. *Arrest of blood supply through the cerebral arteries.*—This may occur in various ways, which require to be separately considered.

(1) The obstruction may arise *outside the cranial cavity*. I have already alluded to a case of Sir William Gull's in which arteritis deformans of the arch of the aorta led to complete obliteration of the innominate and left carotid arteries at their origins, so that the left subclavian artery alone was left to carry on the circulation in the brain. The patient, a woman aged forty-one, died paralysed and insensible; and certain parts of the cerebral centres were found to be in a state of softening. Some years ago a woman affected with carotid aneurysm was admitted into Guy's Hospital, who had nearly a year before been attacked with hemiplegia and loss of speech. Mr Durham performed the operation of ligaturing the common carotid artery, whereupon she regained to some extent the power of speaking. However, she died soon afterwards, and I made an autopsy and found that the aneurysmal sac extended upwards by the side of the internal carotid artery, and pressed upon it so that until the tension was lowered by ligature of the vessel below, the blood which reached the internal carotid from collateral sources had been unable to pass through it. Much more commonly, it is *after* ligature or compression of the carotid artery that hemiplegia sets in. I have notes of five cases of this kind. In most of them the paralysis was the direct result of the operation; but in one instance it did not occur until three days afterwards, when the patient became suddenly hemiplegic while the dressings were being changed. The suggestion has been thrown out that in those cases in which cerebral symptoms manifest themselves under such circumstances the communicating arteries which make up the circle of Willis may perhaps be abnormally small; but I am not aware that this has been demonstrated anatomically.

(2) *Embolism.*—In the immense majority of cases the cause of arrested circulation in the cerebral arteries lies within the skull; and it is generally the presence of an embolism,—a clot derived from some distant source, and washed into the vessel by the force of the blood-stream. Most frequently, the starting-point of the morbid process is some disease of the valves in the left side of the heart; the patient perhaps has ulcerative endocarditis, or there may be simple vegetations on either the mitral or the aortic valves. In one case, in which I made an inspection in 1877, a rough friable mass of calcareous deposit was exposed upon the surface of the mitral valve, the lining membrane of the heart having undergone erosion, and a fragment of the same material was found wedged in one of the Sylvian arteries.

Sometimes stenosis of the mitral orifice is present, without any projections from its surface; the clot is then derived from the left auricle, which

perhaps contains numerous other thrombi in the recesses between the fleshy columns of its appendix; or the formation of *ante-mortem* coagula in the heart's chambers may be independent of any valvular affection. In very rare instances the primary lesion is in the aorta itself.

We have had at Guy's Hospital two such cases; in one there was a patch of softening thrombus which adhered to a diseased part of the wall of the great artery; in the other ulceration existed. Altogether, I find in the records of *post-mortem* examinations for the last twenty-three years, forty-seven cases in which there was reason to believe that embolism of one of the cerebral arteries had occurred. It is true that in a very large number of them (twenty-one) no clot was discovered in any of the vessels at the autopsy, the interpretation of the cerebral symptoms as due to this cause being based upon their occurrence in persons who had disease of the cardiac valves and upon the presence of such changes in the brain as are known to result from it. All writers admit that anatomical proof of plugging of the arteries is wanting in some fatal cases, especially when life has been prolonged for a considerable period. They suppose that the clot has in the meantime undergone absorption. That this may be the case is likely enough, but I would suggest that another possible explanation is that the spot at which the embolism was impacted may have been the bifurcation of the carotid artery in the neck, or one of its bends within the petrous bone or in the cavernous sinus; or, possibly, even where a vertebral artery is winding round the arch of the atlas,—parts which, I believe, have generally escaped examination. The vessel into which the clot passes is, however, almost always one of the Sylvian arteries. I have notes of twenty-three cases in which embolism of these vessels was found at the autopsy, as against three cases in which its seat was the vertebral artery. The fact that embola entering the carotid are almost always carried into the Sylvian branch is doubtless attributable to the circumstance that its course is more in a line with the trunk than that of the two other branches. Writers have stated that the left Sylvian artery is much more apt to be plugged than the right one, and an explanation for this supposed fact has been found in the difference of the angles at which the innominate artery and the left carotid artery respectively leave the aorta. But it is not confirmed by the observations which have been made at Guy's Hospital, for among twenty-one cases of embolism limited to the Sylvian artery of one side I find that there were eleven in which the left one was affected, and ten in which the clot entered the right artery, a difference too slight to be noticed. I am inclined to think that error has arisen from the statistical collection of miscellaneous cases from Journals and Transactions; the interest attaching to aphasia—a frequent result of embolism on the left side—must have often led to the publication of cases in which that symptom was present, whereas those in which it did not occur have been left unrecorded.

Another point in regard to which my cases are opposed to the statements of certain writers is as to the age at which cerebral embolism is most apt to occur. They speak of it as most frequent in very young adults; but I find that at the same hospital the largest number—12 cases—have occurred between the ages of thirty-one and forty years, as compared with 10 between twenty-one and thirty, 9 between forty-one and fifty, 8 between fifty-one and sixty, 6 between eleven and twenty, and 1 above the age of sixty. There have been rather more males than females among the patients; but this was probably accidental.

(3) *Thrombosis* of the cerebral arteries,—the formation of clots *in situ*, independently of any extraneous source,—is far less frequent than embolism. Nothnagel says that it is generally secondary to disease of the arterial wall, either atheroma, or thickening with calcification. Sometimes it seems to be a direct result of feebleness of the heart's action. He is inclined to think that the vertebral arteries are more liable to be affected in this way than the carotids; and this accords with our experience at Guy's, where there have been three or four cases of thrombosis of the basilar artery or of the posterior cerebrals, but (so far as I know) only one case of a similar affection limited to the middle or anterior cerebrals. In one remarkable instance, however, both the carotid arteries, the middle cerebrals, the anterior cerebrals, and (it is said) the "posterior cerebellar" arteries, were all obstructed by adherent clots. The patient was a man aged thirty-five; and our other cases have been in persons aged thirty, thirty-five, forty-eight, and forty-nine years respectively. It would therefore appear doubtful whether Nothnagel is justified in saying that thrombosis, as a rule, occurs at a more advanced period of life than embolism.

(4) *Syphilitic disease* of the cerebral arteries has only recently become known to pathologists; but it is already seen to be one of the most important effects of the *lues venerea*, although I doubt whether its full clinical significance has as yet ever been fully recognised. The first monograph devoted to it is that written by Heubner, of Leipzig, which appeared in 1874. He assigns to a Danish writer, Steinburg, the credit of having first attributed to an affection of the blood-vessels many of the cerebral symptoms which arise as a result of syphilitic infection; and to Wilks that of having suggested that the change in the arterial coats is one of a special kind, and different from the ordinary atheromatous affection with which it had been confounded. Its histological characters have been determined by Heubner himself. He finds that it begins between the endothelium and the fenestrated membrane. Here a number of cells accumulate, which form a mass that encroaches on the calibre of the vessel. In one instance the fenestrated membrane was in part destroyed, the growth penetrating into the muscular coat. He speaks of this as exceptional; but we have at Guy's Hospital had two cases in which an affection that seemed evidently to have begun in the vertebral artery passed right through the walls of the vessel into the substance of the pons Varolii. Heubner himself points out that with the arterial affection there is often associated a gummatous growth in the loose tissue of the pia mater at the base of the brain; but he regards this as independent. The cells in the intima of the artery presently undergo development, the inner run into spindle-cells arranged transversely, the outer into interlacing stellate cells. Heubner compares this process with the formation of a new wall to the vessel, in support of which view he states that a new fenestrated membrane may actually be generated immediately beneath the endothelium. The new tissue becomes vascular, and this perhaps saves it from undergoing retrograde changes. At any rate it has hitherto been constantly found greyish white and semi-translucent, so that Heubner confirms the statement, which was originally made by Dr Allbutt, that it has scarcely any tendency to caseate, differing in this respect from syphilitic affections of all other parts. I am disposed to think, however, that this may partly depend upon the fact that syphilitic disease of the cerebral arteries has hitherto been observed only in the large vessels at the base of the brain. One can hardly doubt that it must also occur in the

smaller branches, where its effects would be less serious, so that it would be less likely to be seen in an early stage at a *post-mortem* examination. As the channel of the artery becomes narrowed the blood often coagulates in its interior, and thus the circulation through it is arrested. Heubner expresses doubts as to whether the growth by itself ever leads to complete obliteration, independently of thrombosis. Ultimately a process of cicatrisation may take place, the cells developing into connective tissue, and the vessel undergoing conversion into a fibrous cord.

When a single vessel has been affected with syphilitic disease it has much more frequently been the carotid artery, or one of its branches, than the basilar; the proportion given by Heubner being as twelve to one. But in most instances several arteries become diseased simultaneously. In one of Heubner's cases the circulation at the base was interrupted at four distinct points; the left vertebral artery was obliterated, and so were also the basilar, the left middle cerebral, and the origin of the right anterior cerebral arteries. Still there is always a marked difference between the syphilitic affection and atheroma; the latter is commonly diffused pretty uniformly over all the trunks in the immediate neighbourhood of the circle of Willis; moreover, it tends to dilate the affected vessels, and to render them tortuous, rather than to narrow and occlude them.

As might be expected, it is generally at an advanced stage of syphilis that the cerebral arteries become affected. Among the cases collected by Heubner the oldest patient was fifty-one years old, the youngest twenty-two; and the numbers were pretty evenly distributed over the three decennial periods between twenty and fifty years of age. It is to be noted, however, that in all his cases, with scarcely an exception, there was a definite history of venereal infection. Now, syphilitic disease of the arteries of the brain is unlike every other remote effect of syphilis, if it is not apt to occur in persons who are ignorant of having the poison in their system, and who are free from eruptions, nodes, and other obvious signs of its influence. At Guy's Hospital, since the first case recorded by Wilks in 1863, we have had in the *post-mortem* room five other well-marked instances of the affection, besides eight or ten of a more doubtful character. In comparatively few of them was there a clinical history of syphilis, the proof of their origin depending rather upon the presence of other internal syphilitic lesions, such as gummata in the liver or testes, or lardaceous disease of the organs.

Effects of obstruction of cerebral arteries.—The state of the brain-substance, in cases in which there has been arrest of the circulation in one or more of the cerebral arteries, nearly corresponds with what might have been expected from the effects of interference with the arterial blood supply to other tissues. In general it may be said that the affected parts are *softened;* and I may remark that the various affections that I am now describing include perhaps the majority of those cases which by the pathologists of the last generation were classified as examples of "softening of the brain."

The anatomical relations of the vessel which happens to be obstructed of course determine the regional distribution and extent of the morbid change; and upon this, in its turn, depends the nature of the symptoms. With regard to the Sylvian artery, we shall find that the whole clinical aspect of a case differs according as this vessel is plugged at its origin, or a little further on in its course.

Thus it is of great importance to the physician to be acquainted with

certain investigations as to the exact mode of distribution of the cerebral arteries, which have been worked out by two independent observers, Duret in France, and Heubner in Germany. As they point out, the small arteries that alone enter the tissue of the brain form two separate systems, which may be distinguished as those of the cortex and those of the central ganglia respectively. These systems are altogether independent of one another. No anastomoses take place between them; the zone at which they meet within the cerebral substance is situated about an inch and a half below the convolutions. Now, the "central" arteries arise directly from the trunks forming the circle of Willis; they are entirely unprovided with anastomoses. But the "cortical" arteries spring from a network in the pia mater, in which it is said that tolerably free communications exist between the different tertiary branches of the same trunk, and even (in some individuals) between the branches of different trunks. Let us now apply these facts to elucidate the effects of obstruction of the Sylvian artery at different points. That artery, close to its origin, gives off a number of small "central" twigs, which supply the whole corpus striatum (except the inner end of the caudate nucleus), and also the anterior part of the thalamus. It then divides into four terminal branches, of which one is distributed to the third frontal convolution, while the other three pass to the second frontal, the two central, the three parietal, and the three temporal convolutions: the statements of Duret and Heubner are not precisely the same with regard to their exact destination. Thus, when the middle cerebral artery is obliterated beyond the point at which its "central" offshoots arise, the superficial parts of the brain are the only ones to suffer. And since the subdivisions of the vessel in the pia mater anastomose with those of the anterior and posterior arteries, it is not certain that any softening will result. There may be merely a temporary interference with the circulation in the area to which the Sylvian branches are distributed; or a limited part of this area towards its centre may be permanently deprived of its blood supply; or, lastly, almost the whole of the convolutions enumerated above may undergo destruction. Thus Charcot has recorded an instance in which an enormous superficial patch of softening involved the ascending frontal and the ascending parietal convolutions, as well as those of the insula, the central ganglia remaining healthy. The differences in the result in different individuals depends partly upon variations in the extent to which the vessels communicate with one another, partly upon whether the plugging takes place suddenly or gradually. It is, of course, quite possible for a single one of the four terminal branches, such as that to the third frontal convolution, to undergo obstruction apart from the rest.

On the other hand, when the seat of the lesion is at the spot where the Sylvian artery arises from the internal carotid, the central ganglia are concerned. And in their case softening is almost inevitable, because their arteries have no anastomoses; it is only when the closure of the vessel takes place very slowly, as the result of chronic disease of its coats, that collateral channels sometimes seem to develop themselves, so that the blood supply is maintained. It is important to notice that when the main channel of the Sylvian artery is obliterated, extensive morbid changes in the corpus striatum and thalamus are not at all unlikely to occur in a case in which the convolutions entirely escape.

The appearances presented by the affected parts of the brain vary considerably under different conditions. When the patient dies rapidly, the

cerebral substance may look perfectly healthy. This was the case, for example, in a man who was attacked with hemiplegia twenty-six hours after ligature of the internal carotid artery, and who lived only fifty-seven hours afterwards. And I have already observed that where closure of an artery takes place gradually, the regions of the brain that are deprived of their normal blood supply often fail to suffer as much as might be expected. Thus Heubner remarks that in the syphilitic affection of the cerebral arteries the nutrition of the cortex is seldom seriously interfered with, unless, out of the six main trunks that arise from the circle of Willis, two adjacent ones are completely obstructed. In the case already referred to, in which the circulation at the base of the brain was interrupted at four distinct points, there was no softening at all, although one Sylvian artery was obliterated.

Histological effects of obstruction.—When arrest of the flow of blood through a cerebral artery takes place gradually—as in thrombosis and in syphilitic disease—I believe that the morbid changes in the brain, if any occur, always take the form of *softening.* The affected parts are sometimes actually diffluent, being replaced by a milky liquid, which occupies an ill-defined cavity, containing some loose shreds of connective tissue, and perhaps roofed in by the pia mater or by the ependyma of the lateral ventricle. Every intermediate degree of consistence may occur up to a point at which one can hardly perceive any difference from the healthy substance around, until one allows a gentle stream of water to play over the diseased surface, when it soon becomes ragged and assumes a worm-eaten appearance. The colour of softened parts of the brain is very variable; it may be white, or yellow, or even brownish. Writers have been accustomed to describe separate varieties of the affection according to colour. But (except in the case of "red softening," which will be described hereafter) I think it is of great importance to ignore such distinctions, for they have no real existence, and they have already led to great confusion. As a matter of fact, the condition now under discussion is that which most English pathologists call "white softening," but by Rindfleisch it is named "yellow softening;" and each of these two epithets is susceptible of an entirely different application; for the German writer uses the term "white softening" for a morbid appearance in the central parts of the brain in cases of hydrocephalus, and we have been accustomed to designate as "yellow softening" the œdematous state of the cerebral substance that is so often found in the neighbourhood of tumours.

The differences in colour in different cases of softening of the brain depend simply upon the presence of extravasated blood in greater or less quantity, or upon its entire absence. The blood undergoes disintegration with the tissue-elements among which it lies, and under the microscope it is easily recognised in the form of yellow or red granules, or of hæmatoidin crystals. The microscope also reveals in softened brain-substance "compound granule-masses," or "corpuscles of Gluge," often in large numbers. These mulberry-like aggregations of minute granules of fat have already been mentioned as occurring in certain affections of the spinal cord and indeed they are found in many degenerating tissues. In the brain, however, it is still uncertain how they are formed, for Huguénin, in a paper on the subject, admits no less than seven sources for them, namely, the nuclei of the neuroglia, the cells which make up the walls of the capillaries, those of the "adventitia" of the arteries, the nuclei of the smooth muscular fibres

of the vessels, those of the perivascular lymph-spaces, the spindle-cells of the cortex, and very probably its ganglion-cells.

In cases of *embolism*, or after ligature of the carotid artery, softening is, as a rule, the only change presented by the parts of the brain deprived of their blood supply; but these affections have certain peculiarities that seem to modify the appearances which are met with in some cases. One thing is that the obliteration of the artery takes place suddenly. Even after ligature of the carotid artery the corresponding cerebral hemisphere is now and then found in a condition of vascular turgescence, precisely like the early stage of an infarctus. Thus in one case in which I was present at an autopsy made by Dr Wilks, all the vessels on the affected side of the brain —including the veins as far as the longitudinal sinus—were distended and filled with coagula, and the substance of the organ, which was pulpy, was of a dark red colour. So, again, in those cases of embolism that have occurred at Guy's Hospital, a condition of "red softening" is described as having been present, and in two other cases tough yellow masses were found which exactly resembled the wedge-shaped patches that are so common in the spleen and the kidney. "Red softening," indeed, is a change of which the significance is doubtful. It may be ordinary softening as above described in which minute extravasations of blood have taken place in unusual numbers; or perhaps it may be an early stage of "white" or "yellow softening"—the reason for its not having hitherto been seen in cases of thrombosis or syphilis being that in these diseases death does not take place soon enough. But, on the other hand, it may be a true local inflammation, such as we shall describe in a future chapter. Now, it is well known that an embolism often sets up inflammatory processes in the parts around the vessel in which it becomes lodged, so that it is found embedded in a mass of inflammatory thickening; and I have notes of one case in which general meningitis was present, while there were at the same time suppurating emboli in the spleen and kidneys. Again, the coats of the artery itself often become softened, and yield immediately below the seat of obstruction, so that an aneurysm is formed. At least four examples of this have been observed at Guy's Hospital within the last few years; in one of them Dr Goodhart found an aneurysm on the Sylvian artery on each side. It was not indeed proved in every instance that plugging had occurred, but all the patients had disease of the cardiac valves, and in other cases the anatomical evidence of embolism sometimes fails us. Death was in most cases due to rupture of the aneurysm, blood being extravasated in considerable quantity into the pia mater as well as into the substance of the corpus striatum, or even reaching the lateral ventricle. But, in addition, cerebral hæmorrhage has been the cause of death of seven other persons who were affected with ulcerative endocarditis or had vegetations on their mitral or aortic valves; in all probability an aneurysm, itself the result of embolism, was present in these cases likewise, although it escaped notice at the autopsy.

Sir William Gull long ago suggested that it was chiefly in young subjects that intracranial aneurysm should be suspected as a cause of cerebral hæmorrhage; and Dr Church, in tabulating a series of cases of this kind, confined his attention to individuals under twenty years of age. But of nine cases which occurred at Guy's Hospital, and in which the ages of the patients were known seven only were between fourteen and twenty-six, one patient being thirty-four and another fifty years old.

We have already seen that embolism itself is by no means so strictly limited to an early period of life as has been generally supposed.

II. *Cerebral hæmorrhage.*—As a rule effusion of blood into the substance of the brain takes place altogether independently of embolism, and as the result of an entirely different morbid process.

Since the days of Morgagni it has been known that cerebral hæmorrhage is far more apt to occur in or near one of the corpora striata than in any other part of the brain. But certain anatomical facts have now been made out by Gendrin and other observers, which have not only led to a more exact determination of its seat than had before been possible, but have also gone far towards explaining why this spot should be so generally the seat of rupture of an artery. Outside each lenticular nucleus there is a mass of white substance, which is now called the "external capsule." In ordinary sections the continuity of the two structures seems to be as close as between any other portions of the cerebral mass which happen to differ in their colour. But when a brain is hardened it is found that they can be separated from one another with great ease, and apparently, although not really, without any nervous fibres being torn through. Thus the connection between them must be somewhat imperfect. Now, the central branches of the Sylvian artery, which supply the corpus striatum, run upwards for some distance outside the lenticular nucleus before penetrating into its interior. Even on mechanical grounds we can see why those vessels should be more liable to laceration than those of the hemispheres generally. They are much larger than the nutrient arteries of the cortex, they have no anastomosis with one another, and they arise directly from a large trunk, which may almost be said to come in a straight line from the heart. Indeed, Watson long ago pointed out that when injections are forced into the cerebral arteries of the dead body it is especially in the corpora striata that the vessels are apt to give way, and the material injected to be extravasated. Charcot goes so far as to speak of one particularly large lenticulo-striate branch as "the artery of cerebral hæmorrhage." It is true that, as we shall presently see, the rupture of any artery in the brain is always preceded by the occurrence of morbid changes in its walls, but I do not know that this fact deprives of their significance the considerations to which I have just alluded. The blood begins by making for itself a space between the lenticular nucleus and the external capsule. At first it forms a thin layer, but as it increases in quantity it gradually becomes a rounded mass. It now flattens out the claustrum and convolutions of the island of Reil, which lie below and to the outer side of it, it pushes inwards the corpus striatum, and also the thalamus, if it extends far enough backwards. When it accumulates slowly, it may press upon the parts around, so as to form a smooth-walled cavity for its reception. But more frequently it tears up irregularly the white matter of the hemisphere and the outer part of the lenticular nucleus. In some rare cases it reaches the superficial convolutions; far more often it ruptures into the lateral ventricle. Coagulation quickly occurs, and thus at an autopsy one finds a mass of clot weighing four ounces or more. Within the lateral ventricles there is sometimes a red liquid, consisting of blood mixed with the cerebro-spinal fluid; sometimes a clot occupies the side which was first reached by the blood, the contents of the other side being only bloodstained with perhaps a little clot in the cornua. Dr Broadbent has noticed that the middle cornu close to the seat of the hæmorrhage is commonly empty,

having been compressed by the extravasated blood before rupture into the lateral ventricle took place. Not infrequently the third ventricle, the aqueduct, and the fourth ventricle are all filled with moulded coagula exactly fitting their cavities; the blood may even escape along the subarachnoid space so as to reach the exterior of the bulb and of the pons, and the parts at the base, as far as the opposite Sylvian fissure. The fissure on the side of the hæmorrhage is too closely compressed to be able to receive any of it. Indeed, the convolutions of the vertex of that hemisphere are often greatly flattened, so that as soon as the skull-cap is taken off one sees at once which side contains the blood.

Seat of hæmorrhage.—I have before me notes of 96 consecutive fatal cases of cerebral hæmorrhage that have occurred at Guy's Hospital.

In 71 the seat of the affection was in or near the basal ganglia. Now, there are only 4 of these cases in which I find it stated that the blood was confined to the substance of the hemisphere, not having found its way either to the surface or into the ventricles. In 57 it is expressly recorded that laceration into the lateral ventricle had taken place; and in 22 that even the fourth ventricle contained either a clot, or at least a bloodstained fluid. In 6 cases the superficial convolutions were reached. In 6 instances the part into which the blood was effused was behind the thalamus, or, in other words, beyond the limits of the region which I have described as the usual seat of cerebral hæmorrhage. Twice the caudate nucleus was alone affected, and the blood passed direct into the lateral ventricle. The right side of the brain was affected in 36 cases, the left side in 34.

In twelve of the remaining twenty-five fatal cases the seat of the hæmorrhage was in the pons Varolii; in one it was in the right half of the cerebellum. Among the cases of "apoplexy of the pons" there were five in which the blood had escaped into the fourth ventricle; two in which it had oozed out through the convex surface of the pons, so as to reach the base of the brain. In no less than eight of the cases in which there was hæmorrhage in the neighbourhood of the corpus striatum, blood was also effused into the pons; in one instance, three independent hæmorrhages were found in this part. Probably they had occurred as a secondary result of the obstructed respiration caused by the primary apoplexy.

Lastly, there were twelve cases in which the extravasation was into the membranes on the surface of the brain. "Meningeal Apoplexy" has been described by most English writers as a distinct affection; and this is to a certain extent justified by the fact that it is sometimes connected with purpura, anæmia, pneumonia, or blood-poisoning, or is caused by blows or falls on the head. But in the cases to which I am now referring it was associated with exactly the same changes in the heart and kidneys which we shall see to be commonly found with ordinary cerebral hæmorrhage, and there is every reason to believe that the difference in seat was accidental. The quantity of blood was often large; the nerves and other structures at the base of the brain were buried in a thick clot, and the extravasation extended along the Sylvian fissures and in the meshes of the pia mater over the surface of the hemispheres to a greater or less extent, and also in some instances along the subarachnoid space to the spine, and into the fourth ventricle.*

Histological effects of hæmorrhage.—When life has been prolonged for a

* Collecting from various sources 912 cases of non-traumatic intracranial hæmorrhage, I find that nearly 65 per cent. were in the cerebrum, and more than five sixths of these in

few days after the occurrence of cerebral hæmorrhage, the brain tissue round the clot commonly exhibits reactive changes. At first it is reddened, or even ecchymosed; then it becomes œdematous and of a yellow colour, exactly as in a case of tumour of the brain. At a still later period it may be found in a state of softening. Whether suppuration ever takes place round a clot, as was previously supposed, is very doubtful. Sir Thomas Watson relates a case for which he adopted this explanation; but it is not unlikely that the clot, which was found lying in an abscess, was really of later date than the pus.

But cerebral hæmorrhage does not necessarily destroy life, so that there are other morbid appearances, besides those that are seen in cases which prove directly fatal. I find in our records of *post-mortem* examinations at Guy's Hospital twenty-one cases in which the remains of effusions of blood of old date were discovered in one of the basal ganglia or in their immediate neighbourhood. And, in addition, ten of the seventy-one cases of recent cerebral hæmorrhage presented patches which had been of earlier origin. Sometimes all that was left was a soft tawny discoloured spot, containing abundant granule masses and crystals of hæmatoidin; or a tough, flat, fibrous mass, an "apoplectic cicatrix." Sometimes there was a well-defined membranous cyst with shreds of connective tissue and a clear or turbid fluid in its interior. In one instance the cavity was of the size of a walnut; in another it was as large as a bantam's egg. Such appearances have been plainly recognised thirteen or fourteen years after the attack, when the patient has died of some different disease. I do not find that any of our cases enable us to say within how short a time it is possible for a clot in the brain to undergo absorption and for a cyst to take its place. It may be worth while to notice, that in no single instance in which the remains of an apoplectic clot have been found in the brain after recovery, has there been any indication that the blood had made its way into the lateral ventricle. But according to the statements of Rokitansky and Charcot it would seem that in some very rare cases even ventricular effusion is not inevitably fatal.

Ætiology.—With regard to the causes of cerebral hæmorrhage considerable differences of opinion still prevail. In this country the most obvious pathological change found in the bodies of those who have died of effusion of blood into the brain is undoubtedly *chronic renal disease.* I have made an analysis of 116 cases* which came under observation consecutively in the *post-mortem* room of Guy's Hospital; and in eighty-six of them some morbid condition of the kidneys is stated to have been present; in only fifteen out of the whole number is it reported that these organs were healthy. But, strange to say, continental pathologists seem not to have found the kidneys diseased in a similar proportion of cases. Charcot and Bouchard say that interstitial or parenchymatous disease was present in only three among forty-nine cases of theirs in which the state of these organs was noticed, there being, however, thirteen other cases in which the kidneys were "simply atrophied." Nothnagel, in 'Ziemssen's Cyclopædia,' appears to be equally ignorant of the close connection between renal affections and cerebral hæmorrhage, for he

the corpus striatum, thalamus, or adjacent parts; that more than 20 per cent. were meningeal, nearly 10 per cent. in the pons, and about 5 per cent. in the cerebellum.

* This number is made up of 85 cases of hæmorrhage into the basal ganglia or into the substance of the hemispheres (71 recent and 21 old), 12 cases of apoplexy of the pons, and 12 cases of meningeal apoplexy. All those instances were excluded in which there was reason to believe that embolism was the starting-point of the lesion, and also certain cases of meningeal hæmorrhage, which accompanied purpura, or were due to accident.

quotes, almost without comment, the statement of Frerichs that among 241 cases of Bright's disease there were only six in which effusion of blood into the brain occurred. This, indeed, is not so incompatible with the English observations as might at first sight appear. For none of our cases have been examples of the epithelial (parenchymatous or catarrhal) affection of the kidney; and comparatively few of them seem to have presented those conditions of mixed epithelial and interstitial changes which are so commonly the cause of dropsy and other symptoms in persons at the middle period of life. In the great majority the renal affection has been of the "granular" kind, the organs having shrivelled up into mere relics of their former structure, but retaining their red colour unaltered. In a few instances one kidney had undergone destruction from calculous affection of its pelvis, or the two organs had each become converted into a large tumour made up of a congeries of cysts. But it is possible that in such cases the occurrence of cerebral hæmorrhage was merely an accidental coincidence.

The fact that it is the red and granular or "cirrhotic" form of Bright's disease which is commonly found when blood is effused into the brain is of itself sufficient to prove that the hæmorrhage is not a mere result of that increased tension in the arteries generally which is caused by the epithelial as well as by interstitial varieties of renal disease. And I think that all trustworthy observers are agreed that morbid changes in the arterial walls precede their rupture, although there are differences of opinion as to the character of such changes.

Sometimes the arteries which arise from the circle of Willis are affected with an extreme degree of *atheroma*, or have their walls extensively calcified, so that one is inclined to suppose that this is the cause of the hæmorrhage. In the case of a man aged forty, who died in Guy's Hospital under my care in 1869, and who had a hypertrophied heart and granular kidneys, there was a small aneurysm on the left middle cerebral artery which had given way and poured blood into the Sylvian fissure, while there was also a large clot outside the corpus striatum, with laceration into the lateral ventricle, apparently of independent origin. But in most cases pathologists have until recently failed to find the ruptured artery, and the vessels at the base of the brain and their branches often seem to be healthy.

In 1866, however, Bouchard discovered, in a case of cerebral hæmorrhage which had occurred in the wards of M. Charcot, two small aneurysms upon one of the vessels. And since that time these two French observers have collected no less than seventy-seven cases of the same kind, in every one of which they say they have demonstrated the presence of what they term *miliary aneurysms*. These are described as minute globular or fusiform swellings, from one fifth of a millimètre to one millimètre ($\frac{1}{25}$th to $\frac{1}{125}$th of an inch) in diameter, so as to be plainly visible to the naked eye. They are sometimes very soft, sometimes firm and elastic, sometimes hard like grains of sand. In colour they are purple, or reddish brown, or greyish, according to the thickness of their walls, and the state of their contents, which may be either fluid blood or clot of greater or less age. They are generally multiple, but sometimes only two or three can be discovered in the whole of the brain after most careful search; sometimes as many as a hundred have been counted. They are most conspicuous upon the surface of the convolutions and in the sulci between them; a favourite seat for them is the deep layer of the grey matter or the line of junction between it and the white. But they may also be found in the thalami and corpora striata, in the pons,

and elsewhere. MM. Charcot and Bouchard themselves admit that, when an extensive effusion of blood into the brain has occurred, it is difficult to find the miliary aneurysm the rupture of which they suppose to have led to the hæmorrhage. The best way is to place the affected hemisphere in a basin, and to change the water frequently by tilting, without shaking it. After a time the clots become loosened and float away, leaving a number of little bloody masses connected by vascular filaments with the brain tissue beneath. Most of these, when magnified, show only the open ends of vessels, torn across secondarily by the extravasated blood, and covered with little caps of fibrin that require to be carefully distinguished from actual aneurysms; but at length some are found which contain spherical ampullæ, continuous with small arteries and split on one side.

It is evident that the discovery of MM. Charcot and Bouchard affords only a partial explanation of the pathology of cerebral hæmorrhage, since miliary aneurysms are not found with especial frequency in those regions of the brain which we have seen to be so generally the seats of effusions of blood. And, indeed, the French observers themselves point out that the lesion which they describe is after all but one of the effects of a diffused morbid change in the cerebral arteries,—a chronic "sclerous periarteritis," —consisting in thickening of the arterial sheath and of the adventitia, with overgrowth of nuclei, while the muscular coat undergoes atrophy. At first they supposed that this condition was peculiar to the vessels of the brain and occurred only in old people. But another observer at the Salpêtrière, M. Liouville, has since demonstrated the occurrence of a similar affection in the arteries of other parts, and even speaks of having found miliary aneurysms beneath the mucous membrane of the œsophagus and upon the surface of the heart. They have also been detected, although rarely, in young adults. In other words, we are brought by their investigations to almost precisely the same point as by the observation of Sir William Gull and Dr Sutton on "arterio-capillary fibrosis." Indeed, it seems clear that the French and the English pathologists have been studying the same morbid process, and their general agreement goes far to prove the accuracy of their conclusions. It is strange, however, that MM. Charcot and Bouchard should have failed to notice the affection of the kidneys on which Gull and Sutton have laid so much stress.

I think that there can be no doubt that the diffused change in the blood-vessels, to which reference has just been made, is often the cause of an impairment of the nutrition of the brain, attended with an obvious softening of its substance, which must greatly increase the risk of arterial rupture. Charcot and Bouchard quote Rochoux as having first promulgated the doctrine of a *ramollissement hémorrhagipare*, which was further developed by Todd; but they maintain that the only "softening" that bears any relation to cerebral hæmorrhage is the secondary affection which we have seen to be of frequent occurrence in the tissue round a clot. I find, however, in the records of *post-mortem* examinations at Guy's Hospital no fewer than twenty cases in which localised patches of white softening were found in different parts of the brain,—sometimes in the corpora striata or thalami, sometimes in the pons, sometimes in the hemispheres. Some of the patients had suffered from gout; a large majority of them had granular kidneys and hypertrophied hearts; there is not a single instance in which the kidneys are said to have been healthy. In the ages at which they proved fatal, and in the circumstance that the large majority of the patients were males, these cases

corresponded exactly with those of cerebral hæmorrhage; and I can feel no doubt that it was a mere accident that death occurred before any of the arteries in the softened patches gave way.

Cerebral hæmorrhage does not occur with equal frequency in the two sexes or at different periods of life. Men are much more liable to it than women. I find that of 113 consecutive fatal cases at Guy's Hospital, 82 occurred in males, only 31 in females.

Among 110 of these cases, in the reports of which the ages of the patients are stated, there were 6 between twenty-one and thirty, 18 between thirty-one and forty, 37 between forty-one and fifty, 29 between fifty-one and sixty, 17 between sixty-one and seventy, and 3 above the age of seventy. These figures do not altogether correspond with the statements of writers in general, who make the frequency of apoplexy increase up to the age of seventy or even eighty years,—not only relatively to the numbers of persons living at different ages, but also absolutely.* It may be that men at a middle period of life are more likely than those who are older to be brought to the hospital when attacked with apoplexy, because their relatives are not so apt to suppose that the case is hopeless.

Whether cerebral hæmorrhage is of more common occurrence in men with short, thick necks and florid faces than in those who are of a different build, is perhaps doubtful. But there can be no question that gout is indirectly a very important factor in its causation, by setting up granular disease of the kidneys. And gout is notoriously infrequent in persons who are thin, as compared with those who are stout and plethoric.

Cerebral hæmorrhage sometimes occurs first while the patient is making some violent effort, or subjecting his vascular system to an excess of pressure, so that one can hardly help regarding this as the exciting cause of the attack. Thus two of the patients lately in Guy's Hospital had been running to catch a train; another was a woman who had recently suffered from sea-sickness in crossing from France. All writers mention emotions, violent efforts, cold baths, straining at stool, prolonged laughing, coughing, or sneezing, indulging in stimulants, as capable of bringing on the rupture of an artery in the brain, provided that the vessel is in the diseased condition which seems to be a necessary antecedent of hæmorrhage. Among labouring men many are attacked while at work; but one must not forget that this takes up a great part of their daily lives. Moreover, cerebral hæmorrhage occurs in a large number of persons during sleep, when the pressure in the cerebral vessels is supposed to be particularly low. Intoxication by alcohol seems to be a very frequent cause. A striking illustration of this (although the hæmorrhage was not of the usual kind) came under my notice some years ago: a man who had a severe injury to the head six months before, got drunk two days in succession, and on the third day was taken in a fit, became comatose, and died thirteen days afterwards. A quantity of blood was found effused in the arachnoid cavity; but the convolutions also showed several tawny patches of superficial softening, which were no doubt effects of bruising of the brain at the time of the accident; and it seemed to me clear that vessels in one or more of these patches had been the source of the recent hæmorrhage, having

* Gintrac's 658 cases (excluding meningeal apoplexy), as quoted by Bastian, give for the corresponding ages: under 20, 6 per cent.; 21—30, 7 per cent.; 31—40, 11 per cent.; 41—50, 15 per cent.; 51—60, 20 per cent.; 61—70, 23 per cent.; and above 70, 19 per cent.

given way in consequence of the disturbance in the cerebral circulation to which the alcohol had given rise. Sir Thomas Watson refers to two cases in which persons were attacked by hemiplegia while in the act of sexual intercourse; and Dr Wilks recently mentioned to me a similar instance.*

The symptoms of these various affections of the cerebral arteries present many points of resemblance, but they also differ in several important respects. It is impossible for us to enter upon their study without having first considered the nature and significance of certain phenomena promising the highest clinical interest, each of which may occur as the result of any one of the arterial diseases in question. I refer to *Hemiplegia*, *Aphasia*, and *Apoplexy;* and I now propose to consider them separately, passing on afterwards to consider the different ways in which they may be grouped together, and how far it is possible to distinguish from one another the various conditions which may give rise to them.

Hemiplegia.—It has already been mentioned that one of the leading effects of diseases of the brain—at least of such diseases as are confined to one lateral half of the organ—is *hemiplegia*, or loss of power in the arm and leg and certain other parts of the body on one side. Now, this form of paralysis presents certain peculiarities which require careful study.

Motor symptoms.—In the first place, it is to be noted that the limbs which are affected are always those on the side opposite to the lesion in the brain. This, as is well known, depends upon the fact that the motor columns cross over in the anterior pyramids: those which emerge from under the right half of the pons Varolii passing down into the left half of the spinal cord, and *vice versâ*. Thus any unilateral affection of the nervous centres, situated above the decussation of the pyramids, if it causes paralysis at all, invariably causes paralysis of the opposite side. There are, indeed, some cases on record which have been supposed to be exceptions to this rule, and these have been marshalled forth by Dr Brown-Séquard, who believes that they establish the possibility of the occurrence of hemiplegia on the same side as the lesion on which it depends. But, whatever the absolute number of cases of this kind that can be collected from medical journals and other sources, their relative frequency is altogether insignificant in comparison with that of those cases on which the rule of decussation is based. And I entirely agree with those critics who think that all the supposed exceptions are mistakes. In some of them it is most likely that the reporters have written "right" for "left," or "left" for "right," in making their notes. Everyone who has done much case-taking will admit how very easy it is to fall into this error; and I may mention that our *post-mortem* records at Guy's Hospital contain at least two cases in which it is stated that the brain disease was on the same side as the paralysis. One of these is in the handwriting of Dr Wilks, who certainly must have been greatly struck by such a fact, if he had observed it. But he leaves it entirely without comment, and I think there can be no doubt that it was a slip of the pen. In other cases, in which pathologists have deliberately thought that they had made a similar discovery, it is probable that there really were two lesions; one, the more conspicuous, on the same side as the hemiplegia; the other, on the opposite side—perhaps a minute spot of softening—which may have really

* The most severe case of apoplexy followed by hemiplegia which I have ever seen recover, occurred under these circumstances in a man fifty years of age.

caused the paralysis, although altogether overlooked at the autopsy. I was once present at a *post-mortem* examination when such a mistake was very nearly committed.

I have spoken of this form of paralysis as affecting the arm and the leg; and it is important to note that there are definite limitations of the extent to which other parts of the body are capable of being paralysed in what may be termed "common cerebral hemiplegia"—the *hémiplégie cérébrale vulgaire* of Charcot. The nature of these limitations can be most conveniently studied in the case of the facial muscles. Commonly all that is noticeable is a little loss of expression about the mouth, which may appear to be drawn over to the opposite side. Todd ascribed this to impaired power of the buccinator muscle (which he believed to be supplied by the fifth nerve), and maintained that the facial nerve escaped altogether. Such a view, however, is quite untenable. The right doctrine appears to me to be that which Dr Broadbent suggested in 1866 in an article in the 'Medical and Chirurgical Review.' It so happens that in 1860 or 1861 I had read before the Physical Society of Guy's Hospital a paper in which I had developed the same theory; and I may be allowed to quote from this communication, which is now before me, although, as I did not print it, the priority rests with Dr Broadbent. The case on which it was founded was that of Henry W—, admitted into Guy's Hospital on February 20th, 1860, under the care of Dr Gull, for right hemiplegia, which was almost complete. The state of his face was very interesting. When told to shut his eyes he could close them both. But whereas he could wink with the left eye alone, he could not do it with the right alone; and when I asked him to compress the eyelids firmly I found that the left orbicularis palpebrarum alone obeyed the will, the right one merely bringing the upper and lower lids into contact, without any wrinkling of the skin. This, and the further observations which I made on the same patient led me to propound the law that "those movements, which are performed in harmony by the two sides of the face or body remain unimpaired in hemiplegia." Dr Broadbent afterwards laid down the same law in very similar terms, and it has received general acceptance. The exemption of movements which are habitually bilateral (but not necessarily performed by corresponding muscles of the two sides) is seen in the case of the muscles of the eyeballs, of the masticating muscles, and of those of the neck, chest, and abdomen, all of which retain their power.

It is of course to be understood that hemiplegia from a lesion of one side of the brain is not necessarily so complete as to present a maximum loss of power. Since the year 1860 I have not myself met with a second instance in which the affection of the facial muscles could be so accurately determined in accordance with Dr Broadbent's rule. Not infrequently the face escapes entirely. Sometimes the leg can be moved perfectly, while the arm is completely paralysed. Almost invariably, when recovery takes place, the patient regains power in the leg earlier than in the arm; he may perhaps be able to walk with a crutch at a time when he can scarcely move the fingers at all, and if any part remains permanently paralysed it is said to be the upper limb. These facts are commonly attributed to the circumstance that the movements of the arm are more independent than those of the leg. It is regarded as a further illustration of the rule laid down in the last paragraph that the limb of which the movements are more highly specialised should be the more constantly and the more persistently affected.

I must confess that this explanation seems to me unsatisfactory, and I have always been inclined to think that a certain part of the fan-shaped expansion of the crus cerebri may be uniformly more damaged than other parts by the lesions that cause hemiplegia. It would certainly accord well with anatomical facts if the innermost fibres of the internal capsule and corona radiata should often escape, since they seem to lie outside the area to which the branches of the middle cerebral artery are distributed*.

On the other hand, Dr Broadbent's law must not be taken to mean that one can never make out any loss of power whatever in muscles which are used in association with muscles of the opposite side. All that we are justified in saying is that there is in no case anything like complete paralysis of such muscles. The tongue, for instance, is very commonly involved to some extent, so that when it is protruded from the mouth its tip is more or less distinctly turned towards the side on which the limbs are paralysed, and the patient often articulates very indistinctly and mumbles in his speech; so much that he may in some cases be unintelligible.

I may here remark that it was formerly considered difficult to understand why the hypoglossal nerve and the portio dura—arising as they do above the decussation of the anterior pyramids—should nevertheless resemble the nerves of the limbs in showing impairment of function on the side opposite the lesion in the brain. I believe that all physiologists are now agreed that this depends upon the fact that fibres which pass to the roots of those nerves undergo an independent decussation within the substance of the medulla oblongata or pons.

Again, it has been proved by several independent observers that the chest sometimes does not expand so fully on that side on which the limbs are paralysed, at least when a deep breath is being taken. Dr Walshe ('Lancet,' 1849) determined this by actual measurement. Dr Broadbent himself points out that when a hemiplegic patient is made to raise himself from the recumbent posture by his abdominal muscles alone, the rectus on the paralysed side can be felt to act somewhat less powerfully, and a little later, than on the healthy side. So also, if he is told to close the jaws firmly, the masseter and temporal muscles do not contract quite simultaneously or with equal force on the two sides.

Dr Hughlings Jackson thinks that it is possible for a lesion of one hemisphere to produce a complete paralysis of all four limbs, but I am not aware on what evidence this opinion is based, or that any cases of universal loss of power have been recorded from an affection of such a nature that it could not possibly have compressed nor interfered with the opposite side of the brain. It is true that, as Dr Jackson many years ago pointed out, patients suffering with aphasia, from disease situated above the corpus striatum on one side, are not rarely altogether unable to protrude the tongue from between the teeth. I have several times observed this, and it seems clearly to be an instance of double paralysis from a unilateral lesion, but I must confess I should be inclined to attribute it to an "inhibitory influence" transmitted downwards upon the associated nuclei of the ninth nerves in the pons, rather than to believe that both sides are really "represented" in each cerebral hemisphere—which is the hypothesis suggested by Dr Jackson to account for the supposed occurrence of a general paralysis of all the limbs as the result of an affection of a single hemisphere ('Brit. Med. Jour.,' 1874).

* See on this point Mr Horsley's anatomical explanation ('Lancet,' vol. ii, p. 9, 1884).

In some cases of hemiplegia, the eyes, instead of being directed straight forward, are turned to one side, and this side is always opposite to that of the paralysis, so that, for the purpose of artificial memory, one may say that the patient is *looking towards the lesion in his brain.* The "conjugate deviation" of the eyes is by some writers regarded as due to a paresis of certain ocular muscles, the external rectus on the side of the paralysed arm and leg, the internal rectus on the other side. Others have supposed it to depend upon spasm of the antagonist muscles, and thus to be analogous to the state of "early rigidity" which will presently be described. But the face also is sometimes drawn over, away from the hemiplegic limbs, and then Prévost has found that the sterno-mastoid and the trapezius muscles are not necessarily in a state of contraction. Moreover, Dr Jackson has pointed out that when "early rigidity" comes on in the arm and the leg, the "conjugate deviation" becomes reversed, the eyes being now directed towards the side of the paralysis; a case in point occurred at Guy's Hospital in 1867 in the practice of Dr Rees. Thus it seems clear that this remarkable symptom is not of a spasmodic nature, and I think that Vulpian and Prévost are probably right in supposing that it represents, in a rudimentary form, the rotatory movement (*mouvement de manège*) which is so commonly developed in animals after unilateral injuries to the head, for the circles in which most animals revolve always lie on that side on which the lesion is situated. Indeed, a woman under the care of Charcot for left hemiplegia with conjugate deviation of the eyes, was several times found lying across her bed with her head to the right and her feet to the left; and another of the patients whose cases are related by Prévost showed a tendency to turn round like a top when placed upright. This writer has proved that the occurrence of conjugate deviation of the eyes is not limited to affections of any one particular region of the brain; among fifty-eight cases which he relates, there were four of meningeal hæmorrhage, and four others in which the superficial convolutions were alone diseased. It is generally transitory —lasting only a few hours or a day or two—but sometimes it persists for several months.

We have seen that in "common cerebral hemiplegia" there is never a complete paralysis of any one of the cranial nerves, but it sometimes happens that such an affection of the third or of the seventh nerve is associated with hemiplegia, and then the seat of the disease can be determined with more than usual exactness. The facial nerve may be affected in two different ways. On the one hand the lesion, if it is situated in the upper part of one lateral half of the pons, may destroy the facial nucleus, and so cause a total loss of power in the face on the same side on which the arm and the leg are paralysed; and since the nucleus of the sixth nerve is very close, paralysis of the external rectus may perhaps be present in addition. On the other hand, an affection of the lower part of the pons on one side may cut the fibres of the facial nerve as they are passing across the motor tract, after they have decussated on their way outwards from their nucleus. The paralysis of the face is then on the opposite side to that of the limbs. It might even happen that an extensive unilateral lesion of the pons should cause paralysis of both sides of the face by destroying the nucleus of one facial nerve, and the fibres of the other one.

Again, association with hemiplegia of paralysis of the ocular muscles supplied by the third nerve shows that the seat of disease is the crus cerebri. The muscles in question are those affected on the side opposite to the

paralysed arm and leg. An instance of this has been placed on record by Dr Hermann Weber ('Med.-Chir. Trans.,' vol. xxviii). A man, aged fifty-two, was attacked with paralysis of the right side of the body and of the muscles supplied by the left third nerve; he died after two months, and an oblong clot of blood was found in the left crus. Following Gubler, recent French writers have given a special name, that of "*Hémiplégie alterne*" to cases in which either the facial or the third nerve is affected on the side opposite to the limbs. This expression has been translated into English as "alternate hemiplegia," but I think it is not worthy of adoption, since it is likely to be misunderstood, and is not really necessary. "Crossed hemiplegia" is a better term.

Sensation.—Statements that *anæsthesia* is absent in hemiplegia appear to have been based upon observations made on cases which were already of long standing. There is every reason to believe that anæsthesia is generally present in recent cases, although its recognition may be difficult, or even impossible, in patients whose consciousness is much obscured. It is, however, far more transitory than motor paralysis, often passing off in a few days, and sometimes still earlier. In this respect lesions of the brain exactly resemble those of the spinal cord and of the peripheral nerves (cf. pp. 397, 436). Subjective sensations of formication and numbness are frequently associated with partial anæsthesia, and sometimes there is a condition of hyperalgesia, or increased susceptibility to painful impressions. Sometimes, too, spontaneous pains are experienced in the joints on the affected side, and particularly in the shoulder-joint. In slight cases of hemiplegia sensation is apparently unaffected, and even in the most profound it is never completely abolished. The hands and feet suffer more than the proximal part of the affected limbs, and the face least.

Trophic changes.—Hemiplegic limbs are often reddened and warmer than those of the opposite side—a difference which is attributed to interference with the functions of the vaso-motor nerves. The inequality of temperature commonly amounts to something less than one degree Centigrade; after some months it ceases to exist. In some cases there is excessive sweating; sometimes a slight œdema is discoverable. I remember one case in which dropsy (of renal origin) was limited to the arm and leg that were paralysed. Charcot lays stress on the frequency of rapid sloughing of the skin over the gluteal muscles—an acute bedsore, as he terms it. One can hardly suppose that, in cases of this kind, there is much difference in the amount of pressure upon the two sides of the buttocks; and he regards the affection as a more or less direct effect of an interference with the nutrition of the tissues, caused by irritation of a particular region of the brain.

Sometimes contractions of the paralysed muscles are less readily excited than in the healthy side; sometimes, but very seldom, more readily. In the latter case, some amount of tonic spasm is always present; the elbow is generally flexed and resists extension; the fingers are bent in upon the palm of the hand. This condition was described by Dr Todd under the name of "early rigidity," and was attributed by him to laceration of the surrounding healthy brain-substance by effused blood; and this explanation has been adopted by most subsequent writers, although I doubt whether one could demonstrate that there is more injury to the nervous tissues than in cases in which the muscles remain flaccid. Durand-Fardel, indeed, ascribed the occurrence of tonic spasms in hemiplegic muscles to the escape of blood

into the lateral ventricle; but it does not appear that any definite facts can be adduced in favour of such a view. So long as the muscles are in a state of early rigidity they do not waste; and subsequently only undergo atrophy from disuse. They do not lose their faradic contractility nor show any other signs of R.D. Rigidity only occasionally comes on early—more often it supervenes as a late contraction of the limbs which will be referred to again under the sequelæ of hemiplegia. Trophic changes in the skin are very rare.

Seat of the lesion.—There is still something to be said with regard to the *regional diagnosis* of hemiplegia due to disease situated in the parts of the brain which are higher than the crus cerebri. And first it has been a moot question whether paralysis of the arm and leg can be caused by a lesion limited to the white substance of the hemisphere, or to the convolutions, or affecting both structures to the exclusion of the basal ganglia. Mr Hutchinson has insisted on the frequent occurrence of this symptom in cases in which meningitis appears to be limited to one lateral half of the brain, as so often happens after surgical injuries, but then it is difficult to say how far the inflammatory process may extend into the interior of the organ. Until quite recently, the numerous instances in which extensive destruction of the superficial parts of the cerebrum has been followed by no definite signs of loss of function have been regarded by most physiologists as proofs that hemiplegia could not result from such an affection. And even when certain experiments made by Hitzig and by Ferrier (to which I shall more fully allude in the next chapter) had shown that irritation of particular convolutions caused movements in the opposite limbs, it was still held by many observers that no disease of these convolutions could produce a permanent paralysis, in accordance with a distinction, laid down by Dr Hughlings Jackson, between the effects of "discharging" and those of "destroying" lesions. These experiments, however, have led pathologists to reopen the question; and they have also greatly facilitated its investigation, by reducing to comparatively narrow limits the area within which cortical affections causing paralysis are to be sought. The result has been that certain French and German physicians have found that diseases of the upper parts of the ascending frontal and parietal convolutions, and of the adjacent parts of the frontal and of the superior parietal convolutions, may, if sufficiently extensive, give rise to permanent hemiplegia although the corpus striatum remains perfectly healthy. I have already, at p. 558, described softening of this particular region of the brain as an occasional result of obstruction of the Sylvian artery, beyond the origin of its "central" branches, whether by thrombosis or embolism. Charcot speaks of having made a collection of cases of this kind. The paralysis seems to have been undistinguishable from that caused by a lesion of the basal ganglia; it is particularly mentioned that the consecutive degeneration of the spinal cord was present which will be described a little further on. On the other hand, Charcot has found that similar affections of the sphenoidal and occipital lobes caused no permanent hemiplegia.

Indeed, it is possible that we may hereafter be able to carry the matter still further, and to localise in particular convolutions the lesions causing paralysis of different parts of the body. This question, however, will be more conveniently discussed in the next chapter.

As regards the possibility of diagnosing what part of the basal ganglia is the seat of disease, Charcot maintains that, whenever a lesion is limited

to one of the grey nuclei of the corpus striatum (the lenticulus or the cauda), the hemiplegia is always transitory and very often incomplete; whereas if the internal capsule is involved it is commonly complete and persistent. But he destroys much of the force of this remark by adding the obvious statement that neither the nucleus caudatus nor the nucleus lenticularis is ever *wholly* destroyed without other parts being affected at the same time. According to this writer the limitation of paralysis to a single limb is never due to the fact that the morbid process is confined to a special seat within one of the ganglia. Dr Jackson's way of expressing the same doctrine is by saying that "the whole corpus striatum is represented in miniature by every single part of it." And Dr Ferrier found that irritation of this part of the brain by faradic currents caused a general contraction of the muscles on the opposite side of the body, without its being possible to differentiate individual movements. But in spite of these high authorities I cannot help thinking it is possible that, at least in the internal capsule, the fibres which belong to the upper limb may hereafter be distinguished from those that pass to the lower limb. We shall hereafter see that Hitzig and Ferrier state that the cortical centre for the leg-muscles lies nearer the median plane than that for the arm-muscles. It seems to me a fair inference that if there be a distinction of fibres within the internal capsule, those for the leg should be situated further inwards (or forwards) than those for the arm. Now, it is certain that in cases of cerebral hæmorrhage the inner and fore part of the capsule often escapes laceration, when the outer and hinder part is torn through. May not this be the reason why the upper limb so often remains powerless after the patient has regained the use of his lower limb? And may we not attribute to the same cause the fact that the arm sometimes becomes paralysed alone, the leg escaping altogether?

Charcot himself thinks that a broad line of distinction must be drawn between affections limited to the anterior two thirds of the internal capsule and those which involve its posterior third. In the former case there is no loss of sensation, in the latter there is hemianæsthesia, affecting the same side as the paralysis. He even believes, in opposition to some other authorities, that a lesion limited to the posterior third of the internal capsule may cause loss of sensation without any loss of power. The anæsthesia affects not only the limbs of one side, but also the corresponding half of the body and of the head. It includes an insensibility to pain and to changes of temperature, as well as to tactile impressions. It extends to the deeper parts and also to mucous surfaces; the patient feels nothing when his muscles are made to contract by the application of a current of electricity. It affects the special senses of hearing and smell and taste.

In these and certain other cases hemiplegia impairs vision, but there is difference of opinion as to the kind of imperfection which results. According to Dr Hughlings Jackson this is "lateral hemiopia," corresponding halves of each retina being affected in such a way that the patient is unable to see towards the paralysed side. Dr Jackson, in 1875, had seen some thirteen cases of hemiplegia attended with the symptom in question. In one instance, in which the paralysis was on the left side, a *post-mortem* examination was made by Dr Gowers, who found a single lesion of the right thalamus, the posterior half of which was softened and of a greyish-yellow tint ('Lancet,' May, 1875). The hemiopia had been of the most marked description; the man had sometimes seen only half of a word, reading "land" for "midland," and remarking to his son that "Liver" was a queer name, when it really

was "Oliver." In opposition to the statements of Mandelstamm, Michel, and others, I am still inclined to adhere to the view that at the chiasma only the inner parts of the optic nerves decussate. This view, to which I shall refer in fuller detail elsewhere, seems to afford a satisfactory explanation of the occurrence of this "homologous lateral hemiopia."

Charcot denies that such is the affection of sight which forms part of hemianæsthesia. On the contrary, he maintains that there is a "crossed amblyopia"—an impairment of vision in both halves of one eye—the one on that side of the body which is deprived of sensation. He says that the acuteness of vision is often diminished by one half or even in a still higher degree. The visual field is generally narrowed on all sides, but for some colours the range is far more limited than for others. The first to be lost is violet, then, in succession, green, red, orange; the colours for which vision is retained longest are yellow and blue, but even they may at last be indistinguishable, so that every object appears of a brown colour, as if painted in sepia. If Charcot's statements are correct, the only possible explanation seems to be that which he gives, namely, that there is a second decussation of the optic tracts where those fibres cross over which failed to do so in the chiasma, and that this is situated beneath the corpora quadrigemina. The only way in which Dr Jackson's observations can be reconciled with such a hypothesis is to suppose that in his cases the disease involved one optic tract, but in the account of the autopsy made by Dr Gowers it is expressly stated that the crus of that side was unaffected. Charcot even quotes a case of Dr Bastian's, in which a lesion limited to one of the anterior corpora quadrigemina caused crossed amblyopia and not lateral hemiopia.

After-effects.—Hemiplegia is often recovered from, the patient gradually regaining the use of his limbs, until at length no difference can be made out between the two sides. I have already remarked that, as a rule—to which there are very few exceptions—the leg recovers before the arm; the patient may be able to walk quite well with a crutch at a time when the hand is still motionless. Trousseau, however, relates two instances in each of which power was restored in the arm earlier than in the leg, and he maintains that when this is the case the prognosis is particularly unfavourable.

In many cases hemiplegia is permanent. The patient may for the rest of his life be utterly unable to move any part of the upper limb, or he may regain power in it to a greater or less extent. In almost every case of this kind the affected muscles pass into a state of contraction, which is commonly known as "late rigidity." Bouchard found that it was abent in only one out of thirty-two cases that he examined. It generally consists in a flexion of the various joints; the elbow is bent at nearly a right angle, the wrist is pronated and folded on the forearm, and the fingers are drawn in upon the palm of the hand, so that the nails may penetrate the skin and produce painful ulcerations. Very much more rarely, a position of extension is assumed; the elbow may then be straight, the wrist may be thrown back, the fingers may assume the griffin's-claw attitude, which I have described at p. 508 as occurring in progressive muscular atrophy. The leg is less apt than the arm to become contracted, even when it remains paralysed; if at all, the hip- and the knee-joints are flexed, and the heel drawn up. This form of rigidity sets in so gradually that the exact period at which it begins cannot be determined. Bouchard speaks of a case in which

it was present in a marked degree two months after the apoplectic attack which had caused the hemiplegia. Sometimes one can overcome the spasm by a little traction upon the affected parts; sometimes it resists the application of an amount of force which causes the patient severe pain. The muscles are generally much wasted, and feel like tight cords beneath the skin; but this atrophy is much later, and seldom so complete as that which follows anterior poliomyelitis.

Until recently it was supposed that late rigidity was caused either by the slow contraction of an apoplectic cyst, or by an inflammatory process in the surrounding brain-substance. But in 1866 Bouchard suggested, in the 'Archives Générales,' that it was probably an indication of the supervention of certain changes in the lower parts of the cerebro-spinal axis, which had been pointed out by Türck in 1853, and had been previously noticed by Cruveilhier as occurring after unilateral lesions of the brain. These changes are to some extent visible to the naked eye; the crus cerebri is smaller than on the healthy side, and flattened, and presents a grey streak towards its inner edge; the corresponding half of the pons may be flattened; the anterior pyramid is of a greyish colour and wasted, so that the decussation of the pyramid is more conspicuous than usual. Below this point there is no marked alteration on the surface, but transverse sections of the spinal cord may show, in the lateral column of the side *opposite* to the lesion in the brain, a triangular grey patch, which gradually diminishes in size, but is traceable down to the lumbar enlargement. Or it may be only after hardening and staining that the change is discoverable by the microscope. It is found to consist, not only in a degeneration of the nerve-fibres which pass down the cord from the brain, but also in the formation of a delicate new connective tissue. In other words, there is a sclerosis of a definite tract of the lateral column, which, however, also contains a number of nerve-fibres which probably arise from the spinal nuclei, and therefore escape the degenerative process. The condition is that of secondary spastic paralysis from descending sclerosis of the crossed pyramidal tracts (p. 488).

Bouchard's theory is that these unaltered nerve-fibres become irritated and cause the contracted state of the muscles. In support of it he adduced the analogous rigidity of the lower limbs which occurs when the upper part of the spinal cord is compressed (see p. 421). Charcot has since developed the same doctrine still further, and maintains that the sclerosis may spread as an independent affection to the anterior grey cornua, causing the muscles to become again flaccid and to undergo a rapid degenerative atrophy; or to the posterior cornua, inducing a partial anæsthesia; or, lastly, to the opposite lateral column, giving rise to a contraction of the other lower limb, as is supposed to have occurred in a case of Dr Bastian's.

More recently, however, Hitzig has proposed a very different explanation of "late rigidity." He points out ('Arch. f. Psych.,' 1872) that prolonged rest is followed by a relaxation of the contracted muscles; that, for instance, after a night's sleep, paralysed limbs are commonly flaccid and supple, and remain so until the patient begins to exert the opposite limbs in getting out of bed, or in some other way. Again, he has observed that voluntary efforts with the unaffected arm often cause an increase in the rigidity of the affected arm. In one of his cases, as soon as the patient was made to lift a heavy weight with his left hand, the thumb and forefinger of the paralysed right hand became quite stiff, although they had before been free from spasm. He supposes that the contractions of hemiplegic limbs represent an

excess of those co-ordinated movements in distant parts which naturally accompany every action of the body. He assumes that the spinal centres are in a state of irritation, and that, as a consequence, the movements in question become far more marked than under normal conditions. In further illustration of his meaning he instances the well-known fact (of which I remember to have seen an example) that patients who are altogether unable to move a hemiplegic arm by any voluntary effort sometimes have it thrown violently into the air when they yawn or stretch themselves.

To decide between the rival theories of Bouchard and of Hitzig would be very difficult. And I am the less disposed to attempt it, because I shall hereafter have to admit that there is a large number of other spasmodic affections of which the seat and nature are still altogether unknown.

Moreover, I must draw attention to the fact that "late rigidity" bears a close relation to certain forms of mobile spasm which may also present themselves in hemiplegic limbs, but more rarely, and only when the loss of voluntary power is incomplete. They vary greatly in character in different cases. Sometimes a more or less violent tremor is observed, which may either continue even while the part is at rest, or occur only when it is made to execute some voluntary effort. Charcot, for example, mentions an instance in which the act of carrying a glass to the mouth was attended with rhythmical movements, so that the liquid in it would be thrown in all directions. In other cases spasms are seen, which are more or less choreiform in character, so that Dr Weir Mitchell (1874) and Charcot have spoken of a "post-hemiplegic chorea."

Lastly, some cases of chronic hemiplegia are attended with remarkably slow movements, principally affecting the thumb and fingers, and exactly like those described by Dr Hammond (1871) under the term "athetosis." The name (ἄθετος, without a fixed position) is intended to signify that the parts concerned in the spasm cannot be kept still. They are constantly moving, without any voluntary effort on the part of the patient. The fingers are alternately flexed and extended, with varying degrees of adduction or abduction, so as to give the hand a very grotesque appearance; and the toes may present similar changes of position. Cases of this kind have been described in Great Britain by Dr Allbutt and Dr Gairdner; and Dr Gowers has discussed "post-hemiplegic disorders of movement" very fully in the 'Med.-Chir. Transactions' for 1876.

Aphasia.—Another symptom which is frequently present in the diseases under consideration is loss of speech,—the defect being of a special kind and presenting characters which merit careful study. It has received several different names,—*alalia*, *aphemia*, *aphasia*; but of these the last is now universally adopted. It is altogether distinct from a mere impairment of articulation, such as I have described as occurring in bulbar paralysis, although this impairment may form part of common cerebral hemiplegia when it is due to imperfection in the movements of the tongue and palate. In either of these conditions the speech is more or less thick and difficult to be understood. But in aphasia, if the patient can utter a word at all, he commonly pronounces it quite clearly and distinctly. He may be altogether mute. Or he may occasionally, when excited, ejaculate some oath, which Dr Jackson will hardly admit to be truly a part of language—particularly as it is often impossible to get him to repeat it deliberately. Or he may possess the power of saying two or three words or short

sentences, these he uses sometimes correctly, but often in contradiction to his real meaning, and in answer to every question that is put to him. Thus Trousseau relates the case of a man named Paquet, who for four months said nothing but *cou-si-si*, *cousisi*, and who kept uttering these three syllables on all occasions, whether he was in a passion, or wished to express gratitude, or wanted to ask for or refuse something. Only, if he became very excited, he would sometimes say *sacon*, *sacon*,—probably an abbreviation of the oath *sacré nom de Dieu*. Dr Broadbent had a patient who could scarcely say anything but *Oh, shameful! shameful!—Oh pity! pity!* Of two celebrated cases that occurred to M. Broca, one was that of a man who said *tan*, *tan*, to every question for twenty-one years; another possessed four words, *oui*, *non*, *trois*, and *toujours*. If there is a more extensive vocabulary, the patient is very apt to use one word for another. There may then be a similarity in sound between the two, as when *purging* takes the place of *perjury*, *pamphlet* of *camphor*, *dispersion* of *dispensary*. Or the resemblance may be in meaning, as where a patient of Dr William Ogle's said *boat* instead of *tub*, or (by an odd confusion of ideas) substituted the word *two-shilling-piece* for *spectacles*. The writer to whom I have just referred draws special attention ('St George's Hosp. Rep.,' vol. ii) to the fact that grammatical form is always observed; substantives are used in the place of substantives, verbs for verbs, numerals for numerals. Thus M. Broca's patient employed *trois* to express any number, but corrected what he said by holding out the proper number of fingers at the same time. Dr Broadbent's patient possessed only one name for a locality,—namely, Burlington, where she had lived as a child; and she used this when she wished to name any place whatever. One curious circumstance is that a person who had known two or more languages may entirely lose the power of speaking one of them, while he retains that of conversing in the others. Trousseau gives an instance of this in a Russian who before his illness spoke French like a Parisian; and Bastian says that he has seen two similar instances, one being in a German who had long been resident in England. Sometimes, a patient who can utter only one or two words by himself is able to repeat a good many other words, if he is prompted. Thus Trousseau relates the case of a man named Marcon who could only say *Ma foi* and *'Cré nom d'un cœur*, but who, when asked, "Are you from the Haute Loire?" replied *Haute Loire*, and then to the query, "What's your name?" echoed again *Haute Loire*,—"Your profession?" *Haute Loire*—"But your name is Marcon?" *Yes, sir*,—"What department do you come from?" *Marcon*, and so forth. We shall hereafter see, however, that such a case as this is not to be considered as one of *mere* aphasia, although that affection was of course present. Indeed, we should probably refuse the name of aphasia altogether to such a case as that recorded by Dr Bateman as having occurred at the Salpêtrière, of a woman who, although she said nothing of her own accord, repeated everything that was said to her, and mimicked all the gestures of the students and of the other patients near her. We shall presently see that the view taken of such cases involves important questions as to the pathology and seat of the disease by which they are caused.

In some, but not in all, cases of aphasia the patient loses the power of expressing himself in other ways, as well as in speech. There is, for example, incapacity to *write*, which has been called "Agraphia." A person who is aphasic is sometimes able to make all his thoughts known with his pen. Trousseau relates the case of a carrier belonging

to the Paris Halles, who came into the consulting room making signs that he could not speak, and handed in a note in which the history of his illness was detailed. He had written it himself, and it was perfectly well worded. A few days previously he had suddenly lost his senses, and remained unconscious for nearly an hour. When he came to, he had no paralysis, but he could not articulate a single word. In the course of five or six weeks he completely recovered; but what was remarkable was that during the whole course of the disease he could manage all his affairs by substituting writing for speech. Such cases are very exceptional. As we shall presently see, paralysis of the right hand is very often present in those who have aphasia; and of course it is then difficult to test the writing. Trousseau, however, mentions one patient, who said *oui* to everything, and who was unable to write even with the left hand. One way of ascertaining whether the power of combining letters to form words is preserved, is to give such a person the loose wooden letters that children play with, asking him to spell his name or to put together a sentence. He may be quite incapable of doing so; indeed he may not even be able to pick out the letters that make up the one or two words that he is perhaps able to articulate. Trousseau's patient, for instance, whose one word was *oui*, could not point to the letters o, u, i, in the title-page of a large quarto volume, containing those letters. Indeed, the impairment of the power to write is sometimes very marked, when the speech is comparatively little affected. Dr Huglings Jackson records the case of a woman who could talk pretty well, although she frequently made mistakes in speaking, and called her children by many names; but when told to sign her name she wrote *Sunnil Siclaa Satreni*, in which there was not the slightest resemblance to the real name either in sound or spelling; and when told to write her address she put down *Sunesr met ts mer tina—lain.* We shall presently see that if there is much impairment of the intelligence the value of agraphia as a symptom is much diminished; but both Trousseau's patient and Jackson's appeared to have full possession of their mental faculties.

Again, it has been shown that in some cases of aphasia the memory of *gestures* is lost. Trousseau says that such a patient may be unable to *put on* the face of a person who is crying, although when he feels grief the expression of his countenance shows it clearly enough. The patient, Paquet, who said nothing but *Cousisi*, was the subject of the following experiment: Trousseau first held out his two arms and hands, and moved his fingers, as though he were playing the clarionet, and asked this man to imitate him. He immediately executed the same movements with perfect precision. When asked whether he knew that the attitude was that of a man performing on the instrument in question he would assent by nodding his head. Yet, when told, a few minutes afterwards, to place himself in the very same attitude he seemed to think, and was most times unable to do it. So, also, a patient whose case was carefully studied by Dr Scoresby Jackson ('Edin. Med. Jour.,' 1867) was found to be totally unable to play the piano. He put himself in the proper position, and placed his fingers on the keys, but he could not play a single bar, not even of a piece of music with which he had been familiar before his illness. With the left hand alone he managed a bar or two very slowly, but transposed several notes. Yet he could hum the same tune pretty well. On the other hand, games of skill are often practised by aphasic persons with unimpaired accuracy. Dr Scoresby Jackson draws special attention to the skill with which his patient

played draughts ; and Trousseau's man Paquet could play backgammon and dominoes perfectly, knowing all the tricks of those games, and cheating when he found himself losing.

It is important to notice that the very words which an aphasic patient is unable to utter are perfectly understood by him when spoken by another person. This, indeed, is but an illustration of a broad distinction which is traceable throughout the faculty of speech. A child learns the meaning of words addressed to it by others long before it can itself speak. A person partially acquainted with a foreign tongue recognises many words, if spoken distinctly by anyone else, which he would have been unable to call from his memory if he wanted them in conversation. Nay, we all of us are accustomed to a far wider English vocabulary in the daily speech of others than we ourselves make use of when we ourselves talk. Adopting terms used by Dr Moxon, we may say that there is a great difference between "incoming" and "outgoing" language ; or, following Dr Broadbent, that words are to be considered in two distinct aspects, first as "intellectua symbols," and then as "motor processes." In aphasia it is the *outgoing language* and the *motor processes* that are interfered with.

Now, there are evidently two ways in which such a limitation of the aphasic condition can be accounted for. It may be that the perception of the meaning of the words spoken by others, and the uttering of words for one's self, are respectively functions of different parts of the brain. Or, again, it may be that both "incoming" and "outgoing" language have their seat in the same locality, or are both of them functions of the cerebral hemispheres as a whole, but that the latter requires for its execution the intervention of some subordinate nervous centres which have nothing to do with the former. The second of these hypotheses seems to me to be the correct one ; it has been defended with great ability by Dr Broadbent in the 'Med.-Chir. Transactions' for 1872.

The local lesion.—It has now been established that aphasia—including agraphia when that is also present—is almost invariably dependent upon a lesion of the *left side* of the brain. This is not perhaps, a new discovery, for Dr Marc Dax, of Sommières, read a paper on the subject before a Medical Congress at Montpellier in 1836, but the paper is now lost. However, I do not suppose that any physician, either in Paris or in London, was acquainted with the views of M. Dax when in 1861 M. Broca published in the 'Bulletin de la Société Anatomique' a case which has formed the starting-point of all our modern knowledge. It may seem strange that the association of right hemiplegia with loss of speech escaped the notice of any one of the acute clinical observers of the first half of the present century ; but it was doubtless incongruous with the views which then prevailed as to the cerebral functions, and, indeed, it still remains an isolated fact, and needs to be specially investigated and explained. The existence of such an association is beyond question, and can at any time be determined by clinical observation. If a number of hemiplegic patients be taken, it will always be found that many of those in whom the paralysis is on the right side are speechless, whereas probably every one among them whose left arm and leg are paralysed will be able to articulate as distinctly as is compatible with the condition of the tongue and lips. Moreover, in a certain proportion of cases, aphasia will be found to exist without any paralysis, the lesion being then almost invariably in the left side of the brain. The exceptions are themselves based upon a rule, namely this,

that in left-handed persons loss of speech goes with paralysis of the left limbs, and not with that of the right limbs; or, in other words, that it depends upon disease of the right hemisphere instead of the left. Examples of such an association have been recorded by Dr Jackson ('Med. Times and Gaz.,' 1866), Dr John Ogle ('Lancet,' 1868), and Dr Wadham ('St George's Hospital Reports,' 1869), and two well-marked cases have occurred in Guy's Hospital.

The next fact in the pathology of aphasia is that it is caused by lesions of a particular part of the surface of the brain on the left side, and is never the result of disease limited to the basal ganglia. Now, Bouillaud was long ago led by clinical observation to connect the faculty of language with the anterior lobes of the brain, and, indeed, Gall had previously suggested the same localisation. But it was not until a second case of M. Broca's was published, in November, 1861, that anything like precision of localisation was arrived at, in regard to loss of speech. The case in question is that of a man named Lelong, aged 84, who after an attack of apoplexy lost the power of uttering words, with the exception of four, although he knew all that was said to him, and could make himself understood by gestures. He died at the end of a year of a fracture of the femur. At the *post-mortem* examination the posterior third of the second and of the third left frontal convolutions were found to have been destroyed by softening, their place being occupied by a collection of fluid in a cavity the walls of which were of an orange-yellow colour from the presence of altered blood.

Ever since the third left frontal has been known as Broca's convolution, and the majority of pathologists have maintained that aphasia is always dependent upon a lesion involving it, or at least affecting some closely adjacent part of the surface of the same hemisphere.

Now, this doctrine involves two distinct statements. In the first place, it asserts that no disease of the corpus striatum, or even of the white substance outside it, is capable of causing loss of speech. The general accuracy of this assertion is proved by the fact that cerebral hæmorrhage, in the position in which it usually occurs, leaves the speech unimpaired. In 1876 Dr Broadbent was able to say ('Brit. Med. Journ.,' i, p. 436) that he knew of no case on record in which lesion of the corpus striatum alone had given rise to this symptom. At first sight such a fact might seem inexplicable, since it is difficult to see how any effect can be produced by destruction of a convolution which may not equally follow the division of those conducting fibres which connect it with the basal ganglia, or even with the spinal centres. No writer appears to have faced this difficulty, except Dr Broadbent himself. He believes that there are two distinct routes by which impulses pass from Broca's convolution to the nerve-nuclei in the pons, medulla oblongata, and medulla spinalis. One is the straight path through the left corpus striatum, the other is by commissural fibres which go to the third frontal convolution on the opposite side, and thence down through the right corpus striatum. So long as either of these routes remains open, speech is possible; it being of course assumed that the nuclei on opposite sides are so closely associated together by cross connections as to be set in action simultaneously. It may be noted, as one inference from Dr Broadbent's hypothesis, that if hemiplegia and aphasia occur together, the lesion must be one which either involves simultaneously Broca's convolution and the corpus striatum, or else affects an extensive area of the hemisphere in the neighbourhood of the fissure of Rolando. Whichever is the case, the cause of the disease must almost

always be obstruction of the Sylvian artery whether by embolism, thrombosis, or syphilis. Now we have already seen, that whereas the central ganglia inevitably undergo softening when their blood supply from that artery is cut off, the convolutions may fail to present any obvious morbid change, although the interference with their circulation may nevertheless have been quite sufficient to impair their functions. In other words, if in making an autopsy one should overlook plugging of the Sylvian artery—and until lately it was not usual for pathologists to devote special attention to the condition of the cerebral vessels—one might easily suppose that the only disease was situated in the corpus striatum, and that this had caused the aphasia. In July, 1877, I made an autopsy in which it would have been quite possible to commit this mistake; the patient was aphasic, and the only obvious lesion of the brain was in the corpus striatum, but the Sylvian artery contained an embolism at its very commencement.

In the second place, the doctrine referred to in the commencement of the last paragraph involves the assertion that aphasia cannot be caused by a lesion of any part of the surface, even of the left hemisphere, with the exception of the third frontal, or at least of some immediately adjacent convolution.* Now, it may be that this position is not as yet completely established. Cases are very rare in which a morbid change is limited to a single spot on the convexity of the brain with sufficient accuracy to throw any light upon the question. If it be softening, it must be due to obstruction of a single branch of the Sylvian artery. We shall hereafter see that the effects produced by tumours are in some respects fallacious. Nevertheless, a case occurred at Guy's Hospital in which transitory attacks of aphasia, in a woman who had cancer of the breast, led to the suggestion that she had a secondary nodule in the third frontal convolution of the left hemisphere of her brain, and this diagnosis was afterwards verified by an autopsy. The facts cited by Trousseau, in opposition to the localisation of aphasia in Broca's convolution, or in its close neighbourhood, seem to me of very little value; and this is the opinion of Dr Broadbent likewise, who said in 1872 that he examined into all the apparently exceptional cases of which he had been able to find the records; and that it was remarkable how large a proportion of them broke down under careful scrutiny.

Theory of aphasia.—Reverting now to the physiological causation of aphasia, we have to ask how it can be that loss of speech is always dependent on a lesion of the surface of the left hemisphere, except in left-handed persons, in whom the association is with disease of the corresponding part of the right hemisphere. By Trousseau this supposition was treated as an absurdity, opposed alike to analogy and to common sense. And I believe Dr Moxon was the first to throw any light upon it, and to suggest an explanation which has virtually been accepted by almost every subsequent writer. His view ('Med.-Chir. Rev.,' 1866) is that the two halves of the brain are originally symmetrical, and resemble one another in their functional capacities, but that in the course of education one side only becomes stored with those ideas of associated movements which are required for the performance of bilateral actions, such as are concerned in speech. Or, as he elsewhere puts it, one side of the tongue *guides* the other; just as the right

* It is the posterior part of the third frontal which is the region in question, and with it are found affected the back part of the second frontal, and the adjacent part of the insula and gyri operti. The front and upper part of the frontal lobe, the corpus striatum, claustrum, and external capsule, form no part of the region.

hand guides the left one when they are made to execute similar motions, it being notorious that this requires infinitely less attention than the execution of opposed motions by the two hands respectively. Dr Moxon does not more fully explain why the process of education should take place on one side of the brain rather than the other; and this has been regarded as a difficulty by some later writers. But, as we have seen, the associated movements which make up the act of writing, and in which only one hand is concerned, are so closely connected with those that constitute speech, that agraphia very commonly accompanies aphasia. Now, it is true that all men do not learn to write; but there is perhaps no savage who does not, from his earliest infancy, become accustomed to employ one hand rather than the other in gestures by which he supplements speech in communicating with others. And it was a suggestion made by Broca himself that this preference for the use of the left hemisphere for so many other purposes led to the education of convolutions of that hemisphere for the "motor processes" concerned in speech.

I have hitherto confined the subject of aphasia entirely to the loss of "outgoing" language, as Dr Moxon calls it; we have still to ask what are the relations of this affection to that other part of the faculty of speech—"incoming" language—which consists in the recognition of words spoken by others. Now, many modern writers, following Dr Sanders and Dr William Ogle, admit a separate variety of aphasia, which they term *amnesic* or *amnemonic*. The characteristic symptom of it is, they say, that the patient is able to utter words, provided that he has first heard them spoken by another person. In fact, one may be able, by prompting, to make him speak pretty freely, although he may be incapable of saying anything in reply to a question which does not suggest its own answer. Now, it is, in the first place, to be observed that the value of this test is limited to those cases in which it yields a positive result. If the man continues altogether speechless, after one has repeated again and again the word or the sentence which he is asked to utter, one gains nothing by the experiment. That the memory for "incoming" language is not altogether lost may, indeed, sometimes be obvious from the gestures of intelligence which such a person makes if the right word is suggested to him, and from his strongly-marked dissent when other words are substituted for it. Moreover, in those cases in which an attack of aphasia rapidly passes off, the patient may be able to give a complete account of all that occurred during his illness, and to state that his power of thinking was altogether unaffected. Prof. Lordat, for example, who once suffered in this way, said that he was able to combine abstract ideas, and to distinguish them accurately. Being accustomed to teach, he thought over the subject matter of a lecture, and found that he could dispose in his mind the chief points without difficulty, and introduce any changes that he pleased in their order. He thought of the Doxology, but he was not able to recollect a single word of it. I must leave it to the metaphysicians to discuss how far it is possible for the mental processes to be carried on without the revival of words, as symbols, in the consciousness. But I think it is clear that in M. Lordat's case the fault lay in the machinery of expression alone.

Again, when aphasia is incomplete, so that the patient can utter one or two words, and uses them in answer to every question that may be put to him, he is often perfectly conscious of the mistakes that he commits. Every physician has seen such cases, and will remember the shake of the head, the puzzled look, the smile—half amusement, half vexation—the

repeated attempts to find the right word, the beam of satisfaction if by chance it at last comes off the tongue.

These facts seem to me to prove beyond dispute that the memory of language may be unimpaired, even when there is complete aphasia. Now let us look at the other side of the question. Many patients affected with partial aphasia go on uttering the same word for months or years, without seeming to know that anything is amiss with them. Trousseau studied very carefully the case of an artist who, according to his own account, suffered from nothing but failure of speech, being able to understand perfectly all that he read, and being in full possession of his intellect. It turned out that he made the grossest mistakes in reading aloud, that he wrote one word for another without being aware of it, and that when asked to sketch a human figure he drew like a child who had never been taught. The same writer, referring to another case, lays stress on the fact that the patient, who read the newspaper, and expressed by signs that he understood it perfectly, was nevertheless in the habit of reading the sheet over and over again in the same day. And a girl who was under his care had for a year one book in her hands—a religious work, the 'Month of Mary'—and almost always read the same page. He suggests that a very good test of the understanding of an aphasic patient is to take up a book, and to read a few lines, telling the patient to follow with his eyes, and to turn over at the proper moment. The artist, whose case has already been referred to, could never do this correctly. From these observations Trousseau concludes, in spite of the facts mentioned in the previous paragraph, that in aphasia there is not merely loss of speech, but also impairment of the intelligence. But it seems to me that this writer did not sufficiently bear in mind the fact that the lesions which cause aphasia are in most cases such as interfere with the supply of blood to a large part of one hemisphere of the brain. We have seen that when this symptom is associated with hemiplegia it is almost invariably dependent upon obstruction of the trunk of the Sylvian artery. A good illustration of the principle that in such cases other symptoms may be present, which have no necessary connection with the aphasia, is afforded by the fact that there is often anosmia—loss or impairment of smell—in the corresponding nostril. In the 'Med.-Chir. Transactions' for 1870, Dr William Ogle refers to seven instances of this: he supposes that the so-called external root of the olfactory bulb becomes "implicated when it is passing to the floor of the fissure of Sylvius." Dr Ferrier's suggestion that softening extends to the *subiculum cornu Ammonis* (which he supposes to be the centre of smell) appears to be inadmissible, since that structure lies beyond the area of distribution of the Sylvian artery.

The question, therefore, is—not what degree of defect of understanding *may* be found in aphasic patients, but what amount of intelligence they are capable of retaining. Instances of pure aphasia, unattended with paralysis of the limbs, are especially worthy of study in reference to this question. I think it will be found that while the loss of speech is often absolute, the memory may in such cases be unimpaired.

On the other hand, one is constantly seeing patients with right hemiplegia who go on for years, unable to utter a single word, or to communicate in any way with their dearest relations. It would be a sad thing to suppose that such persons are really in possession of all their mental faculties, and that they are living, as it were, imprisoned within an iron mask. We may, I think, dismiss such a notion. Indeed, it is certain that those who suffer

from left hemiplegia, dependent upon any extensive lesion of the right hemisphere, manifest an equally marked impairment of intelligence, although they are not deprived of speech.

These considerations have led me to the conclusion that, although loss of speech is often associated with the inability to understand "incoming' language or to recognise mistakes made in "outgoing" language—and I think it would be convenient to transfer the use of the term *amnesia*, so as to make it correspond with this condition—yet the two things are essentially independent of one another, and the latter indicates the existence of a lesion extending far beyond the limits of Broca's convolution.

I have recently found that Dr Broadbent has arrived at the same conclusion ('Med.-Chir. Trans.,' 1872, p. 174). He, like myself, is firmly convinced of the truth of Broca's theory, so far as concerns the seat of the lesions which are attended with aphasia, when it occurs independently of any failure of intelligence. This theory, however, is very far from implying that the whole of the faculty of language is localised in any one part of the hemispheres. It only asserts that a certain spot in the left hemisphere contains machinery, without the use of which a person cannot utter words, nor indeed convey his thoughts to the pen in writing.

Aphasia in lunatics.—There is yet another point of view from which it is necessary to consider the pathology of aphasia; namely, as to whether this symptom, when occurring in persons of unsound mind, is necessarily dependent upon a lesion in, or even upon functional disturbance of, Broca's convolution. It seems to me that such a conclusion is very doubtful. Dr Bastian mentions the case of a lunatic who did not utter a single word for several years, except during two brief intervals, once when he had an attack of pleurisy that lasted some days, and once when he was suffering from toothache. And Dr Forbes Winslow relates a similar instance; a person who had been insane for fifty-two years did not speak during thirty years, but recovered speech and answered questions perfectly well during the last fifteen years of his life. In such cases there is surely no reason to suppose that any local lesion was present.

The same observation may be made in reference to some of the cases which Trousseau gives as examples of aphasia; for instance, that of the lady who was wont to welcome her visitors by exclaiming *pig*, *animal*, *stupid fool*, while she nevertheless seemed not to understand the meaning of the insulting expressions which she used. And, again, it is applicable to an example of supposed agraphia, occurring in a lunatic at Broadmoor, which has been recorded by Dr Bastian. The inability to write was of an "amnesic" type, and seems to me to have been merely one of the manifestations of his insanity. ('The Brain as an Organ of Mind,' 3rd ed., p. 660.)

Apoplexy.—Remarkable changes have occurred in the meaning of this term. From having originally signified a "stroke," in which the patient falls like an ox struck down by the butcher, apoplexy became applied to the effusion of blood upon the brain which was found to be the most common cause of such attacks, and this use of the word was afterwards extended to hæmorrhages in other parts, so that pulmonary apoplexy, retinal apoplexy, and apoplexy of the suprarenal capsules were spoken of. There is, however, no advantage in employing apoplexy as a mere pathological synonym for "cerebral hæmorrhage," a term which is itself both convenient and devoid of ambiguity. Moreover, in clinical medicine it is extremely desirable to

have a name for that form of coma which is due to disturbance of the cerebral circulation from some local cause acting within the cranial cavity—as distinguished, I mean, from mere failure of the heart's action on the one hand, and, on the other hand, from narcotic or alcoholic poisoning, uræmia, epilepsy, or external injury; and for this purpose the word apoplexy appears to be altogether suitable and convenient. A long chain of authorities, including some of the most distinguished names in medical literature, might be quoted in favour of this use of the term.

Symptoms.—A patient in an apoplectic fit lies "deprived of sense and motion." He cannot be roused, but there may be varying degrees of insensibility. Sometimes the well-known voice of a wife or son may elicit an unintelligible muttering or growling sound in reply, or the application of a spoon to the lips may cause them to be closed and the teeth to be clenched in automatic refusal of food; or one hand may be used to rub or scratch the side of the face or body. More often no such indications of cerebral activity present themselves; the limbs remain in whatever position they may happen to fall into: the respiratory movements and the beatings of the heart alone show that life remains. The pupils are generally torpid, and sometimes altogether insensitive to light; they are sometimes equal and of normal size, sometimes both dilated or both minutely contracted, sometimes unequal, one being dilated and the other contracted. The conjunctivæ can often be touched without any reflex movements being exerted in the eyelids. The cutaneous reflexes are diminished or abolished, rarely increased. Pinching or pricking the skin seldom leads to any manifestation of consciousness, but sometimes the hand or the foot is drawn away, and so one may be able to make out that one side of the body is withdrawn from nervous influence more completely than the other side. Or it may be found that when the upper limbs are lifted and allowed to drop upon the bed, one is a dead weight; or, on the other hand, the arm and the leg on one side may be rigid, those on the opposite side may be relaxed. We shall presently see that such indications of unilateral mischief are of considerable diagnostic importance, and another similar sign, which may sometimes be noticed when it is impossible to make out the presence of hemiplegia, is the conjugate deviation of the eyes described at p. 570.

In the foregoing description of an apoplectic seizure I have not attempted to arrange the various symptoms in a systematic way, but have rather endeavoured to follow such an order as would be likely to be adopted by a medical man when called to examine an actual patient. The appearance of the countenance varies greatly in different cases; sometimes it is pale, sometimes congested and of a purple colour, with lividity of the lips and tongue; the features are often turgid and swollen; the forehead and cheeks, and, indeed, the whole surface of the body, may be bathed in perspiration, which saturates the linen and stands in large drops upon exposed parts of the skin.

The *temperature*, as measured by the thermometer, has been carefully investigated by Bourneville. He finds that there is at first a slight fall, amounting to about 2° Fahrenheit. In rapidly fatal cases this continues until death, but if life is prolonged the temperature rises, and for several days it may remain at about 100°. When death occurs at an interval of more than ten hours from the commencement of the attack it may be preceded by a rapid elevation of temperature. I believe that this was first noticed by Dr Hughlings Jackson; it is always to be regarded as a very unfavourable sign. I have notes of one case in which the thermo-

meter registered 107°. When recovery is to take place the temperature commonly returns to the normal point two or three days after the commencement of the attack.

The *pulse* may be either increased or diminished in frequency; the prognosis is bad if it is greatly above or greatly below the average, say below 60 or above 120, but the converse is not necessarily true. Dr Jackson mentions an instance in which the pulse was 72 within five hours of the death of the patient, the rate of the breathing being 14 in the minute. As a rule the pulse becomes more rapid as the case goes on towards a fatal termination, but the worst sign of all is irregularity of the pulsations of the heart, a succession of rapid beats being followed by a series of beats at long intervals, or the heart perhaps stopping altogether for a brief space. Formerly great stress was laid upon the "full" and "labouring" character of the radial pulse in apoplexy, and it was supposed to be a proof of the necessity for venæsection. We now refer this character either to want of elasticity from atheroma of the aorta, or to the high arterial tension of chronic Bright's disease.

The *breathing* is sometimes infrequent—there may be as few as three or four respirations in the minute. Towards the last it often happens that the patient ceases to breathe for perhaps a minute, and a purple flush diffuses itself over the countenance, but afterwards a deep breath is again drawn, the face resumes its natural colour, and the respiration goes on as before. This may be repeated several times, until at length a final pause occurs; the heart may then go on beating for a considerable length of time, but at length its pulsations cease and the patient is dead. In some cases the respiration assumes the characters observed by Drs Cheyne and Stokes, and known by their names.

But in many cases the way in which apoplexy destroys life is by a gradual increase of obstruction to the breathing, which seems generally to depend upon a concurrence of several distinct causes. One of these is very often the supervention of an œdematous pneumonia, beginning in the bases of the lungs, and spreading upwards through the back parts of those organs. Another is the acccumulation of a thin secretion in the air-passages, which becomes beaten up into a froth by the inhaled air, and may completely fill the main bronchi and even the trachea. It is probable that the pouring out of this fluid is often the result, in part, of irritation set up by the entrance of fluid nourishment; for when a patient is comatose, milk and beef-tea and brandy are very apt to run down into the larynx without giving rise to any warning cough or sensation of choking. But I think that there can be no doubt that another important factor in its production is the impediment to the act of respiration which is due to paralysis of the tongue and fauces. Everyone who has made *post-mortem* examinations on the bodies of those who have been suffocated knows how large a quantity of frothy fluid is found in the air-passages, even when death has been rapid; but an apoplectic patient may lie for days in a condition in which there is considerable interference with the breathing, as is shown not only by the lividity of his countenance, but also by the fact that the entrance and exit of air are each accompanied by a noise which is commonly called *stertor*. The causes that lead to the occurrence of this sound were, I think, first clearly pointed out by Dr Bowles, of Folkestone, in the 'Med.-Chir. Trans.' for 1860. He admits that when the mouth is partially open the soft palate sometimes drops upon the tongue, and vibrates as the air rushes in beneath it. But he ascribes far more importance to a change in the position of the tongue

itself; he shows that when the mouth falls open, the point of attachment of the lingual muscles to the symphysis is carried backwards, and he thinks that the tongue then comes into contact with the posterior wall of the pharynx. As might be expected, this is especially apt to occur while the patient lies supine. Dr Bowles finds that turning him over upon his side, with the mouth inclined so that the saliva and other secretions can drain away from it, often causes the entire disappearance of stertor, and may be followed by a decided improvement in some of the other symptoms. When the breathing is noisy the cheeks are often puffed out at each expiration in consequence of paralysis of the buccinator muscle; this is of no significance, however, unless it be as suggesting an unfavourable prognosis.

Duration.—The period that an apoplectic seizure lasts is very variable. In some exceedingly rare instances death takes place a few minutes after the commencement of the cerebral symptoms. Thus Dr Jackson mentions the case of a woman who was sitting at the tea-table, when she stopped in the middle of a laugh, cried out "Oh, my head!" fell back in her chair, and died within not more than five minutes. Abercrombie relates a similar occurrence in a woman who was one evening attending a crowded meeting, and who seemed to be in perfect health. Towards the conclusion she uttered a loud and convulsive scream, and fell down insensible. She was immediately carried out, and was seen by Dr Macaulay, who happened to be present. He found her pale and altogether unconscious, and within five minutes she was dead. In each of these cases blood was found effused under the arachnoid. In 1864 there was brought to Guy's Hospital the body of a woman who had died almost instantaneously as she was returning home from the theatre with her children; in that instance also a large quantity of blood had been poured out over the sides and base of the brain. But some years ago I made an autopsy in a case in which death had taken place nearly as quickly, and in which there was a large clot within the left hemisphere, bursting into the lateral ventricle. The patient, a man, aged forty-one, was in a surgical ward for some laryngeal affection, and had been sent down to the dispensary to fetch the medicines; on his way he had a fit, became comatose, and died within ten minutes. One would have expected that hæmorrhage into the pons would often cause instantaneous death. But Dr Jackson remarks that he has never seen such a case, although he has known a woman lie deeply comatose for some hours, in whom this part of the brain was hollowed out into a mere shell. In the most rapidly fatal case of a clot in the pons that I find recorded at Guy's Hospital death occurred in forty minutes; two patients lived each for two hours, one nine hours and a half, one thirteen hours, one sixteen hours, and one two days.* Meningeal hæmorrhage also seems constantly to prove fatal within forty-eight hours. But in the ordinary form of apoplexy, in which blood is effused into the neighbourhood of the corpus striatum, life is often maintained for a much longer period. I have notes of twelve cases in which death did not occur until between the second and the seventh days; and of six cases which terminated at the end of ten, twelve, thirteen, sixteen, nineteen, and again nineteen days respectively. It is worthy of remark that two among these six cases were examples of what I believe to be a very rare occurrence, namely, the formation of a clot within the substance of the brain, which was large enough to destroy life, but which yet failed to reach either

* Of seventy-eight cases of hæmorrhage into the pons collected by Bode, forty-six proved fatal within twenty-four hours; quoted by Ross.

the lateral ventricle or the surface; and in a third case the seat of the hæmorrhage was altogether exceptional, being the interior of one posterior lobe.*

But, as I have already remarked, an apoplectic attack does not necessarily prove fatal. In many cases consciousness is regained. This always takes place more or less gradually. After a few hours, one is perhaps able to rouse the patient so that he will give his name, although when left to himself he still takes not the slightest notice of anything that goes on in the room. For several days he generally remains drowsy and apathetic, or his ideas may be confused and perplexed; or he may even be delirious, talking incoherently, and throwing himself out of bed. In such cases, however, the prognosis is not good, in spite of the fact that the coma has passed off. Nothnagel says it is exceptional for recovery to occur if insensibility persists for as long as forty-eight hours. In cases which terminate favourably, as the patient regains consciousness, the symptoms of local damage to the brain become manifest, of which the most important is hemiplegia, with or without aphasia.

Onset.—Hitherto, I have, as far as possible, avoided saying anything about the way in which an apoplectic attack begins. Etymologically, its commencement ought to be sudden; the patient falling down in an instant, as if struck upon the head. But in the cases now under consideration such a seizure comparatively seldom occurs.

The older writers laid great stress upon certain symptoms which they believed to be frequent precursors of apoplexy, and which they therefore designated "warnings" or "molimina." But under these names they included a great variety of complaints, without any definite limits as to time: —an epistaxis, for instance, happening several months before the cerebral attack; or an ecchymosis of the conjunctivæ, perhaps the result of a violent effort in coughing or sneezing; or even the frequent recurrence of giddiness or headache in a person advanced in years. These last symptoms, indeed, are not unlikely to be due to a morbid condition of the blood-vessels of the brain, which may presently lead to their rupture; and the local patches of softening, which we have seen to be more direct effects of such vascular changes, afford a ready explanation of other nervous disorders,—such as thickness of speech, diplopia, partial ptosis, sensations of numbness and formication in the hand or in the fingers, partial loss of power in the arm, or dragging of the foot,—any one of which, if occurring in an old man or woman, is pretty sure to be taken as threatening the supervention of an apoplectic seizure, even though it may after a few weeks disappear, leaving the patient apparently as well as ever. One cannot altogether reject this view of the matter; but it is to be remembered that each of the symptoms in question may arise from other causes, and that even if one could be sure that they were due to a diseased state of the cerebral arteries, it would not necessarily follow that apoplexy was about to occur.

On the other hand, the very same symptoms may be actually results of cerebral hæmorrhage, a vessel in the brain having already given way; and, unless the extravasation remains small in amount, the patient is very likely to become comatose a few hours later. In such cases it is obviously incorrect to speak of "warnings."

Abercrombie, many years ago, pointed out that cerebral hæmorrhage

* In twenty-four fatal cases of cerebral hæmorrhage under the editor's care, death took place under twelve hours in seven, under twenty-four in eleven; on the second or third day in three; and on the fourth, fifth, or sixth in eight; and on the ninth in two.

comparatively seldom leads to sudden loss of sense and motion, or (in other words) to the classical form of apoplectic seizure. A very precise clinical history of the cases in which extravasation of blood is most apt to be found is given in his celebrated work. The first symptom, he says, is a pain in the head, which may be so violent as to make the patient scream. The face at the same time becomes pale, the body cold, and the pulse very weak; there is sickness, or even vomiting; and he may fall to the ground faint and exhausted. Often a slight convulsion occurs. After a little while he may be able to walk home; he is quite sensible, but oppressed; he remains cold and feeble, with cadaverous pallor of the countenance. By degrees he recovers his warmth, his face regains its natural appearance, and his pulse improves in strength. Then he becomes flushed, he answers questions slowly and heavily, and gradually he sinks into coma, from which he very seldom recovers.

All subsequent writers have recognised the truth of this picture. It has been shown that after fracture of the skull, when the middle meningeal artery is torn through, a very similar series of events take place; and the name of "ingravescent apoplexy" has been given to cases which run such a course. But, until quite recently, most observers have regarded this as only one form of the disease, and have supposed that cerebral hæmorrhage very commonly begins with sudden coma. It is, indeed, the fact that insensibility is often present when the patient first comes under observation. Perhaps he is picked up in the street, or found lying on the floor, or in a water-closet; or the seizure may have come on during sleep, so as to give no opportunity of tracing its earlier symptoms. But Trousseau says that although his attention was for fifteen years directed to the question, he did not in that time meet with a single instance in which, when an attack of cerebral hæmorrhage occurred in the presence of witnesses, it did not begin more or less gradually. An exception ought perhaps to be made for those cases which destroy life in a few minutes. Another exception is made by Trousseau himself for certain cases which begin with an epileptiform seizure, and this is endorsed by Dr Hughlings Jackson, who has laid special stress upon the frequency of this mode of commencement. Still the conception of "ingravescent apoplexy" as being a particular modification of the disease has to be given up.

Moreover, it has to be admitted that the symptoms before coma sets in are far more variable than would appear from Abercrombie's description. Pain in the head is often absent. Not unfrequently the only thing which is noticed by the patient is a numb feeling, or a sense of weight in one of the limbs. Thus Trousseau relates the case of a woman who while returning from market noticed that she dragged her right leg, and that her right arm felt heavy, so that she changed into her left hand a folded newspaper which she was carrying, lest it should fall into the mud. She walked upstairs into her room, took off her clothes and got into bed; after which she became hemiplegic and comatose, and remained in a state of stupor for three days. Another patient, who came under the care of the same physician, had noticed while at dinner that one of his hands felt heavy; he was not giddy, but faltered a little in his speech. He tried to rise from his chair, but one of his legs being paralysed he fell down. His children lifted him up, and with their assistance he walked into the next room, and sat down. Trousseau arrived in three quarters of an hour, and found him perfectly conscious; but his left arm and leg were almost powerless. Profound coma set in a few hours afterwards, and he died the following morning.

Another modification of the symptoms produced by cerebral hæmorrhage at its commencement is that in which it is attended with a transitory loss of consciousness, from which the patient quickly recovers, and which is separated by an interval from the final coma. To this Trousseau gives the significant name of "cerebral surprise." A satisfactory explanation of it is not easy. One can, indeed, imagine that the injury to the brain-tissue which occurs at the moment when the vessel gives way causes a kind of shock that is diffused over the nervous centres. Trousseau cites, in illustration, the experiment of trephining the skull of a dog or rabbit and introducing a leaden ball into its interior; symptoms of stupor, he says, are immediately manifested, which quickly pass off, leaving a degree of hemiplegia proportionate to the compression. But Nothnagel says that in hundreds of observations upon animals, in which he injected chromic acid into the brain, or produced artificial extravasation of blood, he never saw a similar effect produced. This writer cites, as an analogous phenomenon, the effect of suddenly cutting through the spinal cord, in instantaneously suspending for a time the reflex excitability of the centres below the line of section.

Locality of the lesion.—Some writers suppose that the symptoms of an apoplectic attack commonly present modifications which enable the exact seat of the lesion to be determined, if it is other than the usual one, close to one of the corpora striata. But I do not think that this can often be done, at least while the coma lasts, nor in cases that prove directly fatal. I have already pointed out that some forms of cerebral hæmorrhage are more likely than others to destroy life rapidly; this, however, goes but a very little way towards a differential diagnosis.

In apoplexy of *the pons* the pupils are often contracted to pins' points, and the respirations are exceedingly infrequent—perhaps not more than six or four in the minute. But I have notes of one case in which dilatation of the pupils was present, and similar instances are mentioned by Nothnagel; the breathing too is sometimes hurried. Nothnagel seems to think that convulsions are more apt to occur than when blood is effused into other parts of the brain, but of this I do not find any evidence. Other symptoms enumerated by him are rigidity of the neck and tonic contractions of the limbs generally. He says that paralysis of the limbs on both sides is observed only in cases which quickly end in death; and I have no doubt that he is right. It can seldom happen that an "alternate hemiplegia" is made out satisfactorily while the patient is insensible. I have already mentioned that this form of apoplexy sometimes leaves behind it a permanent difficulty of articulation.

The fact that lesions affecting the cerebral convolutions so often give rise to convulsive seizures has led some observers to suppose that such symptoms are especially apt to occur in cases of *meningeal hæmorrhage*. But so far as I can judge from the cases which I have collected they are not really more frequent than when blood is effused into the interior of one of the hemispheres. Nor is rigidity of the limbs noted as having been commonly present,—a fact which Dr Goodhart has already pointed out in the 'Guy's Hospital Reports' for 1876. On the other hand, it would seem that a definite hemiplegia is comparatively seldom observed in this form of apoplexy; the blood makes its way too easily along the subarachnoid space at the base of the brain to compress one hemisphere more than the other. In some cases the coma is preceded by delirium.

Another question is whether it is possible to make out at the bedside the existence of extravasation into the *lateral ventricle*, when the original lesion

is in one of the basal ganglia. The recurrence of coma, after recovery from a first seizure, has been attributed to this cause by Nothnagel and some other writers; but we have seen that it is a common feature in all cases of apoplexy. Others have supposed that the presence of ventricular effusion is indicated by paralysis of all four limbs succeeding to hemiplegia, or by early rigidity of the arm and leg on the side opposite to the lesion. The latter opinion was maintained by Durand-Fardel. But I am not aware that the exceptional cases in which profound coma and death are caused by a clot limited to the substance of one hemisphere have as yet been made the subject of a critical comparison with those in which there is extravasation into the ventricle. In 1874 I made an autopsy in the case of a lad, aged seventeen, who had died fifteen hours after having been found lying on the ground in a state of insensibility. All the cavities of the brain, including the third and fourth ventricles, were found full of clot, which was present in equal quantities on the two sides. I could discover no cause for the hæmorrhage, all the central ganglia being quite healthy. There had been repeated epileptiform fits, but I could not learn that the symptoms had differed in any other respect from those of an ordinary apoplectic attack. In 1876 an old woman died in the Clinical Ward of Guy's Hospital under my care, of a seizure which was particularly noticed to be unattended with definite paralysis of any of the limbs The right lateral ventricle was full of clot, which had come from the superficial part of the caudate nucleus, the internal capsule being quite uninjured. Thus the absence of hemiplegia is not peculiar to cases of meningeal hæmorrhage.

Diagnosis.—In the diagnosis of apoplexy one has to bear in mind many varied morbid conditions with which it might be confounded; indeed, I do not know of any other disease that can be compared with it in the importance as well as in the difficulty of forming a right judgment. The liability to error arises in two different ways, which it is well to keep distinct; sometimes from the absence of any history as to the origin of the patient's attack, or from the history being vague or untrustworthy; sometimes from the doubtful nature of the symptoms.

Thus a man may be discovered comatose and stertorous in bed or upon the pavement of the street, and there may be no one to say what has happened to him; or it may be impossible to depend upon the statements of those in whose company he is found, or there may be suspicion of foul play. In all such cases three alternatives must be taken into careful consideration before deciding that the case is one of apoplexy; these are (1) that his brain may have been injured by external violence, as by a blow or a fall, with or without fracture of the skull; (2) that he may have taken poison; (3) that the patient may be intoxicated. We must also bear in mind (4) the possibility of the comatose condition being due to pyæmia, to uræmia, or to the effects of an epileptic fit.

1. As regards *injury to the brain* difficulties arise in several ways. There may be no bruise upon the face or head, and no displacement of any part of the calvaria, and at the *post-mortem* examination we may for the first time discover that the skull is fractured, or that the prominent parts of the brain on one side are bruised in such a way as to show that external violence had been the cause of death. Even then, however, one is by no means justified in concluding that the case was originally one of accident or of injury inflicted by others. The man may have had a fit, or he may have been drunk, and in falling he may have fractured his skull. Thus in 1859 a man was admitted into hospital who was driving in a cart when he

fell and was picked up insensible. He died after four days, and the surface of the brain was found to be extensively bruised, but as he was known to be subject to epilepsy, it appeared clear that one of these attacks had been the cause of his falling. Again, we must bear in mind that meningeal hæmorrhage often occurs as the result of disease; it is, therefore, only when the brain-substance itself is ecchymosed that we are justified in attributing to external violence cases in which blood is found effused on the surface of the brain. On the other hand, we have had several instances in which a severe injury of the skull with laceration of the cerebral convolutions has been associated with hæmorrhage into the central parts, exactly like that which occurs in apoplexy. Some of these have undoubtedly been examples of spontaneous effusion of blood into the brain, the superficial injuries having been caused by the patient's falling upon a kerbstone or a hard floor, or the like, but it is certain that a blow or fall upon the head may in rare cases cause rupture of an artery in the interior of the organ. Thus in 1866 a man was admitted into a surgical ward an hour before his death who had been steering a barge on the Surrey Canal when a steamer came too close and struck the rudder; the tiller knocked him over, and he was picked up insensible. There was no injury to the cranium, nor to the surface of the brain. The right lateral ventricle was found full of effused blood, the septum was broken down, and there was a moulded clot in the fourth ventricle. In 1868 a patient in the ward for ophthalmic cases, being unable to see, missed his footing and fell, striking his left temple. A large quantity of blood was effused upon the cerebral convolutions, but the right corpus striatum was also extensively ecchymosed, and had in its interior a mass of blood of the size of a hazel-nut. In 1870 a man came to the hospital with a cut on his forehead, saying he had slipped upon a flight of stone steps at the Victoria Theatre. He ultimately became hemiplegic and comatose, and died, and the right lateral ventricle was found to contain a large clot, the blood having oozed from a rent in the thalamus. In 1855 a patient was admitted who had been found lying by the side of a crane, the handle of which seemed to have struck him on the head, there being a wound of the scalp. At the autopsy the left corpus striatum and thalamus were discovered to have been destroyed by a large mass of blood lying between them. Sometimes it is impossible to decide whether a case is one of injury or disease. In 1862 a man was admitted who had fallen several feet upon his head from a platform on which he had been working. His skull was fractured, but it was found that the lateral ventricles were full of blood, and that the right corpus striatum was broken up by a large clot which projected through an opening in its surface. The opinion of his fellow-workmen was that he had overbalanced himself, as he had shown no sign of having a fit, but Dr Wilks, who made the autopsy, left the question an open one in his report.

It will be noticed how closely the appearances found in these five cases resembled those of spontaneous cerebral hæmorrhage. Now, in the reports of three of them it is distinctly stated that the kindneys were granular, or wasted, or cystic; in the other two the state of these organs was not noticed. In other words, although the rupture of the artery was the direct result of a blow or fall in each instance, it yet seems that they may fairly be regarded as examples of ordinary apoplexy, the main cause having probably been indirect, namely, the existence of chronic disease in the coats of the vessel. Most likely if no accident had happened these patients would have died of spontaneous hæmorrhage into the brain a few weeks or months

later. One cannot be surprised that it should be impossible to diagnose such cases in the absence of a history during life, when after an autopsy their right interpretation is so difficult.

2. Narcotic *poisoning* may be mistaken for apoplexy, or *vice versâ*. We have seen that in some instances in which blood is effused into the pons Varolii, the pupils are contracted and the respiration is regular and very infrequent, just as in persons who have taken large doses of opium. I have no doubt that I once made an error of diagnosis in a case of this kind. I was summoned one night to see a lady some six or seven miles away from London, and I found her comatose, with the symptoms I have just mentioned. There was a suspicion that she had poisoned herself, for the day was the anniversary of the death of a son of whom she had been very fond, and a few hours before her illness had commenced she had said to a sister who had come to visit her from a distance, "Well, have you come to see me die?" or words to that effect. But I was told that she was sitting up in bed talking to this sister, and apparently as well as usual, when she *suddenly* fell back comatose. This fact seemed to me to exclude the possibility of her having taken opium; and I, and her medical attendant also, came to the conclusion that there was hæmorrhage into the pons. I had to sleep in the village, the last train having left for London, and in the morning I went to call at the lady's house expecting to hear that she was dead. To my surprise I found her quite well again. She never confessed that she had any knowledge as to the cause of her illness. The case is interesting as showing that a patient deeply comatose from opium *may* recover without being walked about or swallowing strong coffee or stimulants. It so happens that I have at different times been called to two persons, both possessed of some medical knowledge, who had taken fatal doses of prussic acid, but who were alive when I saw them. In one case, which has been recorded by Dr Stevenson ('Guy's Hosp. Rep.,' 1869), death did not occur for more than an hour and a quarter after the discovery of the fact that the poison had been swallowed. I do not think that the symptoms could have been distinguished from those of apoplexy, but all difficulty was removed by the fact that there was on the table a half-empty, although corked, bottle containing some of the poison. A marked odour of prussic acid was diffused through the room, but the patient's breath seemed not to smell of it.

3. It is often difficult, and even impossible, to distinguish apoplexy from *alcoholic intoxication*. A man who really is dead drunk may be supposed to have cerebral disease, but I think it may be said that the only result that is likely to follow from this mistake is that a patient who had been expected to die, or at least to become hemiplegic, should in the course of a few hours completely recover. A man may, indeed, kill himself by drinking an immense quantity of spirits. In 1868 a boy, aged fourteen, was admitted into hospital under my care, who, with another boy, had stolen two bottles of brandy, and, according to his companion's statement, had drunk a reputed quart (twenty-six and two thirds fluid ounces) without any admixture with water. This occurred at about 2.15 p.m.; by 4 o'clock he was insensible, and he was at once taken to the hospital. He was comatose, but without stertor; his pupils were at first of natural size, but they afterwards became contracted. An œsophageal tube was passed, and the contents of the stomach were pumped out, with a most powerful odour of brandy. He was then put to bed, and an enema of coffee was adminis-

tered. He remained unconscious for twelve hours, at the end of which time he asked for a glass of water. Dr Stevenson, commenting upon this case in the 'Guy's Hosp. Reports' for 1869, says that no doubt the boy owed his recovery to the prompt use of the stomach-pump, and in all probability he is right. But it very rarely happens that anyone swallows a fatal dose of alcohol, except for a wager or out of bravado, when the medical man who is called to the case is sure to be told of what has occurred, and it appears to me that to mistake intoxication for apoplexy is to commit an error which is scarcely ever likely to be injurious to the patient, even though one should omit to use the stomach-pump. On the other hand, to suppose that a man who has apoplexy is merely intoxicated is a most serious matter. This is the mistake which is so apt to be made by the police, and into which medical men themselves have too often fallen, from failing to appreciate the real difficulties in the diagnosis between the two conditions. Perhaps the patient is found in a public-house, or is known to have been drinking heavily, or smells strongly of spirits. The only safe course for us is to ignore these facts altogether. The odour of brandy, indeed, does not even prove that any stimulants had been taken before the attack commenced; it may have been administered afterwards. I would lay it down as a rule, which should never be departed from, that a man who is insensible, and who cannot be roused, is never to be sent away to the police-station nor be left by himself to sleep off the supposed drunkenness. He should always be put to bed and be carefully watched. The presence of convulsions, hemiplegia, or inequality of the pupils may make one certain that the case is due to something beyond alcoholic intoxication, and the same conclusion may be drawn, although with less confidence, from the occurrence of rigidity of the limbs, or of stertor, but the *absence* of all these symptoms is no reason for supposing that there is no cerebral disease.*

After all, however, the most important cases are not those in which coma, or even partial unconsciousness, is present, but rather those in which the patient is noisy and excited, throwing his limbs about restlessly in all directions; one is perhaps sure that such a man is drunk, but it may be impossible to say whether or not he also has hæmorrhage going on within his skull. I have already remarked that the giving way of a cerebral artery seems often to be the direct result of indulgence in drink, and to detect its early symptoms must obviously be exceedingly difficult under such circumstances. Medical men who have had the largest experience are those who most freely acknowledge the impossibility of speaking confidently about cases of this kind, particularly when the patient is advanced in years, or is likely to have chronic disease of his heart, or of his kidneys and cerebral arteries.

4. Even when we have decided that a patient who is comatose, is not suffering from an injury to the head by external violence, nor from narcotic poisoning, nor from the effects of alcohol, there are still some other questions to be considered before we can safely conclude that he has apoplexy, as

* It would seem that by testing the urine one can obtain positive evidence that a poisonous dose of alcohol has been swallowed. According to Anstie, this inference may be drawn if to fifteen drops of his chromic acid solution (made by dissolving one part of bichromate of potass in 300 parts by weight of strong sulphuric acid) one drop of urine is added and turns immediately of a bright emerald green colour. In the case of the boy who was under my care in 1868, it took two drops to produce this effect, one drop giving only a greenish-yellow tint. Of course this reaction cannot enable one to decide whether a man may safely be sent away from a hospital, since it shows that a large quantity of alcohol, which may be dangerous, has been taken.

I have defined at p. 585. One possibility is that the case may be one of *pyæmia*. Strange as it may appear, I have seen two cases in each of which a profound and rapidly fatal coma was proved by the autopsy to be due to this cause. One was that of a man, aged twenty-six, who was brought to the hospital insensible, and died in a quarter of an hour. Very little could be learnt about him, but it was stated that he had very recently arrived on board ship in the port of London, and that he had been giving evidence in a court of law on the day of his death. The brain appeared healthy, but there was suppurative inflammation of the peritoneal cavity, and also of one knee-joint. The other case occurred in a girl eleven years old. She was admitted into the hospital one morning at half-past six, and, immediately after being put to bed, she gave one gasp and died. Her mother said that she had been quite well until the previous day, when she complained of pain in the left side on returning home from school in the afternoon. She went to bed and fell asleep, but at 4 a.m. (two hours and a half before her death) she was found to be unable to speak, and to be rolling over and throwing her arms and legs about. When brought to the hospital she was unconscious, and was breathing heavily with froth upon her lips; the right pupil was dilated, the left contracted. The temperature was 101·2°, the pulse 116, the respirations 32 in the minute. All that I could discover at the *post-mortem* examination was that the surface of the right lung was ecchymosed, and that there was a partial hepatisation of the lower lobe of the left lung, with much lymph effused on the pulmonary pleura over a circular area of the diameter of a Tangerine orange. The probable interpretation of these cases is that there was some early change in the brains or in its membranes, of so intense a kind as to kill before it could be recognised by the naked eye. We shall see that tubercular meningitis may prove fatal at a stage when the microscope is required to demonstrate its presence.

5. Another possibility is that the coma may be due to *uræmia*. The older pathologists were familiar with cases in which, finding no blood effused upon the brain, they were driven to suppose that the cerebral symptoms which had proved fatal were due to the presence of fluid in the ventricles and beneath the arachnoid; and they described the affection under the name of "serous apoplexy." At the present day, however, this name, and the idea on which it was based, have alike fallen into oblivion. Several years ago Dr Wilks suggested that the majority of the cases in question were examples of Bright's disease; and his opinion has been endorsed by all later writers. But I must confess that in reading the detailed clinical reports given by Abercrombie I fail to see that the cause of death in his cases was uræmia, rather than some one of the less obvious cerebral lesions, which at that time would necessarily have escaped recognition. And, so far as I can make out, there have within the last few years been very few cases at Guy's Hospital in which the symptoms were like those of an apoplectic attack, and in which the only disease that could be discovered at the autopsy was in the kidneys. On the other hand, it has very often happened that albumen has been detected in the urine of a patient who had been brought into the ward in a state of insensibility, and that the seizure has consequently been attributed to uræmia, until a *post-mortem* examination showed that it was due to cerebral hæmorrhage. The common occurrence of albuminuria in cases of this kind might naturally be ascribed to the frequency with which the kidneys are found to be granular.

But that particular renal affection comparatively seldom leads to the presence of albumen in the urine in any considerable quantity. Moreover, this symptom is often met with in apoplectic patients whose kidneys are afterwards proved to be perfectly healthy. Thus, it is probably to be regarded as a result of congestion of the venous system in general. And certainly no importance should be attached to it, whether in diagnosis or in prognosis. Among the few instances in which an apoplectiform seizure has been found in the *post-mortem* room at Guy's to have been caused by kidney disease, no cerebral lesion being discoverable, I may cite the following:

In 1867 a man, aged forty-eight, was brought to the hospital, conscious, but unable to speak; his friends said that he had often suffered from headaches; the day before his admission he complained that his mind was wrong; his manner was strange, but he answered when spoken to. Afterwards he became insensible, passing his excretions under him. At one time he regained his senses sufficiently to say "yes" and "no;" and he could swallow, but he soon relapsed into coma, and died on the following day. His pupils were rather contracted, and his breathing was laborious. The brain was found pale and anæmic; the ventricles were nearly empty. The heart was hypertrophied; the kidneys were extremely wasted, as a result of dilatation of the calyces secondary to stricture of the urethra. In 1868 a man, aged forty-four, who had been intemperate and gouty, and who had been suffering from a severe headache for a fortnight, was brought to the hospital in a drowsy state, from which he could just be roused to speak. He had had a fit, and had bitten his tongue. His body and limbs were in a state of constant jactitation. His pupils were rather contracted. His urine was retained, but his fæces were passed into the bed. Before his death he had two more fits. The only changes found in the brain were that it was tough and wasted, with large ventricles, and with an excess of fluid in them. But the kidneys weighed only two ounces and a half, and were exceedingly granular. At the bedside it would probably have been impossible to say, in reference to either of these cases, that an artery had not given way within the brain. But I find only one or two similar instances in our records. It has sometimes happened that patients already under treatment in the hospital for Bright's disease have died quickly with cerebral symptoms; but under such circumstances hæmorrhage is comparatively infrequent. And in the immense majority of cases the stupor caused by uræmia alternates with convulsions, and passes off again and again without leaving hemiplegia,—in such a way as to show that no considerable damage has been done to the actual structure of the brain. I quite admit that when a patient, of whom one knows nothing, is brought to a hospital insensible, uræmia is a possible cause of the coma. But if one should be told that he has not previously had any alarming cerebral symptoms, and if he should steadily get worse and die, it is most improbable that anything but disease of the kidneys will be discovered at the autopsy.

Coma from *diabetes* or from *cholæmia* may simulate apoplexy, but the presence of glycosuria or of jaundice will decide the nature of such cases.

6. Of cerebral diseases the one which is most likely to be mistaken for apoplexy is a form of *epilepsy* which was described by Andral and other French writers as a separate malady, under the ponderous name of "apoplectiform cerebral congestion," until Trousseau pointed out its real nature and distinguishing characters. In all probability Abercrombie would have

included it under what he termed "simple apoplexy," in which after death no morbid appearance could be discovered in the brain. Indeed, I remember that Sir William Gull in his lectures used to teach that "simple apoplexy" was nothing but epilepsy; but a careful perusal of Abercombie's cases leaves one doubtful whether all of them can be fairly interpreted in this way, and whether, if precisely similar ones were to occur now, the more accurate pathological methods which we possess would not enable us to place them in different categories. The question, however, is not of any practical importance.

Among the cases related by Trousseau are the following:—In 1845 a gentleman, aged forty-two, was found in his bed insensible; his face was turgid and livid, there was stertor, and all power of motion and sensation was lost. How long he had been in this condition his wife could not tell—she had been awakened by a strange snoring noise. Trousseau had the patient placed in a half-sitting posture, threw cold water in his face, and applied ligatures round the upper part of the thighs to retain the blood in the legs. Scarcely one hour elapsed before he regained his senses and the use of his limbs, and on the following day great lassitude was the only remaining symptom. Some time afterwards the same physician was fetched in great haste to a neighbour, aged seventy, who was said to have been attacked with apoplexy on the Boulevards. He had been unconscious for a quarter of an hour, but was recovering his senses when Trousseau arrived. He did not at first recognise him, and looked vacantly round, throwing his arms and legs about without knowing what he was doing. By degrees, but within a few hours, he recovered entirely, although no active treatment was adopted. In both instances the real nature of the disease was afterwards established by the recurrence of the seizures at more or less frequent intervals, attended with all the symptoms of epilepsy. Indeed, it must be observed that in each case the attack began when no skilled observer was present, so that there is no proof that spasmodic movements did not occur. And Trousseau himself goes on to say that in almost every instance of the same kind in which he was consulted, and in which the commencement of the seizures had been seen, "nervous twitches" or convulsions had been present. Thus, after all, the identification of "apoplectiform cerebral congestion" with epilepsy involves little more than the recognition of two facts, that the spasms which usher in an epileptic seizure may be but slightly marked, so as to escape the notice of a non-professionsl observer, and that when one is called to a patient who is comatose one must make sure that the disease is *not* epilepsy before committing oneself to a diagnosis of apoplexy. And in determining this latter point the first thing to be ascertained is whether the patient has ever before suffered from any seizures which could be regarded as of an epileptic nature. I was one evening called out in great haste to see an old man living about a mile from my house. I found him lying on the sofa in his sitting-room, comatose and stertorous, with puffing cheeks and a purple countenance. His pupils were dilated, but I should certainly have thought that he would probably die in a few hours had not his housekeeper, who had found him insensible, told me that he had once or twice before had epileptic attacks. Before long he completely recovered. One of Trousseau's cases is that of a solicitor, aged thirty-five, who was sent to him from the country, with the history that in the course of the previous six months he had had three apoplectic fits. They had lasted, however, an hour at the most, and they had left no

paralysis behind them. Trousseau accordingly declared the disease to be epilepsy, and his diagnosis was before long found to be correct. But of course there must in every instance be a first attack, and then the criterion fails, and if this seizure should prove fatal the real nature of the disease must remain a matter of inference. In 1865 a man, aged sixty-seven, a tanner in Bermondsey, was brought into the hospital comatose and died in a few hours. He had followed his occupation until the day of his death, when he was found insensible, having fallen to the ground. He had convulsive movements and appeared to be paralysed on the right side. It could not be ascertained that he had ever before had a similar attack, but he was said to have suffered much with his head. No recent morbid change could be found in the brain, but there was chronic wasting, the convolutions being shrunken, with much fluid in the sulci, and the ventricles being enlarged and their surface granular; the skull also was dense and had no diploë, and the membranes were thickened and opaque.

A very similar case occurred in 1876. A woman, aged sixty-two, was brought into the Clinical Ward, having fallen down in the street in a fit. The right pupil was larger than the left. There was right hemiplegia, with complete anæsthesia in the right arm and in the right side of the face. After a time she partially regained her consciousnesss, but had several convulsive attacks. The coma then again became deeper, and she died at the end of five days. No morbid change could be found in the brain except wasting with dilatation of the ventricles; and there was only slight wasting of the kidneys.

There appears to have been no marked engorgement of the cerebral vessels in these instances; but from the way in which death is brought about it is evident that in similar cases such a condition is very likely to be found. And for my own part I should be inclined to regard as examples of epilepsy the cases which some writers (for example, Hammond) describe as instances of fatal congestion of the brain.

There is a further criterion by which we may often distinguish an attack of epilepsy from one of apoplexy, and on which Trousseau has laid special stress—the mode of onset of the coma. We have seen that it is rare for a patient labouring under cerebral hæmorrhage to fall down suddenly, deprived of sense and motion, and to remain persistently comatose. There are indeed cases in which apoplexy begins with a fit which is perfectly epileptiform in character; but such cases are exceptional. The practical rule, therefore, is that if we are informed that the commencement of a seizure was gradual, we may safely conclude that the case is not one of epilepsy; but, if it began suddenly, we may often feel doubt as to its nature. In such a case our prognosis must be most cautiously guarded; a few hours later the patient may be apparently as well as ever; or he may be dying.

7. Several of the *organic diseases* to which the brain is liable may occasionally give rise to a rapidly fatal attack, resembling an apoplectic seizure in its symptoms. This is the case, for instance, with cerebral abscesses and tumours, and even with tubercular meningitis in certain instances. Sometimes, indeed, cerebral hæmorrhage does really occur as a complication of the softer forms of tumour, the blood-vessels within the growth giving way, and pouring a large quantity of blood into its interior. It is not improbable that in many of the cases in which hæmorrhage has been found in the cerebellum, or in some other unusual seat—the patient being perhaps young and the arteries healthy—the original disease has been a soft tumour

of small size, which has been torn up by the extravasated blood and overlooked at the autopsy.

Diagnosis between the anatomical causes of apoplexy.—We now come to the differential diagnosis of the several lesions, which are each capable of causing apoplexy, aphasia, or hemiplegia: namely, hæmorrhage, embolism, thrombosis, and syphilitic disease of the arteries. We must first rectify what may well have seemed a strange omission, by giving an account of the clinical history of the last three of these diseases. It has been remarked at p. 557 that under these heads are included a large proportion of those cases which were formerly grouped together under the name of "cerebral softening." And since the older pathologists taught that the principal indication of that supposed morbid state was the occurrence of paralysis without loss of consciousness, it may appear that the diagnosis from cerebral hæmorrhage ought to be simple enough. But a more extended experience has shown that the mere arrest of the circulation through one of the large cerebral arteries often causes coma as profound as that which is produced by extravasation of blood. We must therefore break up the description of the symptoms of embolism and the allied lesions into two parts, according as the patient becomes unconscious (*apoplexy*) or retains his senses (*hemiplegia*).

1. For example, among the cases of *cerebral embolism* that have occurred within the last few years at Guy's Hospital I find the following clinical histories:—In 1868 a man, aged forty, was brought into the Clinical Ward under my care, with paralysis of the left side. He had suddenly fallen down insensible while wheeling a barrow. He presently regained his senses and conversed about his symptoms; and then he again became comatose, and remained so until he died a few hours afterwards. In 1869 a man was taken in for paralysis and partial anæsthesia of the left side; he was reported to have suffered from continuous headache for two or three weeks; a few days before his admission he had suddenly become giddy and lost the use of his left arm and leg; afterwards he was unconscious for two or three hours. In the same year a woman, who was in the hospital suffering from dropsy, had a fit which was followed by aphasia and right hemiplegia, and she died within two days. In 1874 a woman, who was in the ward for uterine cases under Dr Braxton Hicks, became comatose with left hemiplegia, and remained insensible until she died on the following day. In none of these instances was there any hæmorrhage into the brain nor any change beyond softening of the part which should have received its supply of blood through the obstructed vessel. Yet, if no autopsies had been made, these very cases might well have been quoted as illustrating the chief varieties in the mode of onset of an apoplectic seizure due to the rupture of a cerebral artery. Nothnagel, indeed, has already stated in the most explicit terms that there is no absolute difference between the symptoms of embolism and those of hæmorrhage. And there can be no question as to the inadequacy and the trivial character of certain points of distinction which he mentions as having been suggested by earlier writers; namely, that in the former affection the face should be pale rather than red; and that the latter should be accompanied by excessive pulsation of the carotids, stertorous breathing, and inequality of the pupils.

Nothnagel quotes Dr Eliza Walker as having shown in her inaugural dissertation at Zürich in 1872 that embolism is very often ushered in by an

epileptiform attack; this was the case in twenty-four out of ninety-seven cases which were collected by her. In one point, indeed, the German writer seems to me to be in error; namely, when he says that the affection in question can never have any prodroma. This statement is contradicted by one of the cases already cited, in which the attack was preceded by headache for two or three weeks.

2. Among the cases that have been regarded as examples of spontaneous *thrombosis of a cerebral artery* at Guy's Hospital there have been the following:—A man, aged forty-eight, was attacked with pain in the head a month before his death; he fell back insensible, with right hemiplegia; afterwards he could walk, but he ultimately again became semi-conscious, and rambling in his talk. Another man, aged thirty-five, was attacked with unconsciousness and paralysis of the left side six days before his death; he then had convulsive fits, in one of which he died. Apoplexy due to this affection cannot be distinguished from that of ordinary cerebral hæmorrhage.

3. As regards *syphilitic* disease of the cerebral arteries, the cases related by Heubner in his monograph suffice to show how closely its symptoms may resemble those of rupture of an artery in the brain. In eight cases out of twenty-two collected by him, the symptoms were ushered in by an attack of apoplexy. In some rapidly fatal cases the insensibility lasted until death. In others there was a transitory loss of consciousness, accompanied by hemiplegia, and followed after an interval by the supervention of coma from which the patient never again awoke; in other words, the clinical features of an "ingravescent" seizure were closely imitated. Case 33, for instance, is that of a man who had suffered from headache and sleeplessness, and who one day, after having appeared to be as well as usual in the morning, was found later on to be comatose and paralysed on the right side; he could at first be roused a little, so as to make an attempt to open his eyes; but afterwards the insensibility became more profound, the face flushed, the pupils immoveable, and he died the next evening. Case 47 is that of a journeyman furrier, admitted into the Leipzig Hospital in a state of unconsciousness. He had been attacked on the previous day with paralysis of the arm; this came on gradually, so that he watched its progress upwards from the thumb to the shoulder, being then in perfect possession of his senses, and able to point out with his left hand how high the numbness extended at any particular moment. There are, however, comparatively few syphilitic cases in which the symptoms and course resemble those of cerebral hæmorrhage.

Diagnostic results.—Thus,—summing up what has been stated in the last four paragraphs,—we find that an attack of *Apoplexy* is by no means necessarily due to the extravasation of blood in the brain. Omitting spontaneous thrombosis, on account of its rarity, we have embolism and syphilis as possible causes of affections of the cerebral arteries which may sometimes give rise to precisely similar symptoms.

The diagnosis is based entirely upon collateral circumstances. Age is an element of the highest importance. In persons more than fifty years old, one is seldom wrong in attributing an apoplectic seizure to hæmorrhage; and the younger the patient the greater the probability that one of the other two causes is in operation. The whole of the body must then be searched carefully; on the one hand, for eruptions on the skin or fauces, enlargements of the testicles or lymphatic glands, and nodes; on the other hand, for indications of cardiac disease. It is well to remember that

cerebral hæmorrhage may itself be an indirect result of embolism, in consequence of the formation of an aneurysm in the obstructed artery. In most cases of this kind the primary disease is ulcerative endocarditis, and the embola possess septic characters; so that the spleen may be found enlarged and tender, or albumen or blood in the urine may suggest the correct diagnosis.

The diagnosis between the several causes of *Hemiplegia* in a patient who is conscious at the time when he comes under our observation must now be considered. The first question that one asks in such a case is whether coma was present when the attack of paralysis occurred. If so, the considerations stated in the last paragraph are applicable. In general it may be said that in proportion to the duration and the severity of apoplectic symptoms, is the probability that there was rupture of an artery greater. On the other hand, if the seizure was unattended with any, even transient, loss of intelligence, the presumption is strongly in favour of its being the result of a mere arrest of circulation in some part of the motor tract. This, however, may itself be dependent either upon embolism of a cerebral artery, or upon syphilitic endarteritis, or upon common chronic atheromatous arteritis. As I have already pointed out, atheroma very often leads to the formation of patches of white softening in the substance of the brain; and that morbid change, if it affects the motor region, is exceedingly likely to cause paralysis. Now, on *a priori* grounds one would have expected that a point of considerable diagnostic importance would have been the insidious and gradual commencement of hemiplegia due to such a cause. But experience shows that this is not the case; the attack is generally sudden. It is probable that the nervous elements continue to perform their functions up to a certain point, notwithstanding impairment of their structure; and that several of them at length suddenly and simultaneously give way.

Hence, the chief way of distinguishing from one another the various lesions of the brain that may give rise to hemiplegia lies in a careful study of the other nervous symptoms that may happen to be present. We have already seen the diagnostic value of the association of aphasia with hemiplegia, as showing that the Sylvian artery is obstructed, whether by embolism, thrombosis, or syphilis. Again, it is probable that persistent hemianæsthesia, when associated with hemiplegia, points to hæmorrhage, or at least to atheromatous change affecting a number of the small cerebral arteries, rather than to the other morbid conditions just referred to. For such a combination of symptoms shows that the posterior third of the internal capsule is involved; and this lies towards the periphery of the area of distribution of the vessel in question, and therefore might probably be fed from another source, if arrest of the blood supply were the cause of the hemiplegia. At any rate, I find that most of the cases hitherto recorded, in which loss of sensation has been permanent, have occurred in old people, so that rupture of an artery was probably the cause of the symptoms. Among fifteen fatal cases collected by Veyssière in 1874 there is only one in which the patient was young, that of a girl, aged twenty-two years; in several instances, indeed, the lesion is described as having been a patch of *ramollissement;* but no details are given which would have thrown light upon the possible causes of the softening

On the other hand, I have already pointed out that syphilis is apt to affect several of the great cerebral arteries, simultaneously or in succession. It is therefore not surprising that hemiplegia dependent upon this cause is sometimes accompanied by a series of incongruous symptoms, which cannot

be referred to a lesion limited to any one spot in the brain. Thus there is sometimes a complete paralysis of one or more of the cranial nerves, or the patient may regain the use of the arm and leg which were first affected; and subsequently he may be attacked with loss of power on the opposite side. This, indeed, is not in itself characteristic of the syphilitic affection; for old people with atheromatous vessels are very liable to have patches of softening develop themselves in the two hemispheres in succession, or to suffer from the effects of small hæmorrhages into both corpora striata in turn. But I think that symptoms due to syphilis are more apt to be irregular in their course than those which depend upon other causes; the patient perhaps becomes able to stand, or even to walk, and then after a few days relapses; and such changes may occur again and again. Heubner lays great stress on a peculiar somnolent condition, as indicating the presence of syphilis: the patient, he says, is half awake, half asleep; he lies with his eyes shut, taking no notice of anything, and refusing to answer questions; but when one tries to examine him, he resists and turns round in his bed. That such symptoms are comparatively rare in cases of embolism or of cerebral hæmorrhage is possible. But certainly they are common enough when the disease is a tumour or some other local lesion. And one must always remember that these affections may also give rise to hemiplegia, if they are so seated as to involve the motor tract. There is, in fact, no one of all the morbid changes to which the brain is liable that may not sometimes induce loss of power in the arm and leg on one side. And we shall hereafter see that other possible causes of hemiplegia are epilepsy, hysteria, chorea, and some other neuroses. Conversely, it occasionally happens that affections of the cerebral arteries may run their course to a fatal issue, without hemiplegia developing itself. Such cases may be attended with headache, loss of memory, drowsiness, delirium, vomiting, thickness of speech, difficulty of swallowing, involuntary evacuations, and a variety of other symptoms. A diagnosis is generally impossible.

Prognosis.—The gravity of a case of Apoplexy may be judged of first by the depth of the coma; the ingravescent cases are the worst, while gradual recovery of consciousness is of good omen. In cases comatose from the first, death frequently follows on the third day, but it may occur after a week or even longer, and on the other hand recovery may follow prolonged unconsciousness if the pulse and breathing are not seriously affected, and if the patient is not fed. Rise of temperature is a bad sign. So are early rigidity or convulsions of the paralysed limbs, flapping of the cheek in breathing, insensitive conjunctivæ, and increasing cyanosis.

Treatment.—That the treatment of Apoplexy is unsatisfactory is universally admitted. There is no disease in which it is more difficult to tell whether the chance of recovery is affected by therapeutical measures of whatever kind. Rest is always essential; the patient should, if possible, be left in the room in which the seizure occurred; a small bed may be made up for him, but a mattress placed on the floor does perfectly well for a time. His head and shoulders must be raised, and he should be turned over on his side, so as to prevent the tongue from falling backwards against the pharynx. In cases in which it is clear that death is actually impending one ought to abstain from all active treatment. I fail to see what good could possibly be done by the application of a blister to the neck, or of mustard plasters to the calves.

A patient whose coma is so deep as to threaten his life cannot be roused by such means; and if he is capable of feeling, the irritation must surely be injurious rather than beneficial. When the case appears not to be altogether hopeless, five or ten grains of calomel, or two drops of croton oil should be placed on the tongue, particularly if there is reason to believe that there is an accumulation of fæces in the bowels; or an enema of turpentine or castor-oil may be given. One must be on the watch for the necessity of passing the catheter if the insensibility should last more than a few hours. Whenever there is serious difficulty of swallowing, the administration of food or drink by the mouth should be altogether forbidden, on account of the danger of its running into the air-passages and setting up pneumonia. No harm results from keeping an apoplectic patient for a day or two without nourishment; but if it is thought desirable, enemata of beef-tea or milk may be employed. The lips and mouth may be kept moist with a feather.

Bloodletting, whether by venæsection, cupping or leeches, is now seldom practised. Sir Thomas Watson, indeed, speaks of patients so insensible as not to feel the puncture made by the lancet, who have yet emerged from their coma while the blood was flowing. But it may be fairly doubted whether in such cases the disease was not rather that "simple apoplexy" which is now known to be really a form of epilepsy, and in which a spontaneous recovery of consciousness often occurs with unexpected rapidity. If the pulse be large and labouring, the face flushed, the carotid arteries full and throbbing, it may be thought right to bleed. But the majority of patients suffering from cerebral hæmorrhage are advanced in years and impaired in health, so that one hesitates to employ a practice which can be of service only by its general effect in lowering the pressure within the vessels of the brain. And in a younger person the coma may be due to embolism or to syphilitic arterial disease, in which conditions the maintenance of a vigorous cerebral circulation is essential to recovery.

There is, however, one particular set of cases, in which it seems probable that judicious management may sometimes prevent a fatal issue. I refer to those instances in which the symptoms of cerebral hæmorrhage are slowly "ingravescent." When one is called to such a patient in the early stage (that of collapse), one should keep him absolutely recumbent, with head and shoulders raised, and he should neither be allowed to speak nor to move. The limbs may be warmed by friction with hot flannels, but the administration of brandy, and even of ammonia, must be rigidly abstained from. As reaction comes on, the question of bloodletting must be most seriously weighed. It is true that a rapid death almost always occurs, and that the autopsy generally confirms the opinion that the case was hopeless from the first. But I think it is possible that free venæsection, just at the time when the vigour of the circulation is being re-established, may now and then, by lowering the pressure in the cerebral vessels, prevent the further effusion of blood.

Hæmostatics, such as ergot, acetate of lead, or gallic acid, are not often prescribed; but Dr Bristowe assents to their employment, and one is certainly encouraged to try them by their apparent success in cases of hæmoptysis. Cooling lotions, or a bag of ice, may be applied to the head.

When coma is due to embolism the only treatment is to keep the patient in a state of the most perfect quietude, so as to reduce to a minimum the functional activity of the nervous centres, deprived of their blood supply. But in cases of syphilitic arterial disease it would appear that the inunction of mercury should be commenced with the first cerebral symptoms.

A person attacked with Hemiplegia without unconsciousness should for some days be kept in bed, and as free from all cares as possible. If advanced in years, or suffering under heart disease, he should be supplied with soup, beef-tea, milk, and perhaps a little wine.

The syphilitic patient should at first have rather a scanty diet while the proper treatment is being carried out: say a twelfth of a grain of bichloride of mercury, with ten or twenty grains of iodide of potassium. Heubner relates some remarkable examples of recovery of power in the paralysed limbs under the method of inunction. A striking case is that of a student, aged twenty-six, who after having suffered from headache and giddiness for some weeks was attacked one night with left hemiplegia during sleep. There were cicatrices on the penis, indurated glands in the neck and at each elbow, and a radiating scar on the velum palati. At the end of a fortnight he was no better; he was feverish, very prostrate, sleepless, and delirious; bedsores were forming; and spasmodic movements of the right arm were noticed. Mercurial ointment was therefore rubbed in for seven days, and then iodide of potassium was given in large doses. Six weeks after the commencement of this treatment he was able to leave his bed, and a fortnight later he could raise his arm to form a right angle with the body. Little by little he regained power in the affected limbs, and several years afterwards Heubner found him perfectly well, except that he used a stick in walking.

But in too many cases of hemiplegia—whatever its cause may have been—the limbs remain more or less completely paralysed, in spite of the most judicious treatment. Recourse is then had to electricity, which undoubtedly is sometimes beneficial. The continuous application of a weak galvanic current to the head, as suggested by Remak, is said by Nothnagel to be occasionally followed by a decided increase of power and diminution of rigidity in the affected muscles. Not more than from four to twelve cells should be brought into operation; the poles should be gradually applied and withdrawn, and should never be suddenly reversed; they should be kept in position for not longer than three minutes at a sitting; whether they are both placed behind the ears, or one upon one mastoid process and the other upon the forehead, seems to be a matter of indifference. Other methods are to galvanise or to faradise the muscles. Dr Reynolds lays down the rule that little or no good can be thus effected if the muscles contract to the normal extent. But if the contractility is diminished, electricity will often in the course of a few weeks restore it; at the same time the muscles will become less wasted, and the previously cold limb will regain its normal temperature. By faradising the extensor muscles of the fingers one may diminish contraction of the fingers into the palm. In general it may be stated that the application of electricity should not begin till two months from the hemiplegic seizure; that it should be at once left off if it causes headache, giddiness, faintness, sickness, or any unpleasant feeling at the epigastrium; and that the current should never be so strong as to cause pain, nor so long continued as to cause fatigue.

The internal administration of strychnia or other drugs is, probably, useless. Nor can one reasonably expect any benefit to result from the baths of Gastein or Pfeffers. To prevent a recurrence of the attack, temperance both in eating and drinking, a loose state of the bowels, and the avoidance of emotional and other excitement, both of mind and body, are the rational and probably the only measures to be advised.

TUMOURS AND LOCAL INFLAMMATION OF THE BRAIN

Tubercle—Syphilitic gumma—Glioma and other tumours—Symptoms: Headache and other cerebral symptoms—Vomiting—Temperature, pulse, and respiration—Optic neuritis—Localising symptoms—for the base—the cerebellum—the motor area and other parts—Diagnosis from hysteria, meningitis, &c.—Diagnosis between the several kinds of tumour—Prognosis—Treatment.

Red softening—Histology—Causes—seldom or never primary.

Cerebral abscess—*Ætiology—Anatomy—Symptoms: general and localising—Diagnosis—Event.*

In discussing in the last chapter the characters which enable us very often to determine with great accuracy in what part of the brain are seated lesions causing hemiplegia and aphasia, we entered upon one of the most interesting parts of the study of cerebral diseases; but we were not able to complete it, because the affections that depend upon lesions of the arteries rarely occur except in certain regions of the encephalon. There is, however, another important group of local organic affections which are under no such anatomical limitation of seat, and it will be in every way convenient to take them next; our doing so will save retracing our steps over any part of the ground already traversed, and it will place the reader in possession of the chief points that must guide him in what is termed the *localisation* of diseases of the brain. The group in question would not, indeed, be recognised as such by the pathologist; for pathology would place as far apart as possible affections so various in nature as syphilitic gummata, tubercles, tumours, and inflammatory changes leading to softening or abscess. But here, as elsewhere, we are compelled by the necessities of practical medicine to ignore strict pathological classifications. At the bedside one can often distinguish these several affections only imperfectly, and upon indirect or collateral evidence. To describe them separately would be to repeat the same facts over and over again, and to suggest false views with regard to their clinical significance. We will therefore begin with a brief account of such of the lesions as may be included among new growths, taking first *Tubercle*, secondly *Syphilitic Gumma*, and thirdly *Tumour*. Afterwards we will discuss their symptoms in common, the differential diagnosis between them, and their treatment. Lastly, *Red Softening* and *Abscess* will be described separately.

1. *Tubercle of the brain.*—The tubercular process may affect the brain in two different ways. Sometimes a number of minute tubercles grow into it from the pia mater, or are scattered through its substance along the vessels. This condition is always associated with meningitis, and will be described under that disease. In other cases there is a single caseating mass, which may reach a great size. By way of distinction some writers call these "solitary tubercles." Obernier has proposed to name them "tuberculous tumours." In size they

generally vary from that of a pea to that of a walnut; the biggest I know of is a specimen in the museum of Guy's Hospital, which was received from Dr Hughlings Jackson by Dr Moxon. It is described as being like a large potato. Sometimes they have a flattened base towards the pia mater, but more commonly they are more or less globular in form, and touch the surface only at one spot, or are completely surrounded by brain-substance. I do not know that they ever adhere to the dura mater lining the skull, but sometimes they become agglutinated to the tentorium when they occupy the cerebellum. Their substance is of a bright yellow colour, firm, or even hard, and generally quite homogeneous, except that at the centre it may be softening down into a yellowish liquid, or may, on the other hand, be partially calcified. But they also have, in most cases, a narrow pinkish-grey soft growing edge, which separates the yellow cheesy material from the brain tissue around. This edge may obviously consist of an aggregation of miliary tubercles, but more often it has no such appearance, and I think that it sometimes consists of elements which are larger than the lymphoid cells that characterise tubercles in other parts. Rindfleisch further insists on the fibrous texture of the marginal zone of the cheesy mass, and declares that many so-called cerebral tubercles are really non-tuberculous, deserving rather to be called fibroid tumours. But a similar transformation of the cellular elements of tubercles is well known to occur even in the lungs, and, whatever its microscopical characters, I do not think there can be a doubt that these solitary yellow tumours of the brain are histologically tubercular.

Among thirty-two cases that have occurred at Guy's Hospital I find only two in which it is stated that no similar lesion could be discovered in any other part of the body. It is true that tubercular meningitis (which was found in several instances) would by some pathologists be regarded rather as an accidental result of infection from the caseous mass than as independent; but in a very large number of instances there was chronic phthisis or disease of the mesenteric or mediastinal glands. In no less than eighteen of the thirty-two cases the cerebellum was the sole seat of the affection; three times it was the pons Varolii, once the medulla oblongata, six times one of the hemispheres; in the other four cases there were several tubercles in different parts of the encephalon. Twenty-one of the patients were males, the remainder females, a proportion which accords exactly with that given by some previous writers. In three instances the age was under five years, in six between six and ten years, in twelve between eleven and twenty years, in seven between twenty-one and thirty, in three between thirty-one and forty, and in one older still, namely, forty-two.

In one case, that of a child, aged four and a half, there is said to have been a fall upon the back of the head five or six weeks before the occurrence of a fit, which was the earliest sign of cerebral mischief; the seat of the tubercle was the pons. Dr Crichton Browne mentions ('West Riding Asylum Rep.,' ii) a similar instance; the same part of the head was struck, symptoms speedily showed themselves, and proved fatal in two months, and a mass of tubercles, of the size of a walnut, was found in the cerebellum. I should have thought, however, that in Dr Browne's case the time was hardly long enough for the development of such a lesion, and a suggestion made by Rilliet and Barthez seems to me very reasonable, namely, that when an injury seems to have been the cause of a tubercle in the brain, it may perhaps in reality have merely set up acute changes in the tissue around a tubercle which was there before.

2. *Syphilitic gumma of the brain.*—This affection also is attended with the formation of a firm, dry, yellow, caseous material, which, indeed, is not always easily distinguished from tubercle. One point of difference is the fact, long ago pointed out by Wilks, that it is constantly seated on the surface of the organ, growing into the cerebral substance from the membranes. Heubner states that an analysis of forty-five cases, recorded by different observers, yielded only three exceptions to this rule, and not one of these was conclusive. Again, when a gumma occupies the convexity of the brain, or the summit of one of the cerebral lobes, the corresponding part of the dura mater becomes thickened and converted into a tough yellow layer and adherent, so as to form with it one inseparable mass. Thus the shape of a gumma is far more irregular than that of a tubercle, which we have seen to be generally more or less globular. Lastly, the syphilitic affection commonly shows a much more considerable proportion of translucent undegenerated tissue; this appears as a moist greyish-red or grey mass, which is sometimes as soft as jelly; it is especially apt to be abundant at the base, where it may involve several of the cranial nerves and fill up the sella turcica as well as the diamond-shaped space. Microscopically it consists of granulation-tissue, containing some spindle-shaped and stellate elements, and having (according to Heubner) a distinctly alveolar structure in many instances. It is very vascular, and sometimes presents many little extravasations of blood. On the other hand, if antisyphilitic remedies should have been freely administered during life, the only morbid change found after death may be a patch of superficial softening of the cortex, beneath a local adhesion of the dura mater to the brain; this was the case, for example, in a patient of Dr Dreschfeld's ('Lancet,' 1877).

It is a curious circumstance that of ten cases of gumma of the brain of which I have notes all but one were in persons between the ages of thirty-one and forty, the exception being a woman, aged twenty-six. In five of them there was a definite history of constitutional syphilis, or else there were in the liver indisputable gummata; only one patient distinctly stated during life that he had never had venereal disease, and in that case the liver contained them. As might be expected, males preponderated over females in the proportion of seven to three. In several instances the report of the autopsy is incomplete, an examination of the testes, in particular, having often been omitted. In one case the patient's illness had been attributed during life to a sunstroke. This was, no doubt, a mistake, but many writers think that a blow or fall upon the head may determine the formation of a gumma in the brain, and it has even been imagined that an inherited tendency to diseases of the nervous system may dispose syphilis to affect that organ rather than any other part of the body; or, again, that a mental shock or excessive study may act as an exciting cause of such a lesion.

3. *Other tumours of the brain.*—There is no region of the body in which so many different kinds of new growth are found as in the brain and in its membranes; yet histological distinctions are nowhere else of so little practical importance, because the clinical history and symptoms of a case are seldom, and but slightly, affected by the nature of the tumour. A brief description of the chief varieties will therefore suffice.

a. The *membranes* may present fibrous and some other simple tumours, such as occur almost anywhere, and malignant tumours of all sorts, primary or secondary. A favourite seat for malignant growths is between the dura mater and the bone; or perhaps it would be more correct to say that they

begin within the osseous substance itself, and either push inwards the dura mater or penetrate it, protruding upon its inner surface, or invading the cerebral tissue. They generally are *sarcomata*. Two or three kinds of simple tumour are peculiar to the coverings of the brain. To one of them Virchow has given the name of *psammoma*, from its containing calcified particles, like those which constitute the brain-sand of the pineal body and choroid plexuses. The lime-salts are deposited in curious little globular structures, made up of elements arranged concentrically, so as to resemble somewhat the well-known "birds'-nest cells." Robin accordingly described this affection as a form of epithelioma; but such a view of its nature is incorrect. Virchow says it generally appears as a semi-globular mass, of firm consistence, pale red or white, and medulla-like. One that I examined had a loose texture and a flocculent surface; it grew in such a position as to indent the brain above the left Sylvian fissure. According to Virchow such growths are less frequently seated in the tentorium or in the falx than in the dura mater lining the skull—particularly at the fore part of the base. Another special form of tumour peculiar to the membranes is that which was formerly called a *cholesteatoma*, but which has of late been termed "pearl-cancer." It constitutes a dry, hard, rounded mass, occupying the pia mater generally at the base of the brain. Its cut surface has a pearly lustre, and it consists of lobules made up entirely of horny epithelial cells, and supported by a stroma of dense connective tissue. Lastly, Virchow has described under the name of *melanoma* a third kind of meningeal new growth; in a case which came under his observation there were numerous black or brown nodules in the pia mater, the rest of the body being entirely free from them.

β. Of the tumours that have their seat *within the brain-substance* there is one kind that is almost special to it, while the rest are like those which are found in other parts of the body. Among the latter *sarcomata* are the most numerous, especially those of the spindle-cell variety. A fibrous tumour is sometimes met with; and sometimes, though very rarely, a myxoma. Primary carcinoma of the brain is exceedingly infrequent. I find only one case in our records at Guy's Hospital which is said to have been of that nature. Indeed, it is not very common for the organ to be affected even secondarily by cancer; I have notes of only four instances of such an occurrence. In the immense majority of cases in which secondary nodules are developed within the cranium the growth is one of the more highly infective forms of sarcoma. A peculiarity of secondary tumours in the brain is that they are almost always multiple. On the other hand, it is a rule to which there are scarcely any exceptions, that a primary new growth is solitary. Among forty-four cases that I have collected from our records, I find only two in which there was more than one tumour in the brain, but in which no obvious source of infection existed elsewhere; and after all, it is quite possible that in those cases there was disease of one of the bones, or of some other structure which was overlooked.

The centre of a cerebral tumour now and then softens down into a *cyst*, and this may become so large that the presence of any solid growth may not be obvious. We have at Guy's Hospital had five cases in which one lobe of the cerebellum or the middle of it has contained a large thin-walled cavity, filled with a fluid that was highly albuminous or deposited spontaneously a fibrinous coagulum. In three instances no adventitious tissue could be discovered, except the vascular membrane forming the cyst itself

but in each of the other two there was a small tumour situated on one side of the fibrous wall. It therefore seems probable that such cavities always arise out of new growths. They are more rare in the cerebrum than in the cerebellum ('Path. Trans.,' 1885, vol. xxxvi, p. 17).

Hydatids are of very rare occurrence in the brain. In a girl, aged nine, Dr Moxon found a large *echinococcus*, occupying the middle and posterior lobes of the right hemisphere; she had also a similar affection of the liver. I believe that no other instance has been met with at Guy's Hospital during the last few years. The *Cysticercus cellulosæ* sometimes infects the pia mater or the ventricular space.

The one kind of tumour to which I have already alluded as almost special to the brain * is that which Virchow termed *glioma*, and which he regarded as an overgrowth of the neuroglia, the interstitial cement and support of the proper nervous elements. This substance has a somewhat indefinite structure, consisting of a granular or faintly fibrillated matrix, in which are embedded small nuclei with scanty protoplasm. Accordingly, a glioma consists of similar cells or nuclei, more or less thickly set in a similar matrix. Such a growth is not very unlike a sarcoma; and many pathologists describe it as a mere variety of that form of tumour. Indeed, Virchow himself admitted that it was often impossible to say of a particular specimen whether it should or should not be called a sarcoma, and he expressly recognised the occurrence of intermediate forms, which he termed glio-sarcomata. But although the distinction may not be of great practical importance, it appears to rest upon a sound theoretical basis, inasmuch as the neuroglia is a completely developed or adult structure, whereas the elements of a sarcoma are embryonic, corresponding with those of connective tissue at an early stage of its growth. Prof. Klebs, now of Zurich, maintains ('Prager Vierteljahrschrift,' 1877) that gliomata also contain newly-formed nervous elements. Certain cells with many-branched processes, which are well known to occur in these tumours, are regarded by him as ganglionic cells; and he believes that they are sometimes developed out of the axis-cylinders of nerve-fibres.

A glioma may be of a pinkish-red colour, or it may look so exactly like the normal brain-substance that the microscope is required to demonstrate its presence. Its substance is always continuous with that of the surrounding cerebral tissue; for there is never a capsule, as with some sarcomata. Indeed, it often assumes the form of the part in which it grows, so that one might imagine the corpus striatum or the thalamus, or some particular convolution, to have become swollen to three or four times its natural size. But in some cases extensive caseation takes place in these growths; and, according to Klebs, they are also liable to sclerosis, exactly like that which occurs in the normal brain-substance.

A soft glioma often contains a great number of thin-walled vessels, which may rupture and pour out blood into its tissue, tearing it up so that one can hardly discover any trace of the growth; as Virchow long ago pointed out, the disease may then be mistaken for a simple cerebral hæmorrhage (cf. p. 559).

Perhaps the most puzzling cases of all are those in which repeated extravasations occur, and in which the coagula become converted into tough opaque caseating masses of various colours.

Not only gliomata, but all the less circumscribed forms of cerebral

* A similar affection occurs in the retina during childhood, and perhaps, also in the suprarenal capsule.

tumour, are apt to set up in the adjacent brain-tissue morbid changes that can only be regarded as inflammatory, and as due to irritation caused by their presence. Such an affection sometimes assumes the form of "red softening," of which I shall give an example further on. More often, it is what Rokitansky first described as "yellow softening,"—a state in which the medullary substance has a faint yellow tinge and looks glistening like blanc-mange, but nevertheless retains its form when sliced or cut, and is not in fact softer than the rest of the brain. This appears to be œdema; the microscope throws no light upon its nature. Another common result of the presence of a new growth in the brain is the accumulation of an increased amount of fluid in the ventricles, which may become greatly enlarged. It might sometimes be attributed directly to compression of the veins of Galen or of the choroid veins; as when the tumour is seated in the cerebellum or in one of the posterior lobes of the cerebrum. But Virchow has pointed out that hydrocephalus often occurs where this explanation is inapplicable; and his statement is confirmed by several cases in our records.

Statistics of cerebral tumours.—To judge from the cases that have occurred at Guy's Hospital there is a remarkable difference in the liability of the several parts of the brain to be the seat of tumours at different ages. We have had twenty-two cases in which there was a primary new growth in one of the *hemispheres*, sixteen on the right side, and six on the left. In only five of these was the patient less than thirty years old; the rest were pretty evenly distributed between the ages of thirty and sixty. On the other hand, out of fourteen cases in which a tumour was seated at the *base* of the brain, ten occurred in persons under the age of thirty. The six instances of cysts in the *cerebellum* were all in patients between twenty-one and twenty-seven years old. There were only two cases in which solid tumours were found in children under the age of ten; in each instance the seat of the affection was the cerebellum.

Tumour of the brain is more frequent in males than in females. I find that among forty-two cases which have occurred at Guy's Hospital, and in which the sex is recorded, the proportion is as 27 : 15. This corresponds very closely with the ratio of 10 : 6 given by Obernier. But it is interesting that in the cases in which one of the hemispheres was the seat of the affection the preponderance of males is much higher, namely as 16 : 6. For men are much more liable than women to blows and falls; and Virchow has expressed the opinion that new growths affecting the upper parts of the brain are often caused by injuries to the head. He has further stated that tumours in this position are generally gliomata, whereas at the base sarcomata and carcinomata are more often found.

In the forty-third volume of the 'Guy's Hospital Reports,' Dr Hale White has collected 100 cases of cerebral tumour which were found in the dead-house from 1872 to 1884 inclusive. The new growth was tubercular in nearly half of these cases (45), and it was a glioma in 24. Among the remaining cases, 2 were glio-sarcoma, 10 sarcoma, 5 carcinoma, 5 gumma, 1 myxoma, and 1 lymphoma; while 4 were cystic.

Most of the cases occurred in male subjects; of the tubercular cases, 34 to 11 in female patients, and more than half of these 45 patients were children under ten years old.

The hemispheres were the seat of a tubercular tumour in 22 cases, the cerebellum nearly as often (20), the pons in 6 cases, the bulb and the crus cerebri in one each. Nineteen of the tubercular tumours were multiple.

Glioma was widely distributed among patients of different ages, from three years to sixty-two. Here also the male sex predominated in the proportion of 19 to 5, and all the patients under twelve were boys. The seat was the hemispheres in 13 cases, the cerebellum in 4, the pons in 2 (one a child), the bulb in 1, the pituitarium in 3, and the dura mater in one only.

Of the four cysts, two were situated in the cerebellum. One of the cerebral cysts was a hydatid.

Considering locality only, 48 tumours of all kinds were in the hemispheres, 28 in the cerebellum, 10 in the pons, 7 grew from the meninges, 5 were in the basal ganglia, and 3 in the pituitarium.

Symptoms.—In proceeding to discuss the clinical symptoms of these various forms of cerebral tumour, we must first remark that in making *post-mortem* examinations one occasionally finds a tubercle or a tumour in cases in which there had during life been no suspicion of the presence of any cerebral lesion. Thus we have in the museum of Guy's Hospital a specimen of a very hard growth, nearly as large as a pigeon's egg, attached by a pedicle to the ridge of the petrous bone; it was discovered accidentally in the body of an aged pauper woman who was said to have had no cerebral symptoms. Another preparation is one of a large carcinomatous mass in the cerebellum, taken by Dr Moxon from a patient who died of cancer of the breast and of the liver. But it may be said that, if she had lived a little longer, the tumour might have manifested signs of its presence; and the same remark applies to the very few instances in which one or more small tubercles have been unexpectedly found in the brain in persons who have fallen victims to phthisis or to some other scrofulous disease. All that is really proved by such cases is that lesions of various kinds may be latent during the early periods of their development. But of this fact there is abundant evidence of another kind. Thus when tubercular meningitis has been the cause of death, and when the patient had appeared to be perfectly well up to the time of the commencement of that disease, one not uncommonly finds one or more yellow tubercles in the brain-substance which must obviously have been of older date. Again, it sometimes happens that a person dies of what appears to be an acute illness, and that the only lesion found at the autopsy is a tumour, with softening of the surrounding cerebral tissue. Some years ago a man was admitted into hospital insensible, livid, and in a high state of fever, so that the diagnosis was typhus; he lived only three days from the time when he was first taken ill, but at the autopsy a small spindle-cell growth, of the size of a bean, was found projecting into the fourth ventricle from the side of the aqueduct. But, with the exception of one or two instances in which psammomata or other meningeal tumours have been quite unexpectedly discovered, I do not find in our records a single instance in which a tubercle or a tumour has been met with in the brain of a patient killed by accident, or who died from some indifferent disease, such as heart disease, or ileus.

The symptoms may be divided into two groups. Some of them are common to the large majority of cases; others are comparatively seldom observed, their presence being dependent upon the seat of the lesion. It will be convenient to describe the former group of symptoms first; and they may be arranged under three heads :—(1) cerebral symptoms proper, such as headache, giddiness, epileptiform seizures, loss of memory, mania, stupor; (2) dis-

turbances of distant parts, including vomiting, constipation, &c.; (3) certain changes in the optic disc, which are revealed by the ophthalmoscope.

1. *Head-symptoms.*—*Pain* is very rarely absent, and is generally the earliest indication that anything is wrong with the patient. Its seat sometimes, but not always, answers to that of the lesion. The superficial nerves of the corresponding part of the scalp may be tender to pressure or percussion; or (as Romberg first noticed) the act of holding the breath or of coughing may increase the pain—no doubt by producing venous congestion. The museum of Guy's Hospital contains a large tumour, three inches in diameter, found many years ago in the left hemisphere of a girl, a patient of Dr Wilks, who had suffered severely from pain in the head, and who had declared that when she turned her head to one side she felt something move in its interior. Another preparation consists of a small growth from the dura mater, taken by Mr Day, of St Neots, from an old woman who died of bronchitis. She had often expressed a wish that her head should be opened, because for years she had experienced anomalous pains in it, and a sense of coldness in one spot, not larger than a shilling; this corresponded very nearly with the seat of the tumour that was found after her death. The pain caused by a new growth may be of every degree of intensity, from a dull headache to the most unbearable agony, such as is said to render a patient delirious. It is sometimes constant, but it generally undergoes exacerbation from time to time. In some cases, indeed, it is intermittent or paroxysmal, so that it may closely resemble ordinary migraine. Thus Abercrombie relates the case of a boy, aged six, who began to suffer from fits of severe sick-headache, recurring at first about once a fortnight, and leaving him in good health in the intervals. After five or six months the attacks assumed a different and more persistent character; and two months later he died, when a tuberculous mass was found in the cerebellum. A similar instance has been recorded by Lebert. These cases seem to have an important bearing upon the general theory of the production of "cerebral symptoms" by local affections of the brain. I shall hereafter have to describe a group of functional disorders of the nervous centres, including migraine, vertigo, epilepsy, and some others,—under the name of "nerve-storms" or "explosive neuroses." And I shall endeavour to show that each of these is liable to be set up by a variety of exciting causes. Now, my hypothesis is that a tumour or a tubercle causes a transitory vertigo, or an epileptiform fit, or an attack of sick-headache in exactly the same way as any other disturbing agent. I conceive that the nerve-storm so produced has precisely the same seat as when it is merely the result of over-fatigue, or irritation of the generative organs, or disorder of the stomach. And I think it is probable that frontal headache, even when unattended with the other characteristic symptoms of an attack of migraine, is yet very often of that nature. If this be granted, it ought to follow that pain in the forehead should point less directly to the anterior part of the brain as the seat of a tumour than occipital pain to the cerebellum or posterior lobes.

It is not known whether growths situated in any particular region are more apt than others to be accompanied by *vertigo*, or by general epileptiform *convulsions*, as distinguished from those seizures, to be presently described, which are limited to the muscles of certain parts. But Dr Reynolds was led by the examination of a large number of cases to the conclusion that convulsions in general were less common when the disease affected the anterior lobes than when it occupied the posterior lobes or the cerebellum.

Irritability of temper, depression of spirits, loss of memory, a general impairment of intelligence may be observed even when a tumour lies in the membranes at the base of the brain. Thus we have in Guy's museum a specimen of a cholesteatoma, of the size of a pigeon's egg, which was situated so as to compress the under surface of the pons and of the cerebellum, but which had also insinuated itself into the Sylvian fissure to some extent; the patient was at one time in the hospital with paralytic symptoms, but he became maniacal, had delusions, and was unmanageable, so that he was removed to Colney Hatch Asylum, where he died. On the other hand, the mental faculties are not infrequently retained nearly to the last, when one of the hemispheres is the seat of the disease.

Friedreich and Obernier speak of excessive sleepiness as a principal symptom in two cases, which severally came under their observation. The most usual termination of all the affections described in the present chapter is by stupor, which gradually passes into coma. Sometimes the patient lies for weeks, without taking the slightest notice, passing all his evacuations under him, and showing no sign of intelligence, except that he slowly swallows the food that is placed into his mouth. Some years ago Sir William Gull had under his care a boy who manifested a remarkable retardation of intelligence. If a question was put to him he seemed not to heed it; but after many seconds, when the questioner had passed on to talk to someone else, he would deliver, word by word, a reply which showed that he perfectly understood what had been said. In other cases the mental condition undergoes changes which one feels disposed to refer to varying degrees of pressure by ventricular effusion. After lying in a stupor for two or three weeks, the patient may recover his senses, remain conscious for several days, and then relapse into his former state.

2. *Nutrition.*—The bowels are usually constipated. *Vomiting* is a frequent and most important symptom: commonest perhaps with cerebellar tumours. As a rule, there is loss of flesh, and extreme emaciation sometimes occurs. But a boy of fourteen, who was in Guy's Hospital in 1867, became remarkably fat during his illness, and remained so until he died; and two or three years previously, the body of a young woman in a similar condition was brought down into the *post-mortem* room. In each case there was a tumour at the base, growing upwards into the third ventricle.

The *temperature* of the body is often one or two degrees below normal; but before death fever may develop itself. Obernier speaks of a rapidly advancing case in which every exacerbation of the headache was associated with a marked fall in the rate of the *pulse*. The mode of death is often by cessation of *breathing*, the heart continuing to beat for some little time; in one instance, of which I have notes, it went on for thirty-five minutes, while artificial respiration was vigorously kept up. In such cases it is wonderful how quietly life departs, without a gasp or a moan, or the movement of a limb. Some years ago I was called to see a female patient, whose symptoms pointed to the presence of a cerebral tumour, with Dr. Blades, of Kennington. On my arrival at the house, he came out of her room to speak to me, leaving her husband and two women with her. When we went to her bedside a few minutes later we found her dead, except that a slight flickering of the pulse was still perceptible. Not the slightest change had been observed.

3. *Changes in the optic disc and retina.*—In 1860 von Graefe communicated to the German 'Archiv für Ophthalmologie' a short paper, in which

he pointed out that inflammation of the optic nerves within the eyes sometimes occurred as a complication of cerebral diseases. It was already known that blindness was a frequent effect of tumours of the brain, even when their seat was not such as to involve any of those parts which were supposed to be functionally related to the sense of vision, but the belief had been that paralysis of the nerves of sight was the cause of the amaurosis, and that if any anatomical change was to be found it would be a simple atrophy from disease. Thus, von Graefe confesses the surprise that he felt when he first discovered (about the year 1857) that the optic discs were swollen and reddened in a case of this kind. His observations were soon confirmed by others, and after a while the remarkable fact was elicited that precisely similar appearances could often be detected when the patient was not conscious of any visual defect. Thus the ophthalmoscope has acquired great value in the diagnosis of diseases of the brain.

It was suggested by von Graefe himself, in his earliest communication, that there were two separate ways in which changes in the discs could arise, and that these were characterised by different appearances. On the one hand, an increase of intracranial pressure, acting upon the cavernous sinuses, might mechanically obstruct the return of blood through the retinal veins; on the other hand, an inflammatory process at the base of the brain might be propagated, as a "neuritis descendens" along the optic nerves to their terminations within the eyeball. Subsequent writers have to some extent modified the interpretation of these two conditions, and it has been shown that they very often co-exist. The one is known to the Germans as the "Stauungs-papille;" following Dr Allbutt, English writers term it the "Choked Disc," or (with less propriety) "Ischæmia of the Disc." The other is called "Optic Neuritis," "Papillitis," or "Neuro-retinitis descendens." There is also a third change, "Atrophy of the Disc," which may either arise independently or be consecutive to neuritis.

The choked disc.—There is no better description of this affection than that which von Graefe gave in his account of the first case that came under his observation. "The papilla," he says, "was greatly and irregularly swollen, rising steeply on one side and falling gradually on the opposite side to the level of the retina. . . . . Instead of being transparent its tissue looked grey and opaque, with an extremely deep reddish tint, and the adjacent part of the retina had the same appearance, so that the choroidal margin was completely hidden. The opacity was diffused, except that with the direct method of examination one could perceive a striated appearance following the course of the fibres of the optic nerve. The retinal veins were dilated, exceedingly tortuous, and obscured here and there by dipping into the opaque tissue; the arteries were comparatively small. The turbidity of the retina diminished gradually from the disc outwards over a zone of rather more than 2 mm. in breadth, so that it occupied an area 5 mm. in diameter (including the disc itself)." In a second case "the disc was still more red, and there were ecchymoses in the adjacent part of the retina."

The prominence formed by the optic disc in a case of this kind appears to be easily recognised after death when the eye is removed and laid open; an admirable illustration of it is given by Dr Allbutt. In one instance in which an intercurrent attack of smallpox destroyed life at an early stage of the ocular affection, Cornil found that the microscopical appearances consisted in an infiltration of the connective tissue with serum, and in a few

hæmorrhages into the adjacent part of the retina ('Arch. Gén.,' 1868, ii. p. 679). The absence of grave morbid changes is proved by a case of Mr Lawson's, in which the presence of a hydatid cyst within the orbit caused an extreme state of "choked disc;" four days after puncture of the tumour the engorgement was found to have almost entirely disappeared. A drawing in 'Pagenstecher's Atlas' (Pl. xxxi, fig. 7) shows the nerve-fibres bulging outwards, so as to separate from the choroid the peripheral layers of the retina, throwing them forwards and outwards. In some more advanced cases of von Graefe's, examined by Schweigger and Virchow, the connective-tissue elements, the vessels, and the nerve-fibres were all found swollen, and the latter were beginning to degenerate, while in the outer coat of the vessels there was an overgrowth of nuclei.

The choked disc was attributed by von Graefe to compression of the cavernous sinus, aided by what he termed the constricting action of the sclerotic ring. He argued that if from any cause the flow of blood along the retinal veins was obstructed, the unyielding structure would give rise to a sort of *strangulation* of them at the point where they pass through it, and so he explained the fact that the congestion was limited to the intra-ocular termination of the optic nerve; but (as Dr Hermann Schmidt has urged in vol. xv of the 'Arch. f. Ophth.') the ophthalmic vein communicates so freely with the facial vein that it is difficult to see how pressure upon the cavernous sinus could appreciably interfere with the escape of blood from the eyeball, nor does it appear probable that the wall of the sinus would yield to any moderate force. These considerations are confirmed by a case under my observation, in which the sinus on one side was completely obstructed by softening thrombus; the corresponding optic disc had been noticed during life to be perfectly normal. In all probability I think Schmidt's explanation is the correct one. He finds experimentally that an injection of Prussian blue into the arachnoid cavity (subdural space of the brain) makes its way through the optic foramen into the space between the two sheaths of the optic nerve, and fills a fine network of lymphatic channels within the lamina cribrosa (formed by the sclerotic ring) between the very fibres of the nerves themselves. He therefore suggests that some of the fluid which is always present in small quantity in the arachnoid cavity is driven into the optic nerve in a similar manner whenever the intracranial pressure is from any cause increased. And, as a matter of fact, I can from my own observations confirm the statement that a watery liquid is found distending the sheath and giving it a bulbous appearance, and that this liquid readily escapes as soon as a puncture is made. One difficulty is that on Schmidt's theory I do not quite see why cerebral hæmorrhage should not cause choked discs. Is it because the great force suddenly exerted flattens the sheath of the nerve, and closes the channel through it? A hypothesis of Benedikt's, that the changes in the fundus of the eye are to be explained by referring them to sympathetic vaso-motor disturbance, seems at present too vague to be met by argument.

Neuro-retinitis.—The distinctive features of this affection, as compared with choking of the disc, are that its swelling and redness are less marked, but that it looks more opaque, and that the morbid process extends further into the retina itself, and (according to von Graefe) involves its middle and outer layers, as well as its inner layer. Dr Allbutt endeavours to describe the appearance of the disc by saying that there is not "a circumscribed intense redness, or brownish grey, but rather a wash of reddish lilac or a grey tint."

And he adds that one does not perceive "a multitude of minute branches and capillaries," such as give a "mossy" look to the choked disc, but that the vessels which become distended and tortuous are rather the main trunks. Moreover, these are often completely concealed in part of their course by the opaque tissue, beneath which they dip.

One of the chief microscopical appearances seems to be that the optic nerve-fibres within the disc are enlarged and beaded, presenting a series of fusiform swellings. The connective-tissue bundles are said by Virchow to present no excess of cells or nuclei, but the coats of the vessels are thickened. The trunk of the nerve in its whole length shows an accumulation of cells and nuclei within its sheath, and also between the fasciculi of which it is made up.

I have already remarked that von Graefe ascribed neuro-retinitis to the direct extension of inflammation downwards; as, for instance, from a basal meningitis. We shall find that this explanation is not always applicable. The probability is that the affection is then secondary to a choked disc, being developed in the same way as those chronic affections which are well known to occur in the lungs and liver and kidneys when there is obstructive heart disease. It is not surprising that (as Mr Hulke has remarked) mixed forms of ophthalmoscopic changes are more commonly seen than typical specimens of a "Stauungs-papille" or of a descending inflammation of the optic nerve.

Dr Gowers holds that congestion of the disc is not the result of intra-cranial pressure, that it is always inflammatory or irritative in origin, and that the distinction between the choked disc and optic neuritis is one of degree only.

Atrophy of the disc.—Neither congestion of the disc nor neuro-retinitis is a permanent affection. They are not even stationary; for the process, if the patient lives long enough, must subside or quickly end in atrophy. The latter is by far the most common termination. The disc becomes less and less swollen, and at length is quite flat, or even sinks below the level of the surrounding retina. Its red and grey tints grow into a dirty white colour. The tortuous veins diminish in size; spots of hæmorrhage fade and are absorbed. The outline of the disc remains for some time blurred and irregular, its margins are ragged, and streaks of exudation are to be seen in the course of the retinal vessels. I have taken this description of "consecutive atrophy" from Dr Allbutt, but it accords with those given by all other writers. He goes on, however, to say that these appearances also are transitory. Little by little the disc clears up; its edge becomes sharply defined; its surface dead white and glistening. Its condition is then undistinguishable from one which has been preceded by no swelling or inflammation of the disc—the "simple white atrophy" of writers. Pagenstecher, indeed, stated that in microscopical sections of the dead tissues the origin of the affection may be recognised by the permanent displacement outwards of the two external layers of the retina from their normal commencement close to the edge of the choroidal ring. As to the histological changes in the disc itself, he shows that all the nervous elements may disappear, and be replaced by coarse connective tissue.

It is surprising how little impairment of vision attends these changes in the optic discs in many cases. Pain and over-sensitiveness to light are constantly absent. If there is any affection of sight, it is that the patient either cannot see at all, or sees objects more or less indistinctly, as if

through a mist. Dr Allbutt speaks of several patients with choked discs who could read a badly-printed news sheet with ease. One should therefore make it a rule to examine with the ophthalmoscope all cases in which symptoms of cerebral disorder are present; unless, indeed, an exception may be made for some of the simple uncomplicated neuroses. On the other hand, if a patient is found to present morbid appearances in the fundus of the eye, one should not be contented with his statement that his sight is perfect. The extent of the visual field in all directions should be accurately explored; and perhaps it may turn out that although vision is good at the centre it is yet very defective at the periphery or towards one side. When blindness sets in, it sometimes seems to come on almost suddenly, although the changes in the discs were no doubt developed very gradually. This occurred, for instance, in a patient under my care in 1878; about three weeks before her admission she went to sleep one afternoon, and on waking up found that she was totally blind. If perception of light is completely lost, I believe that the irides are always widely dilated and motionless. But in those cases in which the impairment of sight is partial, the pupils often appear to be of normal size, and their movements are sometimes not even sluggish. The suggestion of M. Jaccoud, that the excitation of reflex contraction of the iris by light is a proof that blindness is due not to any affection of the optic nerve within the eye itself, but to a lesion situated above the corpora geniculata, seems to be altogether fallacious.

When a choked disc, or one affected with neuritis, passes into a state of atrophy, the patient's sight often becomes progressively worse; but sometimes, on the other hand, it shows a marked improvement. Thus a patient of Dr Goodhart's, to whose case I shall presently refer, and who at one time could see absolutely nothing with her right eye, was ultimately able to read Snellen's $2\frac{1}{2}$, though with difficulty, at a distance of about a foot. When the power of vision has been but slightly impaired during the early stage of the disease, it is sometimes perfectly regained; but even in such cases, if actual neuritis has been present, the disc seems invariably to become more or less atrophied; it never resumes its normal appearance.

Localisation of organic cerebral lesions.—We now turn to other symptoms, which not only indicate in general the *presence* of some local lesion—whether a tubercle, gumma, or tumour—but also point more or less definitely towards its exact *seat;* they constitute what Dr Jackson terms "localising symptoms."

In the interpretation of these symptoms there is a preliminary difficulty, to which I must briefly allude. When at an autopsy one finds a tumour, or a tubercle, in some particular region of the brain, one cannot always tell whether the disturbances of function to which it gave rise were the results of *irritation* of the structures around it, or of *destruction* of those among which it grew. In the latter case the symptoms themselves would be those of the uncontrolled or unbalanced action of the corresponding parts on the opposite side of the brain, or of some other parts physiologically antagonistic to those containing the lesion. It is precisely this difficulty which makes it impossible to attach an exact meaning to inequality of the pupils; and several other instances will present themselves further on. Dr Jackson, indeed, has endeavoured to distinguish certain affections as "destroying," while he speaks of others as "discharging" lesions. And it is remarkable how great a size may be reached by a mass in the brain,

without its depriving the structures in which it is embedded of their function; while again a tumour sometimes compresses adjacent parts, so as to annihilate their activity. Without attempting to lay down a positive rule, it may be said that, in all the diseases now under consideration, the presumption is in favour of irritation, rather than of destruction, when either explanation is admissible.

The base.—Beginning at the base of the brain, and passing from before backwards, one meets with the simplest of all localising symptoms, namely, those which depend on interference with the various cranial nerves.

It is easy to see that complete *anosmia* points to the presence of disease near the ethmoid bone, or involving the inner and lower parts of the two anterior lobes; while loss of smell on one side may be due to a lesion implicating one olfactory bulb, or the root of the nerve extending outwards to the Sylvian fissure.

A growth pressing upon the optic chiasma produces various effects according to its exact situation. Thus there may be more or less complete loss of sight in one eye, or even total blindness; but as changes in the discs are almost certain to be present, it would be scarcely possible to draw any inference from this symptom. What is really significant is the limitation of a visual defect to half the field in each eye, or (as it is termed) *hemiopia.* Of this there are three different forms, but the interpretation of them is still somewhat doubtful, because opposite views are now held as regards the structure of the chiasma. On the theory of semi-decussation, which appears the more probable, they may be described as follows:

(1) Compression of the centre of the chiasma, cutting off the decussating inner fibres of each optic nerve, will cause loss of vision over the temporal field in each eye (double temporal hemiopia); (2) compression of one optic tract will deprive the patient of the temporal field in the corresponding eye, and of the nasal field in the opposite eye (homologous lateral hemiopia); (3) loss of vision in the nasal fields of the two eyes (double nasal hemiopia) can only be due to a double lesion, symmetrically placed at the lateral angles of the chiasma, so as to interfere with the non-decussating outer fibres on each side. Examples of all these forms of hemiopia are known to occur. Homologous lateral hemiopia is very common in attacks of migraine; of double temporal hemiopia two cases have been recorded (one by Sämisch, the other by E. Müller), in each of which a large tumour lay in the middle line, involving the centre of the chiasma; of "double nasal hemiopia," one by Knapp, in which the pressure at the necessary spots was effected by dilated and atheromatous branches of the circle of Willis.*

Intracranial growths sometimes produce *deafness* when they occupy, or extend into, the space between the internal auditory meatus and the side of the pons. Dr Hughlings Jackson has even met with one case in which there was loss of hearing from this cause without any facial paralysis. In another instance, a complete bilateral destruction of the auditory sense was

* I may note that on the theory of complete decussation "double temporal hemiopia" would be caused only by pressure on the *front* of the chiasma, while pressure on the *back* of the chiasma by a single growth would account for "double nasal hemiopia." So far, that theory might hold, although both these kinds of hemiopia are very rare, but it admits of no explanation of the occurrence of the far more common "homologous lateral hemiopia," excepting the untenable one of the presence of a lesion placed *on one side* of the chiasma, in such a situation as to compress a part of the optic nerve, and also a part of the optic tract on that side.

imperfectly explained by the autopsy, for the only growth at the base was one of the size of a hazel-nut on the *left* side. It might be suggested that this symptom ought sometimes to be referable to an affection of the brain itself; but Dr Jackson's experience appears to negative such an opinion.

With regard to affections of the other cranial nerves there is little to add to the remarks made when describing their symptoms (see pp. 401—417). An anatomist can sometimes infer the seat of a growth with very great accuracy from the implication of some nerves while others escape. Thus, in one instance, the fact that all the muscles of the eyeball were paralysed, while no part of the fifth nerve was interfered with except its ophthalmic branch, seemed to prove that the disease occupied the sphenoidal fissure. On the other hand, it is perhaps sometimes possible to draw the conclusion that no single lesion will explain every feature of the case, a conclusion which (as we shall presently see) may be of considerable diagnostic importance.

The cerebellum.—The localisation of a tumour or tubercle in the cerebellum is often attempted, and is not infrequently successful. But one must not forget that this part of the encephalon is a very common seat of such growths; so that the result of the diagnosis may possibly sometimes be due to good luck rather than to skill. Beside pain in the back of the head and neck, vomiting, convulsions, blindness, and vertigo—none of which symptoms are special to lesions of the cerebellum—there are some others which are believed to be more characteristic, and chief among these is a reeling, staggering gait, like that of a drunken person, but with a tendency to fall in some particular direction. It was long ago shown by the experiments of Flourens on pigeons that excision of the cerebellum rendered the bird unable to walk or to fly, or to perform regular determinate movements; and he suggested that the function of the organ was one of coordination. Subsequent investigations have proved that a large part of the process by which the actions of the individual muscles are harmonised and combined take place in the spinal cord. But this fact does not exclude the possibility that a higher coordination may be effected by the cerebellum. And a fair hypothesis has been suggested by Dr Broadbent, namely, that what it does is to bring muscular action into relation with visual impressions, as when movements have to be guided by sight; whereas in the spinal cord they are arranged for tactile and other cutaneous impressions. However, a patient suffering from disease of the cerebellum may still stagger, although, in consequence of his optic discs having undergone atrophy, he can no longer see anything.

When the staggering is accompanied by a tendency to fall in one particular direction, we might fairly expect that this circumstance should not only indicate the presence of disease in one side of the cerebellum, but also show which side is affected. But tumours of the cerebellum often attain a very great size, and annihilate a large part of the organ. Thus it is peculiarly difficult to avoid the difficulty referred to above; the same affection may produce diametrically opposite effects, according as its action is that of a "destroying" or of a "discharging" lesion. In 1877 I made an autopsy in a case of Dr Frederick Taylor's, in which a cheesy tubercle occupied the whole thickness of the cerebellum, rather to the right of its centre; and the right lateral lobe was universally pale and softened, and full of granule-masses. The patient was a boy, five years old. It so happened that the exact seat of the mischief had been indicated several

months before his death, by a bulging of the right side of the occipital bone, which was so much thinned at one spot that it would yield on pressure and rebound like a piece of tin or of cardboard. At the same time it was noticed that when he sat up in bed he had no balancing power; but not that he rolled over on one side rather than on the other. The eyes, however, were affected with a peculiar form of nystagmus, which might perhaps have been turned to account as a localising symptom. "It came on only when he lay on the left side; the eyes would then gradually fall over to the left, and be suddenly jerked back to the right; and these movements would be repeated rhythmically. When he sat up in bed there was a continuous rhythmical movement of the head from left to right." Afterwards, six weeks before he died, "he lay on his back, with his head turned to the left; his eyes were then directed to the left side, and jerked from time to time upwards." Now, Hitzig, following Purkinjé, has shown ('Reichert's Archiv,' 1871) that by passing a galvanic current through the head of a healthy person, from one mastoid process to the other, one can obtain oscillatory movements of the eyes exactly like those observed in Dr Taylor's patient; and in a subsequent paper he accounted for those effects by referring them to disturbances of the cerebellum. Dr Ferrier, too, found that galvanising the exposed cerebellum of monkeys and other animals caused the eyes to deviate in different ways, according as the poles were applied to different regions of its surface. Indeed, his experiments seem to indicate a very simple law of localisation, namely, that when any part of the cerebellum is the seat of irritation, the eyes become turned in the corresponding direction, whether to the right or to the left, upwards or downwards as the case may be. In many cases analogous movements of the head accompanied those of the eyes. Thus I suppose that a tendency to fall backwards, which has sometimes been noticed when there has been disease of the cerebellum, would point to the upper surface of the organ as its seat; for Dr Ferrier found that galvanising that part in monkeys led to upward movements of the eyes, and to throwing back of the head. This writer speaks of Hitzig's results, referred to above, as being essentially the same with those which he has himself obtained; but the correspondence does not seem to me to be so close.

Dr Taylor's patient had also left hemiplegia, which was no doubt due to compression of the right side of the pons by the tumour. I believe that whenever this form of paralysis occurs in such a case, it is always produced in that way, and that it is invariably "crossed," affecting the limbs on the opposite side to the cerebellar lesion.

But the functional relations of each half of the cerebellum are nevertheless chiefly with the limbs of the same side of the body, through the connections of the fibres of the middle peduncle with nuclei in the opposite half of the mesencephalon. This is proved, not only by anatomical and physiological investigations, but also (most convincingly) by the pathological fact that when there is congenital unilateral atrophy, affecting one hemisphere of the brain, one crus cerebri, and one side of the deep part of the pons, the wasting is found in the other side of the cerebellum, as it is in the other side of the spinal cord.

On the whole, it may be said that when there are the general symptoms of an intracranial tumour (including tubercle, gumma, cyst, and abscess), namely, headache, vomiting, and optic neuritis, especially with optic atrophy and amaurosis, and when there is no hemiplegia, but only general muscular

weakness, when there is spasm of the muscles at the nape of the neck, and decided vertigo and loss of power to maintain equilibrium—the diagnosis of the cerebellum as the seat of the lesion is more than probable.

This last symptom, disturbance of equilibrium, is said by Nothnagel to depend on lesion of the superior vermiform process, and this conclusion is confirmed by other observers. Tumours of the lateral lobes of the cerebellum appear to have no special symptoms; but they may sometimes be more than guessed at, by the effects they produce through pressure on the pons or other adjacent structures.

The motor area.—Passing now to the regional diagnosis of lesions involving the motor tract, I may begin by observing that the greater part of the description of hemiplegia given in a former chapter applies also to the same symptom when it is produced by a new growth. But whereas we have seen that certain parts of the brain-substance are far more liable than others to suffer from the effects of embolism, or of rupture of a cerebral artery, the distribution of tumours is comparatively irregular and capricious. They may interfere with the fibres which pass down to the spinal cord at any point in their course, and may either destroy the whole or part of them. On the other hand, it is wonderful how large a tumour may be found in the very substance of the motor tract, without completely abolishing its functions. In 1869, a boy, aged four years and a half, died in hospital under my care, after an illness of fourteen months' duration. Three months before his death he could sit up in bed, although he rolled about and his balance was easily upset. A month later he "could move his legs a very little." Dr Moxon found that the pons was occupied by an immense caseous tubercular mass, which consisted of two halves, fused together, and preserving almost exactly the normal shape of the part; only a thin shell of nervous matter remained. It seemed marvellous that life could have been maintained while the mass was growing to such a size. And a few years ago I made an autopsy in which a tumour (certainly a very minute one) was found in one of the corpora dentata of the bulb, there having been no symptoms of disease of that part.

Then, again, paralysis of the limbs on one side of the body, or even of all four limbs, may be due to a mass pressing on the motor tract from without; for instance, to a sarcoma connected with the base of the skull, and compressing the medulla oblongata or the pons; or to a glioma in the hemisphere above the lateral ventricle, pushing downwards and flattening the basal ganglia. And, in other cases in which the growth itself lies altogether outside the region, lesions of which are ordinarily attended with hemiplegia, this symptom is caused by the yellow softening that so often develops itself secondarily.

But a growth situated in the superficial convolutions of one *hemisphere* may affect the movements of the opposite side of the body, without either disturbing the corpus striatum mechanically by pressure, or involving it in any morbid action. The symptoms so produced are among the most interesting that come under the observation of physicians, and within the last few years they have attracted much attention from physiologists. In the main, their character is generally convulsive; but the spasms are often followed by a transitory paralysis; and it is still a question whether a loss of power may not occur primarily and alone. Bright was the first to point out, in the earliest volume of the 'Guy's Hospital Reports' (1836), that some cases in which fits were due to a local lesion presented the peculiarity that

consciousness was not lost. But, so far as I know, this form of convulsions had never been systematically investigated, until Dr. Hughlings Jackson took up its study, in the hope that it would throw important light upon the nature and seat of epilepsy in general. He insisted on the fact that *hemispasm*—the "mobile counterpart" of hemiplegia—must indicate a condition of "instability" in the convolutions which discharge through the corpus striatum, and must therefore be a symptom of disease in those convolutions or (possibly) in the corpus striatum itself. And he set to work to determine, so far as pathological opportunities might offer themselves, the exact seat of the lesions found where this symptom had been present. The particular form of convulsions which he thus elucidated is now commonly known abroad as "Jacksonian Epilepsy." His theories have since received remarkable confirmation by direct experiment.

The first step taken was in 1870, when Fritsch and Hitzig made known the fact that, instead of the surface of the brain being insensible to galvanic currents, there were in the dog certain parts of the convolutions which reacted to such currents in a very definite way, each setting in motion some particular limb, or even some special set of muscles. Thus they laid down the position of "cortical centres" for the movements of the neck, face, fore- and hind-leg respectively. Three years later Dr Ferrier repeated these experiments with the faradic current, employing various animals, and at length monkeys, in which the principal convolutions are comparable, one by one, with those of the brain of man. Afterwards Hitzig himself operated on a monkey. Between the results of these two observers there were some discrepancies in details. Hitzig used a comparatively weak galvanic current; thus he obtained movements over a far more limited area; indeed, his main object was to discover what parts of the cortex would yield movements limited to isolated groups of muscles, on the most feeble stimulation. He maintains that in the monkey the motor centres for all parts of the body are situated in the anterior central (ascending frontal) convolution. That for the leg is nearest the falx cerebri, at a distance of about three millimetres from it; that for the arm lies three millimetres further outwards; that for the upper part of the face, supplying the muscles of the ear and eye, is situated rather more behind and externally; that for the lips, tongue, and jaws, is six millimetres inwards from the fissure of Sylvius. Hitzig did not deny that irritation of adjacent convolutions by more powerful (especially by induced) currents gives rise to movements in distant parts; but he attributed them to the action of the current upon parts beneath the cortex.

Ferrier placed his centres in the posterior central (ascending parietal) as well as in the anterior central (ascending frontal) convolution; and also in the postero-parietal lobule, in the back part of the third frontal (speech), in the angular (vision)* and in the superior temporo-sphenoidal convolution (hearing). He agreed with Hitzig in placing the centre for the leg close to the median line, but he divided it into two; one in the postero-parietal lobule, for advance of the hind limb, as in walking; the other in the upper parts of the ascending parietal and ascending frontal convolutions, for climbing and other complex movements of arms and legs. He made the centres for the arm and hand occupy the ascending parietal convolution nearly as far outwards as the fissure of Sylvius, as well as the ascending frontal and the

* In the second edition of Dr Ferrier's work, 'The Functions of the Brain,' 1886, he includes the occipital lobes in the visual area (pp. 271, *et seq.*).

superior frontal convolutions outside and in front of the leg-centres. The movements of the lips, tongue, and mouth he connected with a series of centres occupying the lower part of the ascending frontal, and with one in the supra-marginal convolution. Centres for the movements of the eyes were believed to occupy an extensive area in the two upper ("first" and "second") frontal convolutions. Stimulation of the angular gyrus or of the superior temporo-sphenoidal convolution, also caused the eyeballs to move, with dilatation of the pupils; but he supposed these parts to be centres for the senses of sight and hearing, and the movements to be reflex.

Since the publication of Ferrier's book in 1876, Nothnagel, Munk, Goltz, and other experimenters in Germany, and Burdon-Sanderson, Yeo, Schäfer, and Horsley in England, have repeated and varied his experiments. The stimulation of definite cortical areas has been controlled by destroying corresponding parts of the hemispheres. At the International Medical Congress which met in London in 1881, Prof. Goltz brought a dog from Strasburg which had survived extensive destruction of successive portions of the cerebral cortex, and this living witness was confronted by a monkey which was brought forward by Dr Ferrier. After observation of the functions of both animals they were destroyed, and the brains were subsequently submitted to most exact and thorough anatomical examination by Dr Klein, Mr Langley, Mr Schäfer, and Dr Gowers. The account of the discussion in the 'Transactions' of the Congress (vol. i, pp. 218–240), and the detailed reports by Langley and by Schäfer, published in the 'Journal of Physiology' (vol. iv, pp. 248–326), furnish an admirable example of the difficulty of ascertaining and interpreting scientific facts, and of success in at least the former task.

The practical difficulties have been: in faradic stimulation, the danger of a current spreading, either too widely or too deeply; in destructive operations, the appearance of much wider and more complete ablation than proved to be the case; in pathological observations, the infrequency of locally circumscribed, single and uncomplicated lesions; and, generally, the fallacies resulting from vaso-motor disturbance, from inhibition of distant ganglionic centres, and from direct or indirect mechanical pressure.

Nevertheless, pathological cases completely observed, completely examined after death, and free from complications, have gradually accumulated. A new series of experiments on the monkey's brain has been carried out by Profs. Ferrier and Yeo, and an independent series, with no less skill and care, by Profs. Schäfer and Horsley.

The result, so far, has been to confirm the general conclusions of Hitzig and Ferrier—that there are excitable areas in the neighbourhood of the fissure of Rolando, which are connected with definite movements—to further correct and define the localisation of these areas as above given, to prove that other portions of the cerebral cortex are not thus excitable, and to make more doubtful than before the precise localisation of sensory areas. Disregarding, therefore, the anterior frontal, the posterior parietal, occipital, and temporo-sphenoidal regions of the cortex, we have a "motor area" around the fissure of Rolando, which includes the ascending frontal with the closely adjacent part of the third, second, and first frontal convolutions, and the ascending parietal, together with the inner aspect of the same, which forms part of the marginal gyrus, and the front of the superior parietal lobule (postero-parietal of Huxley and Turner). Irritation of these parts is capable of causing definitely distributed clonic spasms of the muscles on the

opposite side of the body—a "discharging" lesion; destruction of the same parts is capable of causing definitely distributed loss of power in the same muscles on the opposite side of the body—a "destroying" lesion.

The area which thus corresponds with certain movements of the *leg* appears to be highest up, occupying the borders of the fissure of Rolando close to the falx cerebri, and extending over the adjacent parts of the ascending frontal (or præcentral), the ascending parietal (or postcentral), and the inner surface of the same part of the hemisphere, which corresponds to the marginal convolution immediately behind the calloso-marginal fissure. The area which corresponds with movements of the *arm* is below and external to the last, and occupies the ascending frontal and parietal convolutions opposite the second frontal gyrus in front and the angular gyrus behind. The area which corresponds with movements of the *facial muscles* appears to be lower down in the same direction, in front and behind the lowest point to which the fissure of Rolando reaches. Lower still, at the angle between the ascending and horizontal branches of the Sylvian fissure, opposite the third frontal convolution, this region marches with the gyri operti, the insula, and Broca's aphasic region.

Lastly, high up and internal, is a motor area which lies in front of that for the lower extremity, occupying the back part of the first frontal convolution, which is believed to be associated with movements of the trunk on the opposite side.*

When we apply the knowledge thus obtained with a view to the diagnosis of circumscribed lesions of the cerebral cortex, we are met by certain clinical difficulties.

In the first place it is to be noted that the occurrence of convulsive attacks without unconsciousness beginning in, or limited to, some particular part of the body, is not of itself a proof that a recognisable local lesion exists in the brain at all. We shall find several instances of epilepsy in which attacks of the kind were almost certainly "functional." And in a case of Dr Hughlings Jackson's, that terminated fatally, the only morbid change discovered at the autopsy was atrophy of both hemispheres, although the fits which had occurred had begun in the right index finger and thumb, and had often been unattended with loss of consciousness ('Med. Times and Gaz.,' 1872).

Again, it is perhaps worthy of notice that, according to Dr Jackson, the seat of localised convulsive seizures, or of general seizures beginning with local spasms, is in the majority of cases some part of the hand, generally the thumb or the forefinger; that the next most frequent seat is the cheek or the tongue, and that it is much more rarely the great toe. For one would be led by these facts to attach comparatively little value to the circumstance that a lesion should now and then be found in some particular convolution when the hand had been the starting-point of the movements, unless cases in which they had begun in the face should constantly show morbid changes in a different part of the cortex.

Nevertheless, as above stated, the confirmation of the results of experi-

* Centres for the muscles of the eyeballs, for movements of the tongue and jaw, and for movements of the tail, have been ascertained with more or less exactitude in the monkey and the dog. The difficulty of comparing the results, even in the former animal, with its different mode of progression and of prehension to human physiology, is obvious, and is fully recognised by Ferrier. Disturbance of the ocular muscles in man appear to be generally connected with lesions of the motor nerves, of the bulb or of the cerebellum, than with those which affect the cerebral cortex.

ment by clinical cases has now (1887) become too numerous to be regarded as mere coincidences. Some of the earlier ones may be briefly cited. (1 and 2) Two cases of Jackson's (one recorded in the Med. Times and Gaz.' for 1872; the other quoted by Ferrier), in each of which the spasms always began in one thumb, and a lesion was found in the opposite first frontal convolution. (3) In an autopsy at Guy's Hospital we found in the back part of the first frontal gyrus a small glioma on the right side; there had been repeated fits without unconsciousness, starting mostly in the left foot, but occasionally in the left arm. (4) A patient of Griesinger's, who was attacked with transient spasms in the right leg, afterwards affecting the right arm, the face, and the tongue; a cysticercus lay close to the left side of the falx cerebri, in such a position that its anterior extremity coincided with a line drawn vertically upwards from the ear. (5) Recorded by Dr Dreschfeld, of Manchester ('Lancet,' 1877). The convulsive movements began with a sudden clenching of the left fist, flexion of the wrist, and pronation of the forearm, while the left angle of the mouth was at the same time strongly drawn downwards; a local syphilitic lesion—probably the residue of a gumma—was found in the adjacent parts of the ascending parietal and supra-marginal convolutions. (6) A case observed by Hitzig. The patient was a French soldier who was wounded on the right side of the head near Orleans on December 14th, 1870. On February 4th, 1871, he was attacked with clonic spasms affecting the left side of the mouth and nose, the eyelids, and afterwards the fingers on that side. After death there was found to be a local necrosis of the parietal bone, and an abscess in the right ascending frontal convolution at the level of the sulcus between the second and third frontal convolutions. (7) Another instance, very similar, has been recorded by Wernher ('Virchow's Archiv,' lvi, p. 289). The patient, who had fallen from a railway truck, was attacked with convulsions limited to certain muscles on the right side, especially those of the angle of the mouth, the ala nasi, the eyelids, and the tongue, but also spreading to the fingers and to the neck; after death the left temporal bone was found fractured, and a small part of the surface of the brain crushed, including parts of the convolutions on each side of the Sylvian fissure in a line with the lower end of the fissure of Rolando. (8) A case of Dr Gowers ('Brit. Med. Journ.,' 1874) in which spasms began in the left angle of the mouth, involving afterwards the frontal muscle on each side, but in which the only local lesion appeared to be a clot of blood situated above the right lateral ventricle, just inside the gyrus fornicatus. This case certainly does not correspond with the observations of Hitzig and Ferrier; but it may justly be said that no instance hitherto recorded stands so directly in antagonism with these observations as there would be, for example, if a small tumour were found in one of the posterior lobes, if spasms limited to some one part of the body had been present during life.* (9) A case reported by Ferrier ('Brain,'

* There is, however, a case of Sir William Gull's in which a large abscess in one posterior lobe was attended with attacks of spasm limited to one arm; and a few other cases have been recorded which would appear inconsistent with the experimental results given above, were it not that legitimate criticism seems to deprive them of decided significance. One of these was observed by Dr Jackson. The foot had been the starting-point of the spasms, and a tumour was found which involved the lower part of the ascending frontal convolution; but the brain also presented other lesions. Another is a case of Dr Gowers', in which there was thrombosis of the superior longitudinal sinus and of some of its afferent veins, with hyperæmia of parts of the three frontal convolutions, situated further forwards than any recognised "cortical motor centres;" but in that

April, 1880) of convulsions in the left arm and leg following hemiplegia, in a man the subject of phthisis. At the autopsy a tubercular growth was found occupying both sides of the fissure of Rolando on the external aspect of the right hemisphere, and extending into the internal aspect as well. Other cases have been recorded by Jackson, Grasset, Gowers and Bastian, and Hadden.

Some of these go far to answer a further question, which is of the highest theoretical interest, whether affections of particular convolutions are capable of causing limited paralysis instead of spasms in the human subject. Dr Hughlings Jackson long ago pointed out that unilateral convulsive seizures were often followed by a more or less complete hemiplegia; but he maintained that this form of paralysis was always transitory, passing off in a few days or weeks, and he thought that it was dependent, not on the organic disease, but on "overwork" of the nerve-fibres passing to the muscles which were convulsed. And it is no doubt true that a persistent local paralysis is seldom or never caused by a cerebral tumour, or by any similar affection, unless it interferes directly with the corpus striatum, or with some part of the motor tract. But the very distinction on which Dr Jackson has laid so much stress—between "discharging" and "destroying" lesions—would tend to show that although paralysis may not be produced by a new growth situated in a convolution, it may yet result from softening of the very same part. When discussing the effects of arrest of blood supply to different regions of the brain (p. 572), we reserved this question; and almost every instance of aphasia is a case in point. Hence the most careful scrutiny of the convolutions near the fissure of Rolando should in future be made whenever a patient in whom a local patch of softening is likely to occur has had unilateral ptosis, or paralysis confined to part of the face, or to a finger, or any part of the upper limb on one side. The results of Ferrier's and subsequent experiments in this direction are most striking, and clinical observations to the same effect are recorded; two of Löffler's were quoted by Hitzig. One is the case of a man whose two parietal bones were both fractured at the vertex by a gunshot wound; he had paralysis of both legs. Another is that of a man who was wounded at the upper and anterior angle of the left parietal bone; his right leg instantly became paralysed, and he fell to the ground. On the seventh day the loss of power extended to the right arm also, but this quickly got better, while in the leg the recovery was very slow. Theoretically, a paralysis caused by a cortical lesion ought sometimes to be characterised by its being limited to some special movements of the affected muscles. An instance of this is afforded by Hitzig's case of the French soldier referred to above (No. 6). His spasms were followed by a partial paralysis of the lower part of the face on the left side, and it is expressly stated that he could voluntarily bring the muscles into action as well as on the opposite side, although that half of the face remained almost motionless when it should have moved in common with

instance the fits were general, although they began with a slow movement of the hand to the head; and death occurred within two hours from the time of their commencement. Again, in a patient of Dr Buzzard's, a girl of 18, a tumour of the size of a walnut was found in the white substance of the *left* hemisphere, extending as far as the grey matter of the gyrus fornicatus; the cause of death was phthisis. The symptom which had suggested the idea that a local lesion might be found was that she had fits beginning with an aura in the *left* wrist; but this is quite a different thing from the occurrence of spasm in the same part. Moreover, the application of a blister to the seat of the aura sufficed to transfer it to the opposite arm.

the right side for the purpose of expression. This is exactly the opposite of what occurs when disease of the corpus striatum causes paralysis of the face (cf. p. 568). In most of the cases reported by Ferrier, Jackson, Bourneville, and other writers, quoted by Bastian, there were first spasms of the thumb, arm, foot, or leg, followed by paralysis, or circumscribed convulsions supervened in a paralysed limb.

Lastly, instances have been observed in which congenital absence of one hand was found associated with atrophy of the opposite ascending parietal gyrus, by Gowers; or a wasted arm with the same condition in the brain, by Bastian; or an amputated limb with similar diminution in size of its supposed motor area in the cortex.

The cerebrum outside the motor tract.—We have still to consider what "localising symptoms" may be present when a lesion is seated in the substance of one hemisphere; or in the superficial convolutions, beyond the regions in which motor centres are placed by Hitzig, or by Ferrier. As we have seen, the cortical centres for the special senses are still imperfectly known, and tactile sensibility is universally less affected than movement. Hence it is perhaps not surprising that no special localising symptoms can as yet be connected with cortical lesions of the front part of the frontal lobes, the temporo-sphenoidal or the occipital. In many cases the only symptoms are lethargy, listlessness, an oddness of manner, taciturnity, and unwillingness to speak, or even loss of memory and impairment of intelligence—the very symptoms that we are accustomed to associate with the existence of diffused morbid changes affecting the whole of both hemispheres more or less uniformly. These, it is interesting to remark, are more or less precisely the symptoms which are found by experiment, in monkeys and in dogs, to result from ablation of the cortex of these parts outside the motor area.

The general symptoms of headache, vomiting, and optic neuritis, may point to the presence of a tumour, but unless it happens to press on some nerve-trunk we have no means of determining its seat.

It is well to remember that cerebral hæmorrhage, the commonest of "destroying" lesions, is, as we found (*supra*, p. 562), rare in these "non-motor" regions compared with the corpus striatum and mid-region of the brain, the pons, or the cerebellum. But tumours and cerebral abscesses are much less rare, so that we have sufficient evidence of their negative effects.

The basal ganglia.—We have seen that ordinary hemiplegia does not, as was formerly supposed, depend upon lesion of either grey nuclei of the corpus striatum, at least not directly, but upon interruption of the motor tract as it passes between the caudate nucleus and thalamus (the internal capsule behind the genu), or upon destruction of the white fibres of the external capsule or corona radiata outside the lenticulus as they run from the motor area of the cortex to the internal capsule. Destructive lesions of the lenticulus or of the cauda may produce no special symptoms.

The thalamus is more directly in the motor tract, but it is rarely the seat of disease, and the symptoms then produced are not characteristic. In the case of a boy in Guy's Hospital, which was published in the 'Pathological Transactions' for 1884, a tumour occupied the right thalamus. There was headache and double optic neuritis, with ptosis and slight motor paralysis of the opposite side affecting the arm more than the leg; no marked anæsthesia, if any, and no blindness or strabismus.

Lesions of the back of the thalamus have caused hemiopia, but there was probably pressure exerted on the optic tracts or the corpora quadrigemina.

The *pituitary body* is not infrequently the seat of a cancer, glioma, or other tumour. Three are noted in Dr White's list: one of them, a man of forty-five, with symptoms of tumour of the base, had a secondary growth in the lung. A fourth, a cystic tumour, is in the Guy's museum.

The *pinea* or conarium may contain a psammoma or sarcoma. Dr Heinrich Reinhold has collected four or five cases in a monograph ('Tumor der Zirbeldrüse,' 1886).

Diagnosis.—The diagnosis of the affections described in this chapter involves two distinct questions. First, they have to be distinguished from other diseases of the nervous centres or of distant parts; secondly, they have to be differentiated from one another. The answer to the former question is often wonderfully positive and exact. To the young student—who perhaps knows the difficulty of distinguishing between tumours and other surgical affections of parts that can be seen and handled—nothing in the art of medicine is more striking than the easy confidence with which the physician sometimes asserts the existence of a tumour in the cranial cavity.

Cases do, indeed, occur which are only cleared up in the *post-mortem* room. A man of whom little is known, or who has hitherto displayed no very marked symptoms of brain mischief, may die in a succession of fits, or in coma of a few hours' duration, and it may be difficult to make out that the cause is not cerebral hæmorrhage, or thrombosis of an artery, or uræmia. Or, if he lives for two or three weeks, the disease may appear to be meningitis.

But, as a rule, the illness caused by a tumour, or a tubercle, or a syphilitic gumma in the brain begins gradually, and goes on for several months or even for some years. In the cases that have occurred at Guy's Hospital within the last few years the duration of well-marked symptoms has generally been from three to nine months, but one patient had had fits for four years. Jackson has recorded the case of a woman who had optic neuritis and staggered in walking in 1865, and who did not die until the summer of 1872, when she had become insane—there was a growth springing from the "floor of the sphenoidal fossa." Now, I believe it may be said that whenever cerebral symptoms of the kind described in the present chapter have lasted for more than five or six weeks, the only other organic affections of the brain by which they can be caused are either abscess or diffused red softening.

With these two exceptions, the only cases in which it is really difficult to diagnose a local lesion, in the brain, are those in which it is doubtful whether there is anything more than functional disturbance. In women the symptoms are sometimes so vague that for a long time they are mistaken for those of hysteria. Some years ago a painter died in Guy's Hospital of what were believed to be the effects of lead-poisoning (drowsiness and epileptiform fits), when a spindle-cell sarcoma of the size of a marble was found in the right hemisphere, with extensive softening around it. When a patient's sole complaint is of having suffered for some weeks, or a few months, from a continuous or paroxysmal headache, unlike any pain to which he had previously been liable, it is often impossible to be sure whether organic disease is present.

The ophthalmoscope.—In such cases, and in many others, the ophthalmoscope is of the greatest service. The discovery of choked discs, or of

optic neuritis, or of atrophy, goes far towards establishing the presence of an organic lesion. At one time, indeed, the significance of these appearances was undoubtedly rated too highly; and I am not at all sure that this is not still the case. In the first place, it is to be observed that neuritis, or choking of the disc, in one eye only, is sometimes a sign that the cause lies in the orbit rather than within the skull. I have already referred to a case of Mr Lawson's, brought before the Clinical Society in 1876, in which a hydatid cyst pressed upon the nerve behind the eye, and so caused great swelling of the disc on that side. But, in other instances, a uni-ocular neuritis is due to pressure upon a single optic nerve between the chiasma and the opening through which it escapes from the cranial cavity; a gumma at the base seemed to have acted in this way in a case recorded by Mr Hulke in the 'Ophthalmic Hospital Reports.' Dr Hughlings Jackson has met with two cases in which the ophthalmoscope revealed an affection of one eye, dependent upon the presence of a tumour in the opposite cerebral hemisphere. But it is a rule to which there are very few exceptions that both optic discs suffer from disease of the brain.

In some rare instances ophthalmoscopic changes seem to precede all other symptoms. Thus Dr Allbutt speaks of a patient under his care, who had amaurosis from atrophy for three years before any signs of cerebral tumour began to manifest themselves, although these afterwards became well marked. But that writer himself frankly acknowledges that further experience has compelled him to modify the strong opinion which he at one time entertained that the existence of disease of the brain might be almost certainly inferred from morbid appearances in the optic discs. Indeed, among a series of unselected cases, recorded by Mr Hulke in the 'Ophthalmic Hospital Reports,' there are several in which optic neuritis, terminating in atrophy, seemed to be either spontaneous, or attributable to such vague causes as childbearing, lactation, leucorrhœa, sexual excesses, or to an antecedent attack of diphtheria or rheumatic fever. And in 1866 von Graefe spoke of it as sometimes due to menstrual disorders.

But even when unmistakeable indications of cerebral disturbance are present, it is a question how far one is justified in concluding from opthalmoscopic appearances that there must be a definite organic lesion in the brain. A remark of Mr Hulke's appears to me very striking, namely, that out of a considerable number of examples of optic neuritis, believed to be dependent upon intracranial affections, he had in 1868 only three times been able to verify his diagnosis by an autopsy. Now, there would be nothing wonderful in this if pathologists were often finding the residues of long past diseases in the brain, or even if it were known that life could be prolonged indefinitely when a local lesion was present. But the facts are exactly the reverse; except, indeed, that patches of old softening are met with, and the remains of hæmorrhages, such as do not cause any changes in the discs. Nor do the other symptoms of the cases of which Mr Hulke gives the details seem to me to support the view that they were dependent upon organic mischief in the brain. Three of them, all of which terminated in recovery, were thought to be probably examples of meningitis; but apart from the ophthalmoscopic appearances, I doubt whether this diagnosis could be justified. Another case would certainly have been regarded as one of ordinary epilepsy if the eyes had not been examined. Two patients, both advanced in years, had hemiplegia, and ultimately died of apoplexy; one, a man, aged thirty-eight, attributed his illness to watching a solar eclipse;

another a woman, aged twenty-four, ascribed hers to standing in the garden with her head uncovered.

Dr Hughlings Jackson, who believes that double optic neuritis is almost certain evidence of what he terms "coarse disease" within the cranium, admits that he has himself met with a few instances in which, on *post-mortem* examination, no such disease could be found. Of one case of this kind he has published full details. A woman, aged thirty-four, had for about a year been subject to attacks of severe headache accompanied with vomiting; for three months she had been blind. Her illness began by her being seized with vertigo and momentary unconsciousness, after which she had headache for four days. At another time the pain lasted for three weeks. She was admitted on December 19th, 1874. On January 6th, 1875, she had an attack of pain so intense as to make her toss her head from side to side, holding it in her hands, and crying, "Oh, my head, I don't know what I shall do." She retched and vomited frequently. Both optic discs were greatly swollen, and the veins in them were dilated and tortuous. After the 10th she sank gradually into what appeared to be natural sleep, which, however, passed into coma, and on the 12th she died by failure of the respiration. A tumour or some similar disease was confidently anticipated, but Dr Sutton, who made the autopsy, found only certain microscopical changes in the substance of the cortex. Dr Jackson speaks of this case as exceptional, but may one not fairly doubt whether it is peculiar in the absence of a visible cerebral lesion, so much as in its having proved fatal without the presence of such a lesion? Compare with it a case that was brought before the Clinical Society in 1876 by Dr Goodhart and Mr Higgens. A girl, aged twenty-one, was attacked with intense headache and vomiting on December 21st, 1874, at the very time when Dr Jackson's patient had just entered the London Hospital. Some months previously she had been stunned by a severe blow from a stone on her right temple. On the 24th there was well-marked double optic neuritis, her pulse was irregular and only 52 in the minute, and she had no fever. Afterwards she had paralysis of each sixth nerve, suffocative attacks in which she could hardly breathe, constipation, transient hemiplegia, delirium, and an affection of the speech, so that her mother could not understand her. But between the 6th and the 14th January, 1875, all her symptoms began rapidly to subside, and before long the impaired state of her sight seemed to be the only thing that troubled her, except that she was unable to take solid food without vomiting. Dr Goodhart discussed the diagnosis as between meningitis and the presence of a tuberculous mass at the base of the brain; he does not seem to have thought any other view possible.

In describing meningitis we shall mention cases in which symptoms very like those of that disease have been associated with double optic neuritis, but in which, although they terminated fatally, no lesion could be discovered either in the membranes or in the cerebral substance. Surely it is but common sense to suppose that, if one could see the state of the brain in cases which recover, it would be found equally free from obvious morbid changes. Ophthalmoscopic appearances have a value coordinate with that of other symptoms in the diagnosis between organic and functional diseases; but it seems to be thought that they stand on quite a different footing from the rest, and this claim does not appear to be established.

Another question is whether any special inference can be drawn from the exact character of the ophthalmoscopic appearances in a case believed to be

one of local organic disease of the brain. Meningitis often accompanies a gumma of the base; and a tumour in any part may set up extensive yellow softening. It might be thought that neuro-retinitis, as distinguished from choking of the disc, would indicate the presence of such secondary affections. But one of Mr Hulke's cases seems to show that the former change may depend upon the presence of a sarcoma attached to the floor of the skull, without there being any evident inflammation of the membranes or of the brain itself. Nor can simple atrophy of the disc, without antecedent exudation into its substance, be taken as a proof of the existence of hydrocephalus, although Dr Allbutt suggests that distension of the third ventricle often causes such an atrophy, by stretching the optic tracts and the chiasma.

Double optic neuritis certainly most often indicates a tumour, less commonly, meningitis or hydrocephalus or tabes, rarely or never cerebral embolism, thrombosis or hæmorrhage. It may occur in Bright's disease or after fever. Single optic neuritis rarely points to a cerebral lesion. Acute optic neuritis is often caused by a chronic lesion, as a tumour. Primary optic atrophy is not so often a symptom of local cerebral lesions as of more general diseases, such as tabes, general paralysis, and insular sclerosis; but when secondary it has the same significance as the descending optic neuritis, of which it is the effect. Atrophy, like neuritis, of one optic disc seldom points to cerebral disease on the opposite side of the brain, much more often to a "peripheral" lesion affecting the optic nerve or tract.

The *differential pathological diagnosis* between the several forms of local organic disease of the brain often rests on no higher basis than that of probability. In a child, there is a strong presumption in favour of *tubercle*. I may take the opportunity of remarking that although Dr Jackson has recorded several instances of epileptiform convulsions or hemiplegic paralysis in children the subjects of inherited syphilis, there is as yet no pathological evidence that intracranial gummata are ever developed in such cases; so far as we know at present the association may have been a mere accident. The older the patient, the greater the chance that the lesion is some other form of tumour than tubercle; and above the age of forty the latter may be left out of consideration. To distinguish between tubercle and glioma or sarcoma is, however, of little consequence; what is really important is that one should never overlook *syphilis* as a cause of cerebral disease. It is particularly to be noted that in many instances in which gummata are developed in the brain, no nodes upon the bones can be discovered, nor any indications of past or present orchitis, iritis, or cutaneous eruptions. In only three or four of the ten fatal cases of this kind that I have recently met with was it ascertained during life that the patient had suffered from any venereal disease. There are of course persons—especially unmarried ladies—in whom it would be unpardonable to suspect the existence of a syphilitic taint. But on the other hand, it may be of the highest importance that neither the high social standing of a patient, nor even a general reputation for purity of life, should prevent our giving him the chance of being saved from what may be the remote effects of a long-forgotten transgression. Dr Buzzard lays great stress on the presence of a muddy complexion, and other signs of a cachexia not traceable to any definite visceral disease, as pointing to syphilis. It is well known that in most cases the pains caused by this disease regularly recur, or at least undergo a marked aggravation in the evening. What seems to have more weight than

anything else in suggesting syphilis as the cause of obscure nervous symptoms, is the impossibility of referring them to any single lesion. Dr Jackson long ago insisted on the value of this principle; and Dr Buzzard has illustrated it by cases in point. Thus two of his patients had paralysis of one arm and of both legs at the same time; the inference was that there was an affection of the spinal cord as well as of the brain. I must confess that it is not clear to me that one can ever positively assert that there must be more than a single lesion in the brain itself. Moreover, it would have to be remembered that secondary tumours are often multiple, and that the presence of a primary new growth elsewhere is very apt to be overlooked. Thus a patient coming to a physician for headache and paralysis may very likely never mention that there has for years been a tumour in the breast, or that one testicle was excised some months before. Again, the original seat of the malignant disease may be some internal organ, where such an affection is difficult of detection. Out of sixteen cases at Guy's Hospital of secondary growths in the brain there were no less than six in which the starting-point of the mischief was a sarcoma surrounding the root of one lung, and in five of them this was not discovered during life.

Prognosis.—All that we know of the progress of cases of cerebral tumour or tubercle would tend to show that these diseases are inevitably fatal. The nearest approach to a recovery that I remember to have heard of occurred in a boy, aged fourteen, who was under my care in Guy's Hospital in 1867. About two years previously he had been taken ill with "pain in the back of the head, loss of sight, and fits in which he used to clench his hands." On May 1st, when he had been in my ward for three weeks, it is noted that "he lies apparently unconscious of everything, and cannot be roused. His head is constantly thrown backwards; and when he is touched there is a sort of opisthotonos. He is completely amaurotic; his pupils are equal and slightly dilated." On May 4th the report is, "He is slightly more conscious, he recognises his mother, and will raise his hand into the air when told to do so. His evacuations are passed involuntarily." During the next three months the chief change was that he slowly wasted away, until he was reduced to a mere skeleton; the only sign of intelligence that he ever manifested consisted in lifting his hand, as already described. But one day, in going round the ward, I spoke to him; and to the astonishment of everyone he slowly articulated a few words in reply. From that moment he began to improve. He took food well, regained flesh, talked more and more every day, got up, walked about the ward, and at last was discharged perfectly well except that he was blind. Afterwards he attended among my out-patients. I have no notes of his symptoms during this period, but I remember that he complained of paroxysmal headache, and of epileptiform fits. He was readmitted, and died. The notes of the autopsy have unfortunately been mislaid. But I recollect enough to be able to state that there was an irregular calcareous mass, of about the size of a marble, embedded in the floor of the third ventricle, with some mucoid fluid round it.

Even when the local lesion in the brain is a syphilitic gumma, the prognosis is not very favourable, so far as concerns the patient's ultimate restoration to health. For a time, active treatment is generally followed by very striking results; consciousness is regained, paralysis is recovered from, headache subsides, epileptiform convulsions cease to recur. But after an interval the symptoms return; perhaps the opposite limbs are now

paralysed, or the affection may assume a paraplegic instead of a hemiplegic form. A second course of medicine may again be successful, but at last our efforts are baffled, and the patient succumbs. Such cases are often prolonged over a period of several years. On the other hand, it sometimes happens that the cure is permanent; and more often, that each return is milder than the last, until the disease gradually wears itself out.

Treatment.—In the treatment of central gummata one should not trust wholly to iodide of potassium, even in large doses. A course of mercury should always be prescribed. The bichloride may be given internally, or recourse may be had to calomel-vapour baths or to inunctions with blue ointment.

Such a line of practice often proves brilliantly successful where there was no proof that the symptoms were due to a syphilitic lesion. The question then arises whether the proof is supplied by the result of the treatment. Upon this point I would not speak dogmatically, but it seems to me not improbable that absorbent remedies may, in some cases, possess the power of arresting the growth of sarcomatous tumours. Wunderlich believed that small doses of arsenic, continued for a long period of time, were sometimes useful.

The palliation of symptoms is always possible. If there be epileptiform convulsions, full doses of bromide of potassium should be given. The same remedy will often relieve headache or giddiness. Or it may be necessary to administer opium or morphia by the mouth, or to inject the alkaloid subcutaneously. Dr Reynolds speaks highly of Indian hemp, as sometimes altogether removing pain in the head. He also recommends the local application of ice.

The recent achievements of antiseptic surgery and the power of localization which we have seen that experiment and observation have now conferred on the physician, have led to the bold attempt to remove a cerebral tumour by trephining and enucleation. Mr Victor Horsley has recently published a remarkable series of not less than ten cases in which the seat of the cortical lesion was diagnosed and its removal successfully accomplished ('Brit. Med. Journ.,' April 23rd, 1887). In half of these cases the morbid condition was not a new growth, but some inflammatory or degenerative change in the brain or meninges. However, in one it proved to be a tubercular tumour, in a second a glioma, in a third a large Pacchionian body, in a fourth a tumour weighing 4½ oz., and in a fifth a tubercular tumour. In the last of these cases the operation was fatal, in the second the glioma returned and proved fatal six months afterwards; in the three others the patient recovered from the operation, and the convulsions were rendered less frequent or the pain was removed.

Some years ago I had a patient subject to convulsions and other symptoms which led to the diagnosis of a "coarse" lesion in the left præfrontal lobe. Hoping that it might be a cyst or abscess, I asked Mr Symonds to trephine over the spot where I supposed it to be. He did so, and found a large and firm tumour. No attempt was made to remove it, and death followed thirteen days afterwards.

It must be confessed that only a small number of cerebral tumours would be amenable to surgical treatment even if we could always accurately determine their situation. Many are too deeply seated, others are multiple, others not sufficiently circumscribed, others of malignant nature, and others only come under notice too late for treatment. The syphilitic growths,

moreover, may be better cured by drugs. A hundred cases from the records of Guy's Hospital were reviewed, with reference to the possibility of operation, by Dr Hale White ('Guy's Hosp. Rep.,' vol. xliii, p. 117). He concluded that not more than three tubercular tumours, not more than four or five gliomata, and only one sarcoma, two cysts, one myxoma, and two tumours of doubtful origin—in all ten certainly, and four probably, of the 100—were removeable by operation, provided that their exact seat had been previously ascertained.

Red Softening.—We have still to describe those organic diseases of the brain which consist in local, circumscribed inflammation. These are commonly said to be two in number—red softening and abscess.

The former, *ramoulissement rouge*, is amongst the most curious morbid changes to which the cerebral tissue is liable. It is attended with great swelling; convolutions so affected are far broader, and the sulci between them much deeper than natural; while the corpus striatum or thalamus is rounded, prominent, and generally increased in size. As is implied by the name, there is a marked diminution of the natural firm consistence, and the colour is altered; grey matter assumes a deep purple tint, and white matter becomes pink or red, with numerous minute ecchymoses. Under the microscope the capillary and other vessels are found dilated and engorged; blood-corpuscles are seen extravasated among the nervous tissues; if the affection has been of sufficient duration, they are fused together, having undergone conversion into shapeless masses. According to Rindfleisch, pus-cells are collected about the smaller blood-vessels. Moxon, however, says that he has been surprised to find how slight were the histological changes in some cases of this kind: the elements of the tissue were softened and granular, but they still retained their form, and no characteristic inflammatory products could be recognised.

Red softening may arise from a variety of causes. One of them is injury. Some years ago a woman died in Guy's Hospital, who had fallen three or four months previously, and struck her head against the wall. Three weeks before her death she had a fit, which was followed by a partial left hemiplegia, affecting the side of the face and tongue, and accompanied with ptosis. On *post-mortem* examination all the parts at the base, between the optic commissure and the cerebellum, were found by Dr Wilks to be in a state of softening, "partly red and partly white;" one third of the thickness of the pons was so affected, and nearly the whole of the crus cerebri. Again, red softening may accompany an inflammation of the membranes, as tubercular meningitis. We have already spoken of it as sometimes arising in the neighbourhood of an apoplectic clot, round a tumour, or in a part of the brain deprived of its blood supply by thrombosis or embolism (cf. p. 560).

It is a question still undecided whether red softening of the brain ever occurs as a primary and independent morbid process. In the records of *post-mortem* examinations at Guy's Hospital there are very few cases that could be so interpreted. In two instances the occipital lobe of the left hemisphere of the brain, and a considerable part of the cerebellum on the same side, presented the change in question; but in each of them the cardiac valves showed recent vegetations, so that it seems probable that the affection was dependent upon embolism of some of the smaller arteries. In 1876 I made an autopsy in which parts

of the right superior and middle frontal convolutions were swollen, soft, and of a grey-pinkish colour. There was no caseation and no definite edge such as would have suggested the presence of a new growth; I should have regarded the affection as an acute cerebritis had not the microscope revealed the presence of a large number of oval and round cells infiltrated between the nerve-fibres. I should therefore be sceptical about any case of supposed primary red softening or local cerebritis, unless a careful examination of every part of the affected structures had been made. Some years ago a woman, aged twenty-six, died in the hospital under one of my colleagues, after an illness of three months' duration. The falx cerebri was found adherent to the anterior parts of both hemispheres by granulation-tissue. On section the frontal lobes appeared of a brick-red colour; their cineritious substance was swollen, and the boundary-line between it and the white matter was ill-defined. On the left side this change extended down to the lateral ventricle. The affected parts were rather harder than natural; but in all other respects the disease corresponded perfectly with the descriptions which writers have given of a local cerebritis; and, unless there was a new growth which was overlooked, I think it must have been of that nature. The microscope only showed a corpuscular infiltration, with granular matter and compound granule-masses; but I am not sure whether attention was directed to the possible presence of a small new growth in the midst of the inflammatory mischief. There was nothing in the symptoms to indicate the character of the morbid process.

Huguenin expresses doubts as to the existence of a "spontaneous encephalitis," but he nevertheless describes various retrograde changes as consecutive to red softening in such a way as to imply that it may be a substantive disease. He says that the patches may either subside entirely, or pass into a condition of yellow softening with cavities full of serum, or undergo cicatrisation, or become converted into tough, dirty-white, indurated masses. I have never seen such appearances in any case in which there was reason to suppose that a condition of red softening had existed, rather than an effusion of blood or an infarctus; and I do not understand on what evidence Huguenin's statements are based.

Abscess of the Brain.—This, again, is an affection which is decidedly of rare occurrence, both absolutely and in comparison with many other cerebral diseases.

Origin.—Most pathologists speak of suppuration in the substance of the brain as a further stage of red softening. It is true that in cases of pyæmia one sometimes finds reddened patches apparently antecedent to the formation of the pus; indeed, a similar condition is also now and then seen in the neighbourhood of an actual abscess. In the latter case, however, it might fairly be regarded as the result of a distinct morbid process affecting the brain tissue round the wall of the abscess, just as it may affect that which surrounds a tumour or an apoplectic clot. And there are not wanting facts which seem to show that abscesses generally arise independently: one is that when several suppurating cavities are found in the same hemisphere—however small and recent they may be—the intervening cerebral substance is commonly pale and of firm consistence; the other is that the causes of red softening are different from those of abscess. We have seen that the former affection is attendant upon some other morbid change in the same part of the brain; but this is very rarely the case with the latter.

Ætiology.—Cerebral abscess is probably never a primary affection, except when it is traumatic; but the diseases to which it is secondary belong not to the brain itself, but to more or less distant parts. Chief among them are *diseases of the ear*, especially suppurative inflammation of the tympanum. Otorrhœa, if prolonged, always involves the risk of extension of mischief through the bone to the internal surface of the skull, so that a person affected with it should never be taken for life assurance at ordinary rates. In the 'Medical Times and Gazette' for 1863 a case is recorded of a patient who for several years had a discharge from the ear, but who lived to the age of sixty-six and then died of a cerebral abscess. And it often happens that an aural affection which had been present from early childhood kills a grown-up man or woman. In many instances the several stages of the morbid process are plainly traceable after death. The tympanic cavity is found to be bare and carious, or even necrotic; the dura mater over its roof is raised from the bone by pus, or it is softened and sloughing; the arachnoid and pia mater adhere firmly to it, and at this very spot the temporo-sphenoidal lobe of the cerebrum contains an abscess which comes close to its under surface. Or, on the other hand, the caries may pass from the mastoid cells or from the petrous bone to the posterior fossa of the basis cranii, and then the abscess occupies the corresponding half of the cerebellum. I find that among twelve successive cases at Guy's Hospital there have been seven in which the middle lobe of the cerebrum was the seat of the abscess, five in which it lay in the cerebellum.

The late Mr Toynbee, many years ago, believed that he had established a rule that abscess of the cerebrum follows caries of the tympanum, abscess of the cerebellum phlebitis of the lateral sinus and external meatus, and abscess of the bulb caries of the labyrinth. The third situation is too rare to be taken into account; but Sir William Gull, and subsequent German writers, confirm Toynbee's statement of the connection between disease of the middle ear and abscess of the cerebrum, and between the external ear and abscess of the cerebellum.

According to Huguenin and Meyer, the right side of the encephalon is much more apt than the left to be affected with abscess from disease of the ear. Indeed, among twenty-four cases of this kind collected by Gull and Sutton, the right side was affected in no less than eighteen, but it is curious that out of eleven of the cases that have occurred at Guy's the abscess was on the left side in six, on the right side in only five.

Aural surgeons formerly laid great stress on the diagnosis of caries of the tympanum, as indicating the danger of extension to the brain. They did not hesitate to pass a probe into its interior in search for rough and denuded bone, but apart from the risk which such a procedure carries with it, of breaking through the wall, and so of setting up the very mischief which is dreaded, experience has shown that the detection of caries is less important than was supposed. In many instances of cerebral abscess set up by inflammation of the middle ear, the bone is found after death to be healthy. There is more than one route by which the morbid process may reach the interior of the skull in such cases. It may pass along the bony canals which transmit the superficial petrosal and other veins, or through the spaces in the diploë which convey vessels from the tympanum to the dura mater, or it may first extend to the labyrinth and then follow the course of the seventh pair of nerves. A curious circumstance is that in many instances there is a tract of apparently healthy cerebral substance between the wall of the abscess and the

petrous bone. It is especially when the inflammation of the ear assumes a putrid character that one should be on the look out for the speedy supervention of disease within the cranium. Von Tröltsch mentions, as a sign of danger, the circumstance that lead lotions are blackened by the pus; other indications are the suppression of the discharge from the external meatus, and a sudden increase of pain in the ear, which may amount to the most intolerable agony. But in some cases there is no pain at all: there may even be no otorrhœa, for the secretion may be retained behind a perfect membrana tympani; and thus, unless one tests very carefully the patient's auditory powers, we may easily overlook the fact that he has anything the matter with his ear.

Another, but a far less frequent cause of abscess of the brain, is chronic *disease of the nose*. Two such instances were recorded by Sir William Gull in the 'Guy's Hospital Reports' for 1857. Each patient had had mucous discharge from the nostrils; in one case the abscess was in the middle lobe, in the other in the anterior lobe. Other writers have given cases in which a nasal polypus was the starting-point of the mischief. I once made an autopsy in which an ulcerating epithelioma of the lip and cheek extended to the base of the skull along the third division of the fifth nerve, and set up an abscess in the middle lobe of the brain. Necrosis or caries of the calvaria from any cause, if attended with sloughing of the dura mater, may have a like effect. Another source of suppurative meningitis or of cerebral abscess is carbuncle of the face.

General *pyœmia* sometimes leads to the formation of one or more abscesses in the brain, as in other parts of the body. In the *post-mortem* records of Guy's Hospital I find six instances of this during the same period within which the twelve cases occurred that I have mentioned of cerebral abscess arising from disease of the ear. Usually the blood disease was directly fatal, but in one remarkable case of a man aged thirty-one, after perineal section for stricture a number of abscesses formed about his body, which disappeared without being opened; he left the hospital and worked in the fields for more than a year, and was then attacked with brain symptoms, which killed him about eighteen months after the operation.

This case is of special interest because it forms to some extent a connecting link with a very curious group of cases of cerebral abscess, which are distinctly secondary to pre-existent suppurative inflammation in a remote part of the body, but in which there are at no period any symptoms of blood-poisoning, and in which no pyæmic infarctus or abscesses are found anywhere but in the brain. I believe that Sir William Gull was the first to point out the ætiology of such cases, although a similar instance had before been recorded by Abercrombie. The most remarkable point about them is the fact that the lung is generally, if not always, the seat of the primary lesion upon which the abscess in the brain is dependent. At Guy's Hospital I find that within the last few years we have had six cases of this kind; six others are given by Gull and Sutton in the second volume of 'Reynolds' System of Medicine,' and some have been recorded in Germany by Biermer, Huguenin, and Meyer. The supposition has generally been that the cerebral inflammation is set up by a portion of thrombus washed out of a pulmonary vein, and carried to the brain in the blood; and Böttcher is said to have found in the floor of an abscess of the brain, which was secondary to a pulmonary abscess, some pigment which he was able to identify as having come from the lung. The nature of the thoracic disease has varied in different in-

stances. Adding the cases which have recently occurred at Guy's Hospital to those related by Gull and Sutton, we obtain a series of twelve examples of this form of abscess of the brain. Among them there are three in which the primary affection was empyema (cf. 'Path. Trans.,' vol. xxviii, p. 4), two of tubercular phthisis, two of some form of acute pneumonia, one of simple bronchitis; in each of the remaining four it seems to have been cirrhosis (chronic interstitial pneumonia), with dilatation of the bronchial tubes or a sloughing cavity in the indurated tissue. Huguenin places bronchiectasis with stagnant putrid secretion at the head of the list of pulmonary diseases which have given rise to cerebral abscess. It is important to note that the mischief in the lung may be altogether latent. In one of the above cases Dr Moxon says that he almost despaired of finding a primary lesion, until at last he discovered that the mucous membrane of the right bronchus was extensively ulcerated, with its cartilages exposed and necrosed; and in the case of which we have spoken, as one of simple bronchitis, the only sign of any pulmonary affection was the presence of some viscid mucus in the tubes. It is of course open to question whether this had anything to do with the cerebral abscesses that were the cause of the patient's death.

Another cause of suppuration within the substance of the brain is direct *injury to the head*, as from a fall or blow. Generally the skull is fractured, and serious symptoms are present from the time of the accident until death releases the patient from his sufferings. Such cases come under the care of the surgeon, and do not require mention from me; but in very exceptional instances the fact that the head has been injured may be overlooked, or the accident may have occurred some time previously, so that its connection with the existing illness is overlooked; and thus the physician may find himself in attendance.

Sir William Gull records the case of a boy, aged sixteen, who was in 1844 taken into Guy's Hospital for what appeared to be slight fever. After he had become convalescent, he was attacked with cerebral symptoms, and ultimately fell into a comatose state. His friends then for the first time mentioned that three weeks before his admission he had been stunned for a minute or two by falling backwards from a cart so as to strike his head upon the ground. There was neither wound nor bruise, but it seems that he complained of nearly constant pain in the head up to the time of his coming into the hospital. He died about two weeks after the accident; a large abscess was present in the left hemisphere. There was also an abscess in the situation of the sphenoidal sinuses beneath the carious olivary process of the sphenoid bone. Could this have been caused by the fall? and, if so, was it not the cause of the cerebral abscess?

Another case occurred to Dr Tuke ('Med. Times and Gaz.,' 1861, i, p. 196). It is that of a man, aged forty-seven, who died after a week's illness with cerebral symptoms. A few months previously he had fallen from a ladder upon his head without having any severe symptoms immediately afterwards. Dr Tuke believed that the accident was the cause of the cerebral symptoms, but it should be noted that, although the patient had suffered from "chest symptoms" a year previously, there is no mention of the state of the lungs at the autopsy.

One would be the more ready to attribute a cerebral abscess, for which we could find no other cause, to any injury of the skull that might have occurred within a year or two previously, because it appears that there is scarcely a case to be met with in which one is absolutely driven to admit that the

abscess is primary and spontaneous. Among seventy-six cases collected by Gull and Sutton for the 'System of Medicine' there are, indeed, a few in which no cause was found; but the only one of which it can be said with certainty that the autopsy was complete is the last of the series, that of a man who died in Guy's Hospital in 1863, and whose body was examined by Dr Wilks. He had been employed at a music-hall, and some years before he had had his chest crushed in an accident, but he was not known to have injured his skull. However, he had led an irregular life, and it is not unlikely that he had at some time or other received an injury. I believe that there has not since been a single instance in which an abscess of the brain has been found in the *post-mortem* room of the hospital, and in which the demonstrator has been unable to trace it to one or other of the causes above enumerated.

Of 100 cases quoted by Mr Barker ('Lancet,' June 11th, 1887), 29 were due to disease of the ear, 27 were traumatic, 20 were associated with suppuration in the lungs or liver, and 7 were due to general pyæmia.

Cerebral abscess, consecutive to disease of the ear, is about equally frequent in males and in females; of the cases secondary to chronic pulmonary affections the larger proportion seem to occur in the male sex.

The great majority of cases, of both forms, are met with in persons between fifteen and thirty years old.

Anatomy.—Abscesses of the brain arising from different causes are to some extent different. Those which result from injury are almost always solitary, and so are those which are secondary to affections of the ear or nose. Out of twenty-seven cases due to aural disease, collected by Gull and Sutton, there are only two in which more than one abscess was present; in one the cerebellum contained three abscesses, in the other there was a collection of pus in the cerebellum, as well as one in the cerebrum. And in all the twelve cases of the same kind that have recently occurred at Guy's Hospital the abscess was single, with the exception of a case in which there was a second smaller abscess by the side of the principal one. On the other hand, the fourteen cases of general pyæmia, in which the brain was the seat of suppuration, yield eight in which the abscesses were multiple, and in most of them there were four, five, or more in different parts of the cerebral substance. So, again, with those cases which are consecutive to affections of the lungs, in seven out of eleven the abscess was multiple. The presence of numerous centres of suppuration might thus go far towards determining the real origin of an abscess.

The cerebral abscess caused by disease of the ear or nose is generally ill-defined, its wall shreddy, and surrounded by softened cerebral substance; but sometimes it is enclosed in a thick capsule. In pyæmia the abscesses seem to be very rarely circumscribed; but in Dr Moxon's case, which lasted eighteen months, the limiting membrane was so firm that it could be lifted out of the brain tissue in which it lay. When the affection is secondary to lung disease there are almost always well-marked capsules. In the 'Guy's Hospital Reports' for 1857 Sir William Gull pointed out that the cyst wall is made up to a great extent of spindle-cells. Rindfleisch has since shown that they constitute its middle layer, there being outside them a plane of fibrous tissue, while the cavity is lined with embryonic tissue, the superficial cells of which are opaque and yellow from fatty degeneration. Even in abscesses of recent formation the pus is commonly greenish and viscid, although it has an acid reaction; but in those which have been of

long standing it is often quite mucoid, of a bright green colour, and alkaline. It may be odourless, or have a nauseous sickly smell, or (when arising by extension from bone disease with necrosis) be horribly fœtid. Mixed with the leucocytes there is always a great deal of granular matter and fat, probably derived from the brain tissue which is replaced by the abscess; and in very old cases one can hardly recognise any pus-cells; they have undergone complete degeneration, and nothing is left but débris.

It is only in cases arising from injury that the commencement of the cerebritis can be fixed with sufficient accuracy to enable an opinion to be formed as to the length of time required for the production of a capsule. So far as I can interpret the evidence which has been collected by Lebert and Meyer with regard to this point, it goes to show that by the end of the third week the abscess cavity may be found circumscribed, but that a definite membranous cyst wall is not found before the sixth or seventh week, and sometimes not until a far longer period has elapsed. Sir William Gull has pointed out that this might become a medico-legal question in some cases of a man dying of cerebral abscess after receiving a blow from another person, if an interval had elapsed so that no direct clinical connection could be traced between the supposed cause and the effect. He cites one instance in which the absence of a limiting membrane was taken as proof that the disease could not have been the result of a severe fall eighteen months previously; and another, in which its presence showed that the suppuration in the brain had not been due to a pyæmia, which might have been set up by an attack of smallpox within the last three or four weeks before death.

An abscess of the brain, when solitary, generally reaches a considerable size before it destroys the patient. It is frequently as large as a hen's egg, and sometimes larger. It often reaches close to the surface of the hemisphere, and is said sometimes to break through beneath the pia mater, and set up a rapidly fatal meningitis; of this, however, I have met with no certain example. When it is secondary to disease of the ear with extensive destruction of the bone, and when local adhesions of the membranes have been found, it sometimes happens that pus from an abscess in the brain is discharged externally through the auditory meatus; such cases have received the name of "cerebral otorrhœa." So, again, an abscess set up by mischief in the nose may break through the ethmoid bone. Some writers suppose that it is possible for an abscess of the brain to cause adhesions of the membranes over some part of its surface, and then to eat its way through the skull, and burrow beneath the temporal muscle, or under the aponeurosis of the scalp; but I am very sceptical about such an occurrence; except, indeed, in cases of severe injury to the head, in which the bone is destroyed by an independent inflammatory process. Far more frequently a collection of pus within the hemisphere penetrates inwards towards the lateral ventricle. At Guy's Hospital this has been found in four or five out of the last seven cases in which such an affection has been set up by mischief in the ear; and it is by no means infrequent in cases which are secondary to some pulmonary disease. Sometimes there is merely an extension of the process along the brain-substance which intervenes between the abscess and the ventricle; sometimes the one ruptures into the other. In the latter case the pus may be found collected in one of the cornua, into which it had fallen by gravitation, or the whole of both lateral ventricles may be full of pus, their ependyma intensely inflamed,

thickened, grey, and velvety; and the morbid action may even spread through the third and fourth ventricles to the subarachnoid spaces of the brain and spinal cord.

Symptoms.—The symptoms of abscess of the brain vary widely in different cases. It has been said that there may be absolutely none, and the affection be accidentally found after the death of the patient from some other cause. Of this, however, I have met with no example, either in books, or in our records of *post-mortem* examinations. But in the large majority of cases, symptoms are present during a period much shorter than that through which the mischief in the brain must have been going on; in other words, the disease is commonly altogether latent during a certain part of its course. This latent period may be only a week or a fortnight, but often it extends to several months, and occasionally even to years. In cases of general pyæmia, the occurrence of suppuration in the brain is not infrequently masked under the delirium and stupor which may accompany any form of severe blood-poisoning.

The earliest symptom of cerebral abscess is, as a rule, *pain*. This varies greatly in severity; sometimes it is of the most agonising character. Gull and Sutton speak of one patient as continuously holding his head with both his hands, and of another as walking about with his hands pressed against one side of his head, and calling out, "Oh my head! Oh my head!" Another could not help screaming; and although perfectly sensible, would tear and bite anybody or anything near him, at the same time expressing contrition for what he was doing. The pain is generally continuous; but it is sometimes intermittent, especially at first. Anstie mentions the case of a boy who for three months complained of no symptom whatever, except of a pain which came on in attacks very closely resembling those of migraine, not oftener than once in ten days or a fortnight, and lasting for some hours at a time, nearly always ending in vomiting, and disappearing after sleep. In some instances the seat of the pain corresponds closely with that of the abscess; Gull and Sutton mention the case of a boy who had almost constantly a burning pain over the front and right side of the head, and in whom the disease was in the anterior lobe of the right hemisphere. But they go on to speak of a patient who had an abscess in the cerebellum with pain in the forehead, and of another who complained of the left side of his head, but had an abscess in the right middle lobe.

Much less frequently an *epileptiform seizure* forms the starting-point of the symptoms; and such seizures may be repeated at intervals for a considerable period, before any further sign of illness manifests itself. Or *rigors* may occur, sometimes with such regularity that the case might be mistaken for one of ague. Or there may be *vomiting* from time to time, without any apparent cause. Or the first thing noticed may be a dulness of expression, a change of disposition from cheerful to morose or melancholy, a disinclination to speak, loss of memory, or inability to sleep. The pupils are sometimes sluggish and they may be unequal in size; the optic discs have been found congested and œdematous. Gull lays stress on rapidly increasing emaciation as having been a principal symptom in some cases. The pulse is sometimes slow; Huguenin relates a case in which it fell to fifteen and even to ten in the minute.

The temperature is often normal, probably as a rule, unless there is meningitis or pyæmia in addition. In a case under the editor's care in January, 1886, the patient, a girl about twenty, had a temperature of 106°,

with symptoms of pyæmia and basal meningitis. There were found, beside suppuration of the liver and other organs, a large abscess in the left hemisphere, which had opened into the ventricle, a second in the lower part of the ascending parietal gyrus, and a third in the præcuneus. There had been no hemiplegia.

Localising symptoms.—In the symptoms hitherto mentioned there has been little or nothing to indicate that one part of the encephalon rather than another is the seat of disease. But in exceptional cases "localising" symptoms are not wanting. Aphasia may be present, which shows that the abscess is in the back part of the left frontal lobe. Much more frequent is hemiplegia, partial or complete. This indicates that the part affected is near the fissure of Rolando; or that the inflammatory process extends inwards, so as to involve the internal capsule or corpus striatum. Paralysis of the seventh or of the ninth nerve, the arm and leg being free, is said by Huguenin to show that the mischief is seated in the anterior lobe. We have already cited a case of Hitzig's in which spasms confined to certain muscles were set up by a small abscess limited to a small part of the cortex. But, on the other hand, Sir William Gull has related in detail the case of a gentleman who on many occasions had a sudden convulsive affection of his right arm—so violent that he had to support himself by holding on to the table with his other hand, and yet so devoid of pain that he was amused by it—but in whom the abscess—a large one—was in the occipital lobe.

Huguenin finds that about one fifth of the total cases of cerebral abscess are situated in the frontal lobe, one eighth in the occipital, one fifth in the temporo-sphenoidal, a very few in the parietal, fewer still in the pons or bulb, and the majority of the remainder in the cerebellum.

It seems to be very doubtful whether there are any "localising" symptoms peculiar to abscess of the cerebellum. Huguenin says that the pain is not only referred to the occiput, but often extends down the back of the neck. He also states that the pupils of both eyes are commonly dilated, that vomiting is peculiarly severe, and that the gait is sometimes unsteady, like that of a person affected with locomotor ataxy. In one case which occurred at Guy's Hospital I find it noted that the patient was so feeble as to be hardly able to sit up in bed; but in that instance (as in many other cases of cerebellar abscess) the lateral ventricles of the cerebrum contained a large excess of fluid, a result, in all probability, of pressure upon the veins of Galen.

Mode of death.—None of the symptoms which have been described are such as would suggest to an unskilled observer that the patient's life is in any imminent danger. But after they have been present for some days or even for several weeks—or, sometimes, without any one of them having existed to indicate that the brain is diseased—he falls, often suddenly, into a state which is obviously most alarming. Violent delirium may set in, followed by stupor and coma; or a severe epileptiform fit may occur, after which he may never regain his consciousness. In 1876 a girl, aged eighteen, was admitted into the Clinical Ward one afternoon at five o'clock. She had for six days been suffering from sickness and diarrhœa, with severe headache, so that she was said by the medical man who attended her to have typhoid fever. She then spoke rationally and answered the questions that were put to her, but seemed rather strange in her manner. At eight o'clock the same evening she suddenly made a great noise; the house-physician was hastily summoned, and found her partially insensible, but

capable of being roused so far as to say that she was going to die. She seemed to have loss of power in her left arm and leg. An hour later, she all at once ceased to breathe. Artificial respiration was kept up, and the heart continued for some little time to beat rapidly, but it soon slackened, and in ten minutes she was dead. We found four or five abscesses in the posterior and middle lobes of the right hemisphere of the brain.

In the case just referred to—and in several other cases in which the fatal termination was equally sudden—there was no extension of the inflammatory process towards the lateral ventricle. I am inclined to be sceptical as to the possibility of diagnosing that occurrence. Huguenin thinks that the rupture of an abscess into the ventricle may sometimes be recognised by the supervention of spasms in both sides of the face, or in both legs, without loss of consciousness; and he gives cases in which these symptoms were present. The point, however, is one of no practical importance.

The terminal stage of a cerebral abscess is seldom of long duration. But it may last for a week, and in exceptional cases, longer still; during which time the patient is alternately delirious and in a state of stupor, and commonly passes all his evacuations under him in bed. Sometimes there is a transient recovery from such symptoms, only to be followed by their return, and by a fatal termination to the case.

Diagnosis.—That the diagnosis of abscess of the brain is often beset with difficulties must be sufficiently apparent from what has been said of its symptoms and of the conditions under which it occurs. More than once—when speaking of ague, of enteric fever, and of neuralgia—we have pointed out how necessary it is to bear in mind cerebral abscess as a possible explanation of doubtful cases. As between abscess and other organic affections of the encephalon, the only positive criterion is the presence of shivering; and that symptom is often absent.

Thus, in practice, we are able to detect the disease in question only when some one of its recognised causes is known to be in operation. And since it seldom, if ever, arises spontaneously, this would generally give us a good opportunity of diagnosis, but for the fact that both the patient and his friends too often ignore, or even explicitly deny, the existence of these several causes, otorrhœa, deafness, or bronchial and pleuritic affections. Moreover, each of the affections that may set up suppuration in the brain is liable to give rise to other forms of cerebral disease. Thus injury is not infrequently followed by meningeal apoplexy. Again, we can seldom speak with certainty of cerebral abscess as being consecutive to any pulmonary affection, unless we are in a position to exclude the possibility of its being phthisis; for that disease is sometimes accompanied by solitary tubercle of the brain, and very often sets up tubercular meningitis, which is itself a very insidious malady, and attended with the most varied symptoms. Lastly, disease of the ear may either be the starting-point of a general meningitis, or cause thrombosis of the lateral sinus, which for some unexplained reason is now and then accompanied by well-marked signs of cerebral disturbance. To this affection we shall return when dealing with inflammation of the meninges.

Treatment of abscess of the brain resolves itself into the possibility of surgical interference.

Mr Hilton used to quote in his lectures a celebrated case of Dupuytren's in which he trephined the skull expecting to find pus under the bone; he then incised the dura mater and finally thrust a bistoury into the brain,

gave exit to a quantity of pus, and cured the patient. Similar cases have occasionally been recorded since. In 'Ziemssen's Cyclopædia,' Huguenin mentions twenty-six cases of supposed success in opening abscesses of the brain. The operation was a much more desperate one before the methods of antiseptic surgery were adopted.

When the abscess results from otorrhœa, the mastoid bone has been trephined and pus successfully evacuated.

In a patient under the editor's care in Guy's Hospital, in July, 1879, the symptoms and previous history pointed strongly to an abscess in the anterior parietal region of the left side. Mr Lucas trephined, opened the dura mater, and incised the brain, but without result. No harm was done, and the symptoms of pressure were somewhat relieved; but when death occurred a few days later we found a focus of red softening with infiltrated pus in the corona radiata, close to the part operated on.

With increased means of accurate localization and advancing surgical experience, we may hope that the number of cases of cerebral abscess successfully treated by operation will increase.

CHRONIC AFFECTIONS OF THE BRAIN DUE TO DIFFUSED ORGANIC LESIONS

Chronic diffused inflammation: its great rarity—Hypertrophy of the brain.
HYROCEPHALUS: *in children—Origin—Pathology and local anatomy—Effect upon the skull—Symptoms, course, and event—Treatment—Hydrocephalus in adults—Cases.*
GENERAL PARALYSIS OF THE INSANE—*History—Ætiology—Symptoms and course—Anatomy—Diagnosis—Treatment.*
Other forms of cerebral atrophy—Senile—Alcoholic—Saturnine, &c.

IN 1860, Griesinger, in a paper in the 'Archiv der Heilkunde,' drew attention to the importance of distinguishing among organic affections of the brain those which are limited to some particular region, "focal" (*herdartig*), from those which are diffused. Of the diseases which he placed in the latter class some, belonging to the membranes rather than to the cerebral substance, will be separately described. But there remain others, which we will discuss in the present chapter. As might be expected, they are characterised by an absence of "localising" symptoms. Griesinger briefly sums up their effects as consisting of "giddiness, diffused headache, delirium, and mental failure—from slight dulness of intelligence up to imbecility—drowsiness and stupor, tremors and involuntary quiverings of the muscles, vomiting, and, lastly, the more positive indications of augmented pressure within the cranial cavity, slowing of the pulse and of the respiration."

In the diagnosis two points have to be borne in mind. (1) That local affections may produce like symptoms, if they happen to take up a large space, and especially if they are seated in a part of the brain to which no special function is ascribed; (2) that they are very apt to be confounded with certain Neuroses, and particularly with the various forms of insanity, in which no organic change is present.

One peculiarity of the chronic diffused affections of the brain is that many of them occur at particular periods of life with especial frequency, or that their symptoms vary with the age of the patient. And, as the order in which they are to be taken is otherwise unimportant, we arrange them, for convenience of clinical comparison, in an ascending series, beginning with those which are seen in children, and leaving to the last those which are most common in middle-aged and in old persons. We shall then have:

1. Chronic diffused "cerebritis" or encephalitis.
2. Hypertrophy of the brain.
3. Hydrocephalus of early childhood.
4. Hydrocephalus of adult life.
5. Atrophy of the brain, including—
   - *α*. General paralysis of the insane, or Dementia paralytica.
   - *β*. Atrophy from old age, alcohol, lead, or other causes.

Chronic Diffused Inflammation—*General sclerosis of the brain.*—If we consider how important a part in the pathology of the spinal cord is played by diffused chronic inflammatory changes, we shall surely find it remarkable that similar lesions are hardly known to occur in the brain ;* certainly there is no form of disease which comes under that description, and which is met with commonly and indifferently among persons of both sexes and at various ages. I have, however, made autopsies in two cases in which such a lesion was the cause of death.

One of them occurred in the practice of Dr Wilks, who has already recorded it in the 'Guy's Hospital Reports' (Series iii, vol. xxii, p. 22). A girl, aged fourteen, was admitted in 1876 for a general failure of mind or body, which had been coming on for about a year. The earliest symptom was said to have been an attack of weakness and loss of sight, which came on one day while she was out on an errand. Soon afterwards she had two epileptiform seizures, which began with a scream. She generally lay quiet, with a vacant stare, making no complaints, but smiling when spoken to. She could evidently see, and the optic discs were normal, but the pupils were dilated. If asked what her name was, or any other simple question, she answered sensibly, but remarkably slowly. When food was put into her mouth she would cease to chew without swallowing it, so that it lay there until removed by the nurse. She often vomited. She was unable to stand, and had very little power over her legs ; even her arms were moved but slowly and feebly. However, a month before her death she tried to strangle herself, and was found with a towel tied round her neck. Her skin appeared to be sensitive. Towards the last she lay with her eyes open, looking forwards, but making no attempt to speak beyond a slight motion of the lips. Her temperature ranged from a little below to a little above normal. Before death her extremities became remarkably livid ; on the feet large bullæ made their appearance, which looked as though gangrene were beginning.

At the autopsy a deep purple discolouration was seen extending to above the ankles, the cuticle was detached from the rete mucosum, and the deeper tissues were all infiltrated with blood. The calvaria was thick and heavy and very asymmetrical. The membranes were a little opaque. There was an excess of fluid at the base. But over the convolutions the pia mater was everywhere abnormally adherent, so that in attempting to strip it off one peeled away a thin granular-looking layer of the cortex, leaving a roughened surface. The brain was small, weighing only thirty-four ounces. Its cortex was not obviously wasted. Its substance generally was tough. The boundary-line between the grey and the white matter was everywhere well defined. The ventricles were very considerably dilated, but their ependyma was not granular, except in the fourth ventricle.

The other case, one of much less clinical interest, occurred in 1878 in a child, eighteen months old, a patient of Dr Pavy's. Its illness was said to have begun with a fit at the age of four months, and from that time it had been subject, especially at night, to seizures, in which it would fight for its breath and appear to be choking, but would afterwards lie insensible for three days at a time, taking very little food, and twitching at the corners of the mouth. Fifteen days before its death it had a fit more severe than any previous one. Eleven days later it was again attacked, and on the following

* Acute myelitis is, however, almost as rarely represented by acute idiopathic softening in the brain as chronic sclerosis of the cord by sclerosis of the brain. The various systemic diseases of the cord have also no counterpart in cerebral pathology.

day it was brought into the hospital. It then lay comatose on its left side with the corners of the mouth drawn down and constantly twitching: when it was moved over, its whole body would become rigid, and the twitching at its mouth was more marked. It appeared to have lost sensation in the left side. The temperature, at first 102·4°, rose to 103·8° before death, and the pulse became scarcely perceptible.

At the autopsy I found the child to be wasted, with a pigeon-shaped chest and rickety enlargement of the ends of the ribs and of other bones. The brain was pale on the surface. Its membranes looked healthy; they were not at all thickened or opaque, but very thin and delicate, as is usual at an early age. On attempting to strip the pia mater from the convolutions, however, it was at once found to be abnormally adherent, so that a thin, uniform, superficial layer of the cortex peeled off with it, leaving a rough granular surface behind. The substance of the hemispheres also was indurated, cutting firmly, and giving sections with sharp resistant edges, but the most striking appearance of all was a discolouration of the white matter of the hemispheres, which had a yellowish tint. This was more obvious on the right side than on the left, and in the anterior and lateral parts of the hemispheres as compared with the posterior parts. It was also more marked in the prolongations of the white matter into the bases of the convolutions than towards the centre of the brain, and in some of the convolutions the limit between the white and the grey substances was ill defined. The grey matter itself looked natural. The ventricles were not dilated. The basal ganglia, the pons, the bulb, and the cerebellum were all of the natural soft consistence.

Portions of the affected tissues from each of these cases were examined after the most approved methods by Dr Savage, of Bethlem Hospital, and also by Dr Frederick Taylor, but no decided histological changes could be detected in them, except possibly a slight excess in the cellular elements of the interstitial neuroglia.

Hypertrophy of the Brain.—It has long been known that in certain cases in which the head appears unduly large, so that the presence of hydrocephalus is suspected, a solid brain really fills the whole interior of the skull. Instances of this kind were recorded by Morgagni; and others in the early part of the present century by several French observers, among whom were Laennec and Andral. The condition has generally received the name of hypertrophy of the brain; but its nature and its relations to other diseases still remain among the most obscure questions in pathology. Virchow several years ago suggested that it consisted in an overgrowth of the neuroglia rather than of the nervous elements of the hemispheres. Sir William Jenner, not long afterwards, maintained that it was due to an "albuminoid infiltration" of the tissues, like that which he believed to cause enlargement of the liver and spleen in rickets; he regarded it, in fact, as one of the minor effects of that diathetic disorder. But it appears to be certain that the subjects of cerebral hypertrophy are not always rachitic. On the strength of a case in which Magnan (in 1874) is said to have minutely examined the structure of a child's brain so affected and to have found it perfectly normal, D'Espine and Picot, with other writers, continue to describe the disease as a true overgrowth of all the various elements of the cerebral substance. This conclusion, however, we can scarcely accept, not only because of its inherent improbability, but still more on

account of there being other lesions of the brain (*e. g.* "diffused chronic cerebritis" and "yellow softening") which are easily recognised by the naked eye, but in which the microscope equally fails to reveal marked histological changes. Indeed, it is worthy of notice that in each of the two cases of which Andral has given full descriptions, the grey substance of the convolutions is said to have been undistinguishable from the white by its colour, while the white substance itself resembled white of egg hardened by boiling, and in some parts was harder still. How can such a condition be distinguished from sclerosis? Surely no reliance can be placed on cases of supposed hypertrophy of the brain recorded at a time when so very little was known of the pathology of the organ.

Dr Fletcher Beach, of the Darenth Asylum for Idiots and Imbecile Children, has kindly furnished the following notes of two among six cases of this affection that have come under his notice. He has found that the white matter showed "a uniform granular appearance under the microscope, with nerve-cells scattered sparsely throughout. There were a number of leucocytes present. The increase in size was evidently due to the large amount of granular matter."

In each instance the size of the head was a conspicuous feature during life. In one, a boy, aged sixteen, it measured twenty inches in circumference, in the other, a boy, aged ten, the measurement was twenty-two inches. Its form was, in each instance, square, not, as in hydrocephalus, globular. Other points of distinction from that affection on which Dr Beach lays stress are that the enlargement is more marked just above the superciliary ridges than at the temples, and that the fontanelle is often depressed instead of being full and elastic. This last character seems hardly to consist with the accounts which have been given of the dura mater bulging as soon as the calvaria was opened, and of flattening of the convolutions. Indeed, although the brain in the older of Dr Beach's patients weighed no less than sixty-two ounces, he speaks of the subarachnoid fluid being increased, and of the ventricles being rather dilated.

None of Dr Beach's patients presented any signs of rickets, but, as he himself remarks, it is possible that they may have been rachitic at an earlier period, since they were, with one exception, above the age of ten years when they came under his observation, and since they were in fairly good bodily health. It appears to be still doubtful whether or not the present category will hereafter be found to include all cases of rickety enlargement of the head without hydrocephalus. Another affection, the relations of which to hypertrophy of the brain have still to be determined, is one which is liable to be mistaken for tubercular meningitis, namely, what is called spurious hydrocephalus.

Clinically it would seem that hypertrophy of the brain is characterised by a more or less marked deficiency of intelligence, drowsiness, headache, and a liability to epileptiform convulsions; the gait is sometimes slow and tottering, the weight of the head may cause it to hang forwards, or may even from time to time throw the patient down upon his face when he is walking. Under favourable circumstance life may be prolonged for years. The disease ends either by some intercurrent pulmonary affection, or by gradual exhaustion, or by the supervention of acute symptoms and coma which, in one of Dr Beach's cases, were due to acute suppurative meningitis.

With regard to treatment nothing is as yet known.

HYDROCEPHALUS OF EARLY CHILDHOOD.*—Among the diseases to which infants and young children are liable there is one which consists in a distension of the cerebral ventricles with fluid. The epithet "chronic," although until lately needed to distinguish the affection in question from that which was known as "acute hydrocephalus," is unnecessary now that the latter is always spoken of by its proper name of tubercular meningitis.

Origin.—Hydrocephalus is not infrequently *congenital.* At birth the fœtal head may be so big as to prevent its passage through the maternal pelvis until it has burst or has been perforated by the instruments of the accoucheur, with a necessarily fatal result. Or, the enlargement being less considerable, expulsion may at length take place, and the child may live for a shorter or a longer time. Or it may be noticed that the head of the newly-born infant is softer and more pulpy than natural, there being no obvious increase of size until a few weeks have passed. Or, lastly, no one may observe anything is amiss with the child until it is some months old. Among twenty-six cases collected by Dr Dickinson ('Lancet,' ii, 1870) there were four in which the disease was said to have been present from birth, sixteen in which it was discovered within the first six months, six in which it was not noticed until between the sixth and the twenty-sixth month. One must bear in mind that these figures express, not the relative frequency of the congenital and acquired forms of the disease (for the list probably includes no infants born dead, or dying within the first week or two), but the extent to which a congenital origin is traceable among cases of hydrocephalus brought under medical care later on in childhood. It is evidently impossible to determine in how many of them the hydrocephalus really began *in utero;* but in all probability the brain was healthy at the time of birth in the large majority. We shall afterwards see that the affection sometimes arises at a more advanced period of life than in any of Dr Dickinson's cases.

Thus it is clear that a great practical inconvenience must result from the attempt to describe separately the congenital and the acquired forms of hydrocephalus, as is done by Huguenin in Ziemssen's 'Handbuch,' nor does there appear to be any difference in their pathology, such as would warrant one's drawing a sharp division line between them. There is, indeed, one supposed cause of "water on the brain" which can give rise to the disease only in the fœtus, and during early infancy, namely, a deficiency of resistance on the part of the parietes of the head, allowing the normal ventricular fluid to accumulate in excessive quantity, and to distend both the ventricles themselves and the parts outside them. Thus Huguenin says, it is very common for rachitic children, if they are attacked with whooping-cough and bronchitis, to get a form of hydrocephalus, attended with considerable enlargement of the head, and with widening of the intervals between the bones, but in which there is no flattening of any of the cerebral structures, nor any morbid change in the brain, except dilatation of the cerebral ventricles. But I should doubt whether Dr Dickinson and other observers, who have insisted on the great frequency with which water on the brain occurs in those who are the subjects of rickets, would be prepared to admit the validity of these distinctions. The occasional association of hydrocephalus with congenital syphilis is supposed by Dr Dickinson to depend on a defective growth of the cranial bones, resulting from the constitutional malady.

What little beside is known as to the ætiology of hydrocephalus tends,

* *Synonyms.*—Chronic Hydrocephalus—Water on the brain—Dropsy of the head.

for the most part, to support the view that it is of an *inflammatory* origin. Even when it arises in the fœtus some writers have attributed it to a blow or fall affecting the abdomen of the mother. Gölis, however, adduced some facts to show that drunkenness on the part of either of the parents may cause it. A few striking instances of its occurrence in several children of the same father and mother have been recorded. Frank saw in one family six, in another seven cases. Gölis reported the case of a woman who aborted six times in succession with dead hydrocephalic fœtuses at the sixth month, and bore three living children, two of whom died of the disease when eighteen months and three years old respectively.

When it begins after birth, hydrocephalus appears sometimes to be directly traceable to an injury which has set up inflammatory changes in the brain. West records the case of a little girl who, some months before her head began to enlarge, had fallen out of the arms of her nurse, and had subsequently been convulsed and comatose for some hours. In that instance the only structure that is said to have been thickened was the ependyma. But in many cases the membranes at the base of the brain are found opaque and adherent. There has been much difference of opinion as to whether the disease often arises out of an acute meningitis. We shall see that in the epidemic form of that affection such a termination is not infrequent; but this is probably not the case so far as the simple and the tuberculous forms are concerned. Huguenin, however, speaks most positively of having seen hydrocephalus begin with acute symptoms exactly like those of infantile lepto-meningitis, but which, instead of terminating fatally, have subsided and passed into those of a chronic cerebral affection, attended with great enlargement of the head. He says that in such instances he has found the pia mater at the base of the brain thickened and opaque, and the choroid plexuses also showing traces of a former inflammatory change. But it is worthy of notice that in a case of this kind related in full detail by Rilliet and Barthez, in which the early active symptoms lasted six days, it is expressly stated that the ventricular fluid contained only a trace of albumen, that the ependyma was normal in appearance, and that there were no adhesions of the pia mater at the base. On the other hand, it must not be forgotten that such adhesions, when they are present, may have been the result of a *chronic* meningitis.

Pathology.—It has been thought by Huguenin and others that in many cases of hydrocephalus the physical and chemical properties of the fluid enable one easily to determine whether the morbid process was of a passive or of an inflammatory origin. The normal cerebro-spinal fluid is well known to have a very low specific gravity, and to contain scarcely a trace of albumen. Now, in some cases of hydrocephalus the fluid has possessed similar properties; and, according to C. Schmidt, the proportion in it of potass salts to soda salts, and that of phosphates to chlorides, have differed altogether from those which are met with in ordinary serum, and even in fluid derived from the membranes on the surface of the brain, a fact which is cited by Vogel as proving that the hydrocephalic fluid is a specific secretion of the choroid plexuses. But there are other cases in which the fluid has been of higher specific gravity, and has contained from 3 to 11·5 parts of albumen in 1000. Huguenin takes this as proof that there has been an inflammatory change in the plexuses or in the ependyma; in his opinion such a conclusion is justified by the presence of albumen in any proportion above 2·5 parts in 1000. He of course recognises the fact that inflammation is very likely to come on

secondarily in cases in which at first there was a mere accumulation of the normal ventricular fluid; something of this kind is seen when the operation of tapping is several times repeated, the quantity of albumen in the fluid being greater on each successive occasion. But what he does not seem to be prepared to admit is that in cases which are from the first of an inflammatory origin the fluid may be almost free from albumen. I have notes of no less than four instances of this kind which I have observed in the *post-mortem* room. The patients were all adults, and there was no reason to believe that they had had hydrocephalus for more than a few months. Their brain-symptoms, at any rate, were of quite recent development, and in one instance were distinctly traceable to an injury.

There is, indeed, a view which would account on mechanical principles for the presence of a normal cerebro-spinal fluid even when inflammation had been present; but I doubt whether it is tenable. Several pathologists have shown that there is sometimes closure by adhesions, either of the foramen* at the lower angle of the fourth ventricle, or of the aqueduct of Sylvius. And Hilton propounded the doctrine that the obliteration of these channels is often the cause of hydrocephalus, by preventing the outflow of the secretion of the choroid plexuses into the subarachnoid space of the cord, which ought normally to occur whenever an increase in the physiological activity of the brain leads to an augmentation of its blood supply. But it is difficult to see why the fluid should continue to be poured out under the increased pressure which must necessarily result, and which certainly is present in most cases of hydrocephalus. And as the adhesions themselves are the results of a more or less widely diffused meningitis, such as is often attended with an inflammatory change in the ependyma, it would seem more reasonable to regard this as the cause of the effusion than to adopt Hilton's theory. It is well known that in one and the same case the various serous membranes may pour out fluids of very different specific gravity; and I think it is not improbable that the lining of the ventricles of the brain may continue to secrete a fluid containing scarcely any albumen, even when it is affected with a low degree of inflammation. But the question requires further study, and can only be settled by careful observations as to the state of the openings into the fourth ventricle in a series of cases in which the physical and chemical properties of the fluid are also accurately determined.

The quantity of fluid in cases of hydrocephalus of long standing is sometimes very great; six, eight, twenty, even twenty-seven pints have been measured after death in different cases. The hemispheres are then transformed into a thin shell, which perhaps is not more than a line or two in thickness, so that it is difficult to understand how they could have retained any of their functions. The distinction between the white and the grey matter is lost; and the sulci, if visible at all, appear only as shallow grooves beneath the pia mater. The nerve-cells are said to be more or less completely atrophied and destroyed; but accurate histological details seem to be still a desideratum. The corpora striata and the thalami are flattened and very broad, and so are the crura, the optic tracts, the pons, and all the structures at the base. The weight of the brain is in most cases much reduced; but Huguenin says that it has sometimes been found normal or even excessive. Some of the more delicate structures, such as the septum lucidum

* See Cruveilhier, tom. iii, p. 385, and Hilton, on 'Rest and Pain,' 3rd ed., p. 23, and figs. 1, 2, 8, and 9.

and the soft commissure, are defective or absent, probably in consequence of the stretching to which they have been subjected. The foramen of Monro, and the aqueduct between the third and fourth ventricles are generally widely dilated, the latter being sometimes large enough to receive a pencil. The ependyma is generally thick, tougher than natural, and of an opaque white or grey colour. It often contains a large number of amyloid bodies. Its free surface is covered with granulations, or with a number of translucent beads which give it an appearance that has been aptly compared by Dr Moxon with the leaf of an ice plant.

Hitherto I have assumed that the fluid is contained within the ventricles. Most pathologists, however, describe under the name of *Hydrocephalus externus* a separate variety of the disease, in which the seat of the effusion is said to be the subdural or arachnoid space. Now, there is no doubt that this may be the case where there is an extreme degree of malformation of the brain, as in some microcephalic or anencephalous fœtuses, but I am much disposed to agree with Dr Wilks, who has always expressed an utter scepticism as to the occurrence of external hydrocephalus, such as is supposed to be clinically undistinguishable from the ordinary form. The classical example which is quoted by Huguenin and other writers, is that of the young man, James Cardinal, recorded by Bright. The total quantity of fluid in this case was seven or eight pints, and all of it, with the exception of one pint, lay beneath the dura mater at the time of the autopsy There was, however, a hole in the corpus callosum, and Bright himself supposed that the ventricles were the original seat of the effusion, although he seems to have imagined that the fluid increased in quantity after it had escaped from them, so as gradually to bring the hemispheres down to the base of the skull, but Dr Wilks has suggested that the rupture through the corpus callosum did not take place until just before death, and though this way of interpreting the facts is not free from difficulties, I am, on the whole, inclined to adopt it. It is at any rate evident that the case cannot be fairly cited as an instance of a special form of hydrocephalus.

I must not omit to mention that a congenital hydrocephalus is often associated with malformations of other parts as well as of the brain itself. I have two or three times seen it in combination with spina bifida, and in one case the central canal of the cord was also greatly dilated. Again, as I have already remarked, a certain degree of dilatation of the lateral ventricles is commonly found in cases of cerebral tumour. This is seldom a matter of clinical interest, but sometimes when the cranium becomes greatly enlarged the disease has been supposed to be an ordinary hydrocephalus, until at the autopsy the presence of a new growth has been brought to light. A case of this kind is recorded by Huguenin; the patient was a girl, aged six, whose head measured twenty-three inches in circumference. A point of interest is that during the operation of paracentesis an attempt was made to measure the pressure exerted by the fluid, which was found to equal that of a column of mercury an inch and a half high. The fluid contained 3·5 parts of albumen in 1000, so that it could not be supposed to be a mere accumulation of the normal secretion, the result of pressure upon the veins or upon the subarachnoid openings.

The general result of this discussion as to the pathology of hydrocephalus is to lead to the conclusion that the congenital and acquired forms of the disease which are seen in early childhood are alike dependent, in most cases, upon a chronic change, which is probably always of an inflam-

matory nature, in the ependyma and choroid plexuses. There can be no doubt that a deficiency of resistance on the part of the parietes of the head is often an auxiliary cause, but it does not appear that any sharp line of demarcation can be drawn between cases arising in this way and those which are due to the ependymal affection. Now, as we shall hereafter find, older children and adults are liable to a disease which from a pathological point of view is almost precisely similar, and which therefore might seem to have a claim to be described in common with that which occurs at an early age. Clinically, however, this is altogether impracticable. In its symptoms and in its course the hydrocephalus which occurs in those whose cranial bones are firmly united together is entirely distinct from that which is observed in infants and young children; the other diseases with which they may be confounded, and from which they have respectively to be diagnosed, are different, and there are other points of difference in regard to treatment.

Anatomy.—In fact, in the majority of cases enlargement of the head is the first, as well as the principal, symptom of the hydrocephalus of early childhood. Among forty-five cases collected by West there were twelve in which the frequent repetition of fits first drew attention to the existence of cerebral mischief, four in which it began with some other indication of brain disturbance, and six in which it arose out of an acute attack; in the remaining twenty-three cases no definite cerebral symptom preceded the discovery that the head was increasing in size, or that the fontanelles and sutures were unduly wide, although it must be added that the child had often been noticed for some little time to be failing in strength and losing flesh, or to be otherwise out of health.

The effect of the disease upon the cranial bones is to separate them more and more widely except at the base. Trousseau aptly compares the change in their relations to one another with the falling back of the petals of an opening flower. The frontal bone rises vertically above the eyebrows, or even overhangs them; the temporal and the parietal bones arch outwards, so as to hide the ears when the scalp is viewed from above; the occipital bone extends backwards almost horizontally. Thus the head may acquire an enormous circumference. Dr Dickinson had under his care a child, nine months old, in whom it measured thirty-one inches, and instances in which it ultimately reached forty and even fifty-two inches are cited by Trousseau. A strange contrast is afforded by the shape of the face, which appears unnaturally small, with angular features and a sharp chin. The base of the skull is generally narrow, with shallow fossæ. The presence of the fluid within the anterior lobes of the brain affects the orbital plates, so that they become convex downwards instead of upwards, and thus arises a very important character of the disease in an abnormal position of each eyeball; a large part of the iris, and even of the pupil, is hidden by the lower lid, while the sclerotic above the cornea is exposed to view.

The whole of the vertex of the head forms an open area, which may be said to correspond with the natural fontanelles, expanded so as to meet together between the parietal bones with a breadth of some inches, and dividing the two halves of the frontal bone by a cleft that reaches nearly to the root of the nose. There is, however, always a work of ossification going on which tends to cover the brain in, provided that the process of expansion is not the more active of the two. In examining the crania of hydrocephalic children, whose death has occurred while the disease was in progress, I have

found that the original outlines of the bones were still plainly visible, but that they were surrounded by broad zones of new osseous material, marked by radiating lines which showed the direction of their growth. In some cases the closure of the head is greatly advanced by the formation of a large number of *ossa triquetra*. The date at which it is completed varies widely in different individuals. In Dr Bright's patient, Cardinal, the anterior fontanelle is said to have been finally ossified at about the twenty-seventh year. Sometimes irregularities seem to occur in the union of the several bones. At least this has been assumed to be the cause of a want of symmetry in the cranium which has sometimes been noticed. As a rule, the shape of a hydrocephalic skull approaches that of a sphere. It often, however, appears rather quadrilateral in consequence of the very marked character of the frontal, the parietal, and the occipital prominences. The bones are generally very thin and have no diploë; they may even be transparent. But where life had been prolonged past middle age they have sometimes been found greatly thickened; the museum of Guy's Hospital contains a specimen of extensive ossification of the dura mater, which was taken from a case of this kind.

The separation of the bones of the head by an accumulation of fluid within the cranial cavity is not altogether confined to very young children. There is a classical case, recorded by Matthew Baillie, of a boy, aged seven, whose skull had appeared to be firmly united, but in whom at the time of death there was an interval of three quarters of an inch at the sagittal, and one of half an inch at the coronal suture. Dr Dickinson says that the same thing has been known to occur in adults. I believe, however, that even in children it is exceedingly infrequent where the bones have once become interlocked.

Physical signs.—In well-marked cases of hydrocephalus it is easy to transmit a wave of fluctuation from one hand to the other across the distended scalp. The skin and the subcutaneous tissues are exceedingly thin, and the ramifications of large veins are often plainly visible through the scanty hair. The head may appear obviously translucent when a candle is held on the opposite side of it. In Cardinal's case this is said to have been observed when the sun was shining behind him, and up to the time when he was fourteen years old.

Some German observers state they have elicited a *bruit de pôt fêlé* on percussing over a hydrocephalic head, especially when the mouth was opened; it is supposed to have been due to resonance of the air in the vault of the pharynx. Auscultation has also been brought to bear upon the diagnosis of the disease. It is well known that on listening over the fontanelle of a healthy child one hears a systolic murmur; this has been supposed to arise in the veins that open into the longitudinal sinus, as a consequence of their being compressed at the moment when the cerebral arteries become distended with blood. Now, Rilliet and Barthez maintained that in hydrocephalus this murmur is not to be discovered; but more recent observers have shown that it may often be plainly heard, although they admit that it is absent in acute meningitis or where effusion is going on very rapidly. The contradictory statements which have been made with regard to it appear to be in part due to the fact that even under normal conditions it is only audible within certain limits of age (between the eighteenth week and the fourth year).

Symptoms.—One consequence of hydrocephalus is a difficulty in keeping

the head supported. An infant may be unable to raise it from the pillow, or, if placed in a sitting posture, may let it roll backwards and forwards, as if it had no power in its cervical muscles. An older child perhaps sits with the head resting on the table, or walks with it carried between his hands, "just as a milkmaid steadies her pail." This, however, is simply a mechanical effect of the weight of the fluid. The extent to which the vital functions of the brain are interfered with varies widely in different cases. In those rare instances in which a skull already fully developed expands under the influence of the disease (as in Baillie's patient already referred to), headache, stupor, and paralysis of all the limbs may be present for several months before any enlargement is noticed. Such cases in fact are to be regarded as transitional between the hydrocephalus of early childhood and that of adult life. But in young children, in whom the cranial cavity readily yields, it is often surprising how few symptoms of cerebral disturbance can be made out, even when the quantity of fluid is already large. Headache is often present; even very young children show that they are in pain by restlessness and a sad whining cry. Vomiting is of frequent occurrence, and, like the headache, it is apt to be excited by movements of the head, especially when the child assumes the erect posture.

The *sight* sometimes remains good throughout the whole course of the disease. But in many instances there is blindness almost from the commencement. This is due to changes in the optic discs, which are commonly white and atrophied. It is probable that such a condition sometimes occurs primarily as the result of the pressure of the ventricular effusion upon the optic tracts; but in many instances it is secondary to "choking" or to neuritis. Huguenin examined three infants with congenital hydrocephalus between the twentieth and the thirty-fifth day after birth, and in each case found the discs reddened and swollen. The hearing is very seldom impaired; as Dr Dickinson remarks, a young child who is perfectly blind may at once recognise its mother by her voice. The smell is said to be sometimes altogether absent, and the olfactory lobes atrophied. I suppose that in such cases there would generally be other malformations of the nervous centres as well. The taste seems often to be perverted; the appetite, at any rate, is voracious and indiscriminate. Impairment of the muscular power of the limbs may be present in all degrees from a slight failure of coordination in standing or walking, up to a total paralysis. There is sometimes more or less complete hemiplegia or paraplegia, but such symptoms probably depend in most cases upon some additional local lesion of the brain or cord. Spasmodic affections of various kinds occur; nystagmus especially, but also partial clonic or tonic spasms of the limbs, and even general epileptiform convulsions, the latter being very apt to be brought on by any accidental circumstance which shakes the head or gives it a mechanical shock. The legs and arms are often stunted in their growth, and their muscles very small. Huguenin mentions that in one case in which the lower limbs were for a time contracted, but afterwards became relaxed, the muscles were found to have lost their contractility to faradic currents; this of course indicated that there was some secondary or concomitant affection of the grey matter of the cord.

In some cases of congenital hydrocephalus there is no advance of intelligence from the time of birth onwards; they generally terminate fatally at an early age. In other instances the brain develops to a greater or less extent, but very slowly, taking perhaps ten times as long as under normal

circumstances. During childhood such patients would generally be classed as imbeciles. Cardinal, however, went to school at the age of six, and he soon learnt to read well and to write a little; but he was obliged to give up the latter accomplishment, as stooping caused a pain in the head. When he was twenty-nine years old his mental faculties are said to have been "very fair. His memory was tolerable, but it did not retain dates and periods of time; and it was stated of him that he had never been known to dream. There was something childish and irritable in his manner, and he was easily provoked. He was stated not to have sexual desire, but he was fond of society and affectionate to his mother. His voice was not manly, but feeble and somewhat hoarse." He died about three months later, having become exceedingly feeble, with a protracted cold, febrile symptoms, diarrhœa, and loss of appetite. Perhaps if the lungs had been examined it would have been found that phthisis was the cause of death. In the Fulbourn Asylum, Dr Bacon some years ago showed me a woman, aged fifty-three, whose head measured twenty-seven inches in circumference, and who could sing and talk well, and had a tolerably good memory and intelligence. I do not know at what age the disease arose. In some rare cases, in which hydrocephalus thus becomes arrested, the intellectual faculties are said to reach a normal or even an exceptionally high standard. The name of the author of 'Vanity Fair' has often been cited as affording an illustrious example of this. But it is doubtful whether the difficulty of distinguishing a rachitic enlargement of the head from "water on the brain" has been sufficiently taken into account in regard to such cases.

For, strange as it must appear, it is a fact that errors have not infrequently been committed, even by skilled observers, as to the presence of fluid within the cranial cavity; and not only has hypertrophy of the brain been mistaken for it, but it has sometimes turned out that the disease has been nothing more than rachitis. However, one can for the future avoid this particular blunder by measuring the circumference of the skull, and comparing it with what it should be, according to the age of the child, in a table of standard measurements, such as one which is given by Huguenin. And that writer says that even where the yielding of the softened bones leads to an accumulation of ventricular fluid in a rachitic infant, the ophthalmoscope enables the true character of the affection to be easily recognised, since the optic discs retain their normal appearance. But, for my own part, I doubt very much whether these statements are applicable to the cases in which there is really most danger of diagnosing hydrocephalus wrongly. These are probably examples of the affection which has been described as hypertrophy of the brain (p. 646), in which the skull is really much enlarged. I suppose that it was a case of this kind which many years ago occurred to me at the Evelina Hospital, but of which I have unfortunately preserved no notes; during the child's life the presence of hydrocephalus was never doubted by me, nor by anyone else who saw it, although the necessity of caution in the diagnosis of that disease had already before that time been impressed upon my mind; but when the skull was opened it was found to contain nothing but a large solid brain.

Course.—I have already incidentally remarked that the course and termination of hydrocephalus vary widely in different cases. Sometimes the disease undergoes a rapid advance, and destroys life in a few months by coma or by means of a succession of epileptiform seizures. Very often the child dies through some intercurrent malady, such as measles or whooping-

cough; or, if it is rachitic, it is cut off by laryngismus stridulus or by some other complication. In certain very rare cases the fluid makes its way through the parietes of the head, and may even be discharged externally. Rokitansky once saw it poured out through the open sutures and diffused beneath the tissues outside the cranium. Several writers have recorded instances in which it has escaped through the nose or even through the upper eyelid; and some of them have ended in the recovery of the patient. When the discharge takes place into the nasal fossæ, it is supposed that the ethmoid bone must have been loosened from its attachments by the pressure to which it is subjected.

We had lately a child in Guy's Hospital with extreme hydrocephalus, whose case is worthy of note from its having been under continuous observation from the beginning of the disease till its end. The patient was a remarkably fine boy, six years old, free from family proclivity to diseases of the brain. He first, while living in the country, began to lose power over his bladder, then he had occasional fits, vomiting, headache and staggering, with slight loss of power in the limbs, and then dimness of sight. We found optic neuritis. It was at first thought that there was a tumour of the superior vermiform process pressing on the veins of Galen and causing secondary hydrocephalus; but the subsequent course of the disease did not confirm this supposition. The child went through a slight attack of diphtheria. The head gradually enlarged, the gait became feeble, complete blindness with double optic atrophy followed, and at last he lay passive and almost motionless in bed with incontinence of fæces and urine, but almost always free from pain and with no convulsions or paralysis. During the last week there was tonic contraction of the arms with ankle-clonus, and for the first time rapid loss of flesh. He died at the age of eight, having lived nearly two years in the hospital. *Post mortem*, all the organs were healthy, except the brain, which was distended by serous effusion in both ventricles. The central canal of the cord was wider than usual in the cervical region, and there was early sclerosis of one lateral column.

Prognosis.—If the quantity of fluid is not large, and if the tendency for it to go on accumulating has never been very active, it often happens that the morbid action undergoes arrest after a time and that as the child grows older the increased size of the head ceases to be conspicuous. Some years ago, in examining the body of a man, aged forty-three, who had been killed by an accident, I found well-marked hydrocephalus, which I supposed to have been a residue of an attack of the disease in childhood; I could get no information as to the man's mental capacity or attainments. Persons have been known to live under such circumstances to advanced age.

It is, however, believed that cases of this kind have a marked tendency to relapse, and that excitement or overwork is very apt to light up active morbid changes afresh. And some writers have supposed that there is a special risk of their occurrence at the period when the ossification of the skull is finally completed.

Treatment.—The treatment of hydrocephalus by medicines very rarely leads to satisfactory results. As might be expected, the drugs which are chiefly prescribed are those which increase the secretions of the kidneys or of the bowels, and those which are believed to possess the property of accelerating the absorption of the fluid products of inflammation. Small quantities of grey powder or of calomel, pills containing mercury,

squill, and digitalis in doses adapted to the age of the patient, the liquor hydrargyri perchloridi, and the various preparations of iodine are most frequently prescribed. It is doubtful whether benefit is obtained by such remedies.

The frequent presence of rickets in hydrocephalic children would rather suggest the administration of cod-liver oil and of the preparations of iron; with which, however, one may combine digitalis, or the acetate of potass, or the perchloride of mercury, if it should be deemed advisable.

But the mode of treatment which has the largest amount of testimony in its favour consists in the application of pressure to the head. At one time it was usual to cover the whole of the scalp with strips of adhesive plaster. But of late it has been found sufficient to surround the head with a fillet of elastic webbing, two or three inches wide, and of which the length must be carefully adjusted, so as not to cause redness, or to impress the pattern of the material upon the skin. Care must also be taken to shift it from time to time, so that it may not irritate the frontal eminences. The necessity for such precautions is shown by the fact that sloughing of the integuments, leading to the death of the patient, has been known to occur when they have been neglected. Dr Dickinson speaks in very high terms of this procedure, and says that it may be expected to succeed in arresting the disease in the majority of cases, provided that the child is young and that the enlargement of the head is of recent development. One instance which he relates in detail is that of a boy, aged thirteen months, whose skull had been growing out of proportion to the rest of the body for nine months, and measured twenty-two inches round. The eyes were depressed. There were occasional convulsive attacks. An elastic bandage was put on, and it was worn for three years, being renewed as often as was necessary. Diuretics and cod-liver oil were given during the same period. Within four months the circumference of the head became reduced to $20\frac{5}{8}$ inches. At the end of the treatment the bones were fully ossified; and although the head, which was then completely covered with hair, measured $21\frac{1}{2}$ or $21\frac{3}{4}$ inches, its disproportionate size was much less conspicuous. The position of the eyes was natural. There had been no convulsions for eighteen months. The child, at this time more than four years old, was sensible, and could make use of simple words for talking; but he appeared to be somewhat deficient in memory.

Paracentesis of the head with a fine trocar, at the outer angle of the great fontanelle, has been recommended by some physicians, and it has been performed on several occasions at Guy's Hospital. Perhaps the best thing that can be said in its favour is still the fact that in a case recorded many years ago by Mr Greatwood, a hydrocephalic child, having acccidentally fallen upon a nail which penetrated its skull, recovered after three pints of fluid had slowly escaped through the wound. Cases are also on record of recovery after evacuation of the fluid through the nose. Scarcely less successful results have been reported after paracentesis; but the cases in question do not stand criticism.* Only a small quantity of fluid (not more than two or three ounces) should be withdrawn at a time, on account of the danger of setting up convulsions. If the result appears to be good,

* See, for example, Dr West's remarks in 1842 on fifty-six cases of hydrocephalus treated by tapping the skull ('Lectures on Diseases of Children,' p. 130); and Dr Wilks ('Diseases of Nervous System,' p. 175).

one may have recourse to the same procedure again and again, a bandage being applied in the intervals. But such repeated operations are very likely to be followed by acute meningitis, or to set up suppuration within the ventricles. And we are inclined to think that this mode of treatment should not be adopted, unless it be in the hope of warding off death for the time, when it is threatened by epileptiform seizures or by coma. The injection of iodine into the interior of the brain has occasionally been practised, and has not been followed by the serious results that might have been anticipated; but there is no ground for supposing that it is likely to do good. After having several times practised paracentesis, Dr Eustace Smith has seen no decided benefit result, and sometimes decided evil. Dr Goodhart speaks but little more favourably of the operation.

Hydrocephalus of Adult Life. Chronic Meningo-Ependymitis.—Many writers mention the fact that adult patients are sometimes affected with a chronic disease of the brain, in which the most conspicuous anatomical change is the distension of the ventricles with a more or less considerable quantity of fluid. Dean Swift is said to have died of this malady, after an illness of three years' duration, in 1745; but one cannot speak confidently of the accuracy of an observation made so long ago, when scarcely anything was known of pathology, and when no account could have been taken of the possible relations between the effusion and a primary atrophy of the nervous tissues, or a cerebral tumour, or even a chronic affection of the kidneys. Sir Thomas Watson, however, speaks of a young and distinguished lawyer, who, after one or two sudden attacks of unconsciousness, became dull, stupid, and insensible, and at length died; the only alteration that could be detected in the brain was the presence of a large quantity of serous fluid in the ventricles.

I have already remarked that pathologically there is a close correspondence between cases of this kind and those of hydrocephalus occurring in childhood. Similar changes are found in the ependyma of the ventricles; it is tough and thick, and may be granular and feel rough to the touch, or may have an areolated appearance (like that of the capsule of the liver in some cases of perihepatitis). The floor of the third ventricle often forms a protrusion like a bladder of fluid. I once saw the distended descending cornu of the left lateral ventricle projecting in a similar manner when the middle lobe had been removed. The membranes at the base, too, are found greatly thickened and opaque, and matted together; even more so in the affection of adults than in that of children. In the case just referred to the velum interpositum could not be dissected off from the corpora quadrigemina, nor could the pineal body be isolated. There is, of course, a great difference in the quantity of the fluid, which seems not to have exceeded fourteen ounces in any of the cases observed at Guy's Hospital within the last few years. Whenever its characters have been recorded it has been clear, and has contained only a very small quantity of albumen. The cranial bones are generally thin; there is a deficiency of the diploë; the interior of the skull is marked by sharp ridges and projecting points, with sulci between them, which are obviously attributable to the outward pressure of the brain. There is a very marked flattening of the cerebral hemispheres.

Clinically, the relation between the hydrocephalus of adult life and that

of childhood is one of contrast rather than of resemblance. The enlargement of the head, which renders the diagnosis of the disease so easy, is wanting; and in the symptoms which are present there is scarcely anything to distinguish it from other chronic affections of the brain. The only way to give any account of them is by briefly abstracting the notes of some of the more striking among fifteen cases recorded at Guy's Hospital.

1. A man, aged twenty-three, a patient of Dr Wilks, said that he had been well until a year before, when he noticed a numbness in his feet and legs, which gradually extended up to his face. He had kept his bed for three months. A fortnight back he once found himself unable to micturate, so that a catheter had to be used. Within the last weeks he had had two fits, in which he was insensible. On admission there was a slight convergent squint; the pupils were dilated; the optic discs were ill-defined and red, with some plugging of the veins and retinal hæmorrhages. He had numbness of the feet and legs, and of the face about the mouth. He did not chew his food properly; portions of it would fall out of his mouth while he was eating. His mind seemed not to be clear; and his statements about his case varied from day to day. He passed his urine into the bed. After a few days he died suddenly.

2. A man, aged thirty-two, under the care of Dr Pavy, had been obliged to give up work eighteen months before his admission into the hospital, on account of a pain in the head which prevented his sleeping. At that time he was laid up a week or ten days. Nine months ago he lost his speech, became hemiplegic on the right side, and was insensible for three weeks. From that time his memory was imperfect. Eight days before his death he again became affected with partial hemiplegia and inability to speak. He passed his urine and fæces under him. During the last few hours of his life he had a series of fits, which began in the left side of the face, and in which the eyes were turned to the right.

3. A man, aged fifty-seven, a cooper, was taken in on account of a partial right hemiplegia, which he said had come on suddenly four days previously, while he was in the act of striking a piece of iron; he managed, however, to get home afterwards, the distance being half a mile. He did not seem very ill on admission, and looked a strong, healthy man; but he soon afterwards died rather suddenly, having been ill only nine days. It must be mentioned that in addition to an immense distension of the ventricles, with marked roughening of the ependyma, there was in this case softening of the superficial parts of the corpora striata and here granule-masses were discovered with the microscope.

4. A man, aged fifty-five, was admitted for chronic dilatation of the stomach, in order that the treatment with the stomach-pump might be carried out. But he became light-headed, and two days later he was convulsed and fell into a semi-comatose condition, in which he lay groaning and muttering until at the end of a few hours he died. When his cerebral symptoms set in, it was found that he had complained of severe pains in the head three months previously.

5. A boy, aged fifteen, who had two sisters in an asylum, was taken in on account of very obscure symptoms, which at first seemed to point to some chronic disease of the peritoneum. He was anæmic; he vomited occasionally; there was slight fever; some of the superficial glands were swollen; he complained of pains in the head and in the back of the neck.

After a time his mind began to wander; he lay on his side, with his legs drawn up; when questions were put to him, he answered slowly and unwillingly; his eyes were half closed; his pupils were dilated; the temperature was now below normal, being sometimes not above 97°. He died very gradually.

6. A man, aged twenty-two, was admitted under the care of Dr Habershon in 1871 for severe cerebral symptoms, which were supposed to be the result of an accident. He had once fallen from a scaffold, striking the left side of his head; he was insensible for a fortnight, and had bleeding from the mouth, the nose, and the left ear. At the end of three months he resumed his work, but it was noticed that he was strange in manner. For a time he was free from headache. Seven months before his death, however, he was attacked with violent pain in the head and with shivering. He gradually became unable to stand, and passed his urine and fæces under him. While in the hospital, he lay all day in a drowsy condition. He would answer questions, but soon began to ramble in his talk. He was occasionally sick. His pupils were dilated. His head was drawn back. Two months before his death he had a fit; a month later he had another fit. After this he lay perfectly still, saying nothing, and eating no solid food; towards the last he became extremely emaciated. I made the *post-mortem* examination, and found well-marked indications of chronic meningitis, and a greatly dilated state of the ventricles. The anterior and middle lobes of the brain were also adherent to the dura mater at the base, especially on the left side. There was a little ochrey-yellow discolouration, extending into the brain-substance, and no doubt the result of effusion of blood at the time of the injury. The foramen of Magendie at the apex of the fourth ventricle appeared to be closed, so that the case might perhaps be cited in support of Hilton's theory with regard to the origin of hydrocephalus (see 'Rest and Pain,' 3rd edition, p. 39, and Mr Jacobson's note). But it must be added that there was great thickening of the velum interpositum, and that it therefore seemed quite as probable that the chronic ependymitis had arisen by direct extension from the surface. The ventricular fluid contained scarcely any albumen.

7. A man, aged thirty, a patient of Dr Wilks, died in the hospital in 1876, of the effects of disease of the aortic valves, and bronchitis. Towards the last he seems to have had no very marked cerebral symptoms, but when admitted he was comatose, passing urine and fæces under him, and he remained so for several days, before he gradually recovered his consciousness. His insensibility arose out of a succession of fits, which he had been having very frequently. Afterwards, while in the ward, he had one or two of them, affecting the left side. He had a fairly intelligent appearance, and answered questions readily. He stated that eight years back, at which time he was a healthy young man, he had fallen from a ladder, and cut his head. He was brought home insensible, was delirious for two days and was laid up for eleven weeks. Ever after he was unable to do any hard work. His memory failed him; he had headache; the sensibility of the left side of the body gradually became defective. His first fit occurred five years before his admission. Dr Goodhart made the autopsy, and found the lateral ventricles, the third ventricle, and the aqueduct all distended, and forming a large cavity which held fourteen ounces of fluid. There was a repaired fracture at the base of the skull. The prominent parts of the brain on its under surface were discoloured where they had been bruised.

It is clear from these cases, even in the condensed form in which they are here recorded, that the diagnosis between hydrocephalus and other cerebral affections is in the adult exceedingly difficult, if not impossible. In the first of them, the fact that the numb sensations appear to have been bilateral might perhaps have suggested the nature of the disease. But in the second and third cases there was hemiplegia, which seems not to have beem distinguishable from that which a tumour might have caused.

Huguenin has attempted to give a systematic account of the hydrocephalus of adults. But in his article on the subject in 'Ziemssen's Handbuch,' the description of the symptoms by no means corresponds with the observations just cited. He speaks of the affection as bearing the closest resemblance to general paralysis (*dementia paralytica*), and he gives full reports of two cases in which such a resemblance undoubtedly existed, although there was not the *delire des grandeurs* on which so much stress used formerly to be laid in diagnosis. The apparent discrepancy is to be accounted for by the fact that neither of Huguenin's patients died in the wards of a general hospital; one was an inmate of the lunatic asylum at Zürich, the other was nursed at home. His cases, therefore, should be taken as supplementing the above; and they afford additional proof of the variety of aspects that the disease may assume.

In our last two cases the hydrocephalus was distinctly attributable to a severe injury from a fall on the head several months or some years previously. In one of Huguenin's patients a similar origin was no less directly traced, although the accident itself appears to have been a comparatively slight railway collision. It is a very interesting and important question whether the occurrence of serious cerebral symptoms under such circumstances may not generally be taken to indicate that a chronic meningo-ependymitis with effusion into the ventricles is developing itself. There are, indeed, other possibilities to be taken into account. As mentioned at p. 610, a tumour has sometimes been found where the supposition during life had been that the patient's symptoms were of traumatic origin; and it may be that new growths are, in fact, sometimes set up by injuries. It has been commonly believed that an abscess is not unlikely to be met with in such cases. This appears to be very doubtful, unless the patient's fatal illness has gone on without interruption from the date of the accident. But probably there may sometimes be considerable cerebral disturbance, lasting for a long time, and the only lesion discoverable be an ochrey-yellow discolouration from bruising of the under surface of the brain. I once examined the body of a man who died of some other disease, but who was said never to have recovered from the effects of an injury to the head, having been unable ever afterwards to take stimulants even in moderation; and in that instance the brain, the membranes, and the bones, all appeared to be thoroughly healthy.

Huguenin, indeed, alludes to cases in which persons who had had blows or falls on the head, after suffering more or less severely for several months, or even for years, have at length partially or completely recovered. And his opinion is that they depend on a meningitis of limited extent. He remarks that the chief symptoms are headache, giddiness, and other subjective sensations of a distressing character, which often cause the patient to withdraw himself from society. The pupils may be unequal and sluggish. I have a case of this kind under observation in the person of a bank porter, who,

seven years ago, received a blow on the head from the heavy door of an iron safe; ever since he has been liable to pain in the occipital region, vertigo, and a peculiar light feeling in the head, but sometimes he is free from these symptoms for some weeks. The least thing, he says, affects his head, so that he can take scarcely any stimulant. Reading often makes him feel giddy; and he has been obliged to go out of church on account of indescribable sensations of discomfort. Once he complained to me of a numbness in the left side of his head, and at another time of cramp round the neck, as if his collar were too tight. The optic discs are normal.

I have found the bichloride of mercury more useful than any other medicine to this patient; on one occasion it kept off his symptoms entirely for about a year. He has also taken bromide of potassium and the ammoniated tincture of valerian; and a blister has once been applied to the back of the neck. He has several times been obliged to be away from work for two or three weeks; and, no doubt, the rest which he has had has been an important part of the treatment.

In the more severe cases of hydrocephalus, similar measures appear to be the best that can be adopted whenever its presence can be determined. Huguenin advocates the continued application of a bladder of ice to the head, or of a stream of cold water, for weeks or even months together; he also recommends periodical leeching, saline purgatives, and small doses of chloral for the relief of pain. The bromides he has found useful, but not the preparations of mercury or of iodine.

Atrophy of the Brain.—Under this head it is at present necessary to inlcude two groups of cases, which in some of their clinical features have to be sharply contrasted with one another. The first group consists of a somewhat special form of insanity attended with definite paralytic symptoms; it is for the most part studied in asylums, it occurs chiefly in males and at a particular period of life, and there is reason to believe that it is generally traceable to sexual excesses. It differs from all the other forms of insanity in being constantly dependent on an organic change in the brain, and yet the change in question, so far from being peculiar to it, seems to be no less marked in the second group of cases, in which there may be the utmost variety of symptoms or even no symptoms at all, and which includes adult persons of all ages poisoned by alcohol or by lead, as well as old people in whom the atrophy appears to be a mere consequence of advancing years. Lastly, and as if to complete the confusion and to render an accurate description of these affections impossible, the morbid appearances themselves present many differences in individual cases, so that there is scarcely one of them which may not sometimes be wanting.

General Paralysis of the Insane.*—With the exception of a slight reference in the works of Thomas Willis, the celebrated anatomist and physician, who died in 1675, the earliest mention of this disease seems to have been made by another Englishman, John Haslam, in 1798; but afterwards its study was taken up in France, and the first complete account was published by Calmeil in 1826. Of late years it has attracted much attention both in England and in Germany, but even

* *Synonyms.*—Dementia paralytica—Softening of the brain—Folie paralytique—Polyparésie—Délire des grandeurs—Progressive Paralyse der Irren.

Griesinger refuses it an independent position in the nosology, and describes it in a chapter on the complications of insanity. Yet we shall find that its symptoms and course are remarkably definite. It shows little or no tendency to shade off into the other forms of mental disorder; it is not apt to come on secondarily in persons who are already the subjects of chronic insanity, nor does a relapse of mania or of melancholia ever assume its characters. It is, in fact, from the first distinguished more or less plainly by features of its own. The impairment of muscular power, too, is of a peculiar kind. One could not make a greater mistake than to suppose that the mere association of hemiplegia or paraplegia with unsoundness of mind constitutes paralytic insanity. In 'Ziemssen's Handbuch' the article on this disease, which is written by Hitzig, is rightly separated from those on the psychoses proper, and appears among the organic affections of the brain. Perhaps the best name for it is that which he uses, namely, progressive paralysis of the insane, but in this country it is commonly called "General Paralysis of the Insane," or more briefly "General Paralysis." It constitutes a large proportion of the cases which in popular (but not in scientific) language are spoken of under the name of "Softening of the Brain." Its importance may be estimated from the fact that in some lunatic asylums as many as one in four of all the male patients are said to suffer from it.

Ætiology.—The persons most apt to be attacked by general paralysis are married men in the prime of life. The proportion of males to females is variously stated by writers; some give it as eight or even ten to one, others as no higher than two to one. Dr Blandford observes that the women who fall victims to this disease almost all belong to the lower classes, whereas it is commonly seen in men who have been highly educated and who possess powerful frames and handsome faces—men who have enjoyed life and lived hard. According to Mr Austin the subjects of it are generally fair-complexioned and thin-skinned, with blue or grey eyes. The time of life at which it usually occurs is between thirty and fifty years of age, and particularly about the middle of that period. In persons over sixty it is scarcely ever seen, although Mr Austin refers to one instance in a man aged seventy-six. Those who live in towns and cities furnish a far larger proportion of cases than do rural populations.

It is commonly said that general paralysis differs from other forms of insanity in being less frequently due to inheritance or congenital predisposition, and if this statement is correct one would rather expect that it should be traceable with corresponding facility to acquired conditions. But there are considerable differences of opinion with regard to its exciting causes. Dr Blandford is strongly disposed to attribute it in most cases to sexual excesses. He does, indeed, admit that he has not always been able to refer it to such an origin, but then, as he says, in married men there may be great difficulty in ascertaining the fact, and an amount of sexual intercourse which to some would be perfectly harmless may be very prejudicial to others, if they are possessed of less natural vigour or have their energies fully taxed in other ways. Dr Blandford speaks of having seen several cases in men who had formerly been dissipated, or who when no longer young had married wives much younger than themselves. Dr Sankey found that at Hanwell a large proportion of the women affected with general paralysis had led irregular lives. Griesinger is rather disposed to attribute the disease to excessive mental excitement, but, he adds, especially to "emo-

tional agitations." Mr Austin says that it commonly follows a painful moral shock, such as would be caused by bankruptcy, or destitution, or might arise from remorse. Hitzig speaks of it as probably resulting most frequently from the combination of intense work with venereal excesses and indulgence in alcoholic beverages. He and other writers also mention that it sometimes follows injuries to the head, or may occur in those who have before passed through some acute febrile disease.

Symptoms.—The course of general paralysis is commonly said to be divisible into three stages.

During the *first period* an alteration in the character is the most striking symptom. Perhaps the man is extravagant in his expenditure, making presents to persons of whom he knows scarcely anything; or he may be dull and sulky in his demeanour, or depressed and melancholy. So far there is nothing distinctive of this rather than of other forms of insanity; but there is commonly some impairment of memory. Dr Blandford—from whom I take most of the details in regard to the symptoms of the disease—says that such a patient is regardless of appointments, forgetful of the time of meals, of the hour for going to bed, and the like. He comes and goes, scarcely noticing those about him, giving absurd and conflicting orders to his servants, and falling into a passion if they are not instantly executed. He neglects his business and is careless and indifferent to things which formerly interested him. When he takes up a new scheme his interest in it soon flags. He may commit indecent actions, but if he exposes his person he often seems to be half unconscious of what he is doing; or he perhaps commits assaults upon women in a foolish manner, without regard to opportunity, place, or consequences. He sleeps badly, eats irregularly or voraciously, and drinks to excess from inattention and forgetting how much he has already taken. He eats hastily, and is apt to spill his food on his dress. He is neglectful of his appearance, and his costume is often unsuitable for the occasion, or the different parts of it are incongruous with one another. At this time there may be no physical change which can be detected by the eye of the physician, unless, indeed, the pupils are unequal and less mobile than natural, as appears sometimes to be the case.

It is of the utmost importance that the distinctive characters of incipient general paralysis should be well known to the physician and the general practitioner, since they alone are likely to see the patient at this period of his disease. The early stage is not of long duration; after a few weeks, or at the end of a month or two, further symptoms develop themselves, and the patient becomes manifestly insane (*second stage*). The delusions which he now exhibits are in certain respects peculiar. They are almost always connected with ideas of colossal size, or magnificent wealth, or extravagant numbers. He may say that he can walk 100 leagues in a day, or write 100 tragedies and 1000 poems in the same space of time; or that he is going to make his fortune by buying up all the joint-stock banks; or that he is about to marry the Queen and all the princesses. French writers give to this form of insanity the name of *monomanie des grandeurs*, but it is to be noted that these "large delusions' are not maintained from day to day, nor are they consistent with one another. In fact, in spite of his eager excitement, the patient's mental condition continues to be one of progressive decay. He is extraordinarily self-satisfied, full of ideas of greatness, importance, and riches, but he takes no notice of the fact that the palace in which he

believes himself to reside is really a lunatic asylum, and that the great men whom he supposes to surround him are mad people like himself. Thus he in reality cares much less about being placed under restraint than most other insane patients. In some instances, indeed, the delusions are not all of such a kind. Dr Blandford mentions that one of his patients thought he was going to be arrested, that people were about to injure him, that they were maligning, and would rob him. Yet although his symptoms so far resembled those of melancholia, this man was often cheerful and talkative; he was very vain of his appearance, and exceedingly fond of his food. Dr Blandford also notes, as instances of ordinary delusions, that one patient believed himself given over to the devil, another thought that poison was put into his food, and a third that he had committed sins too enormous to be forgiven.

Though cases of general paralysis commonly begin with maniacal excitement and with ideas of an extravagant nature, a certain number begin with hypochondriacal ideas. It is common to meet with patients of the age and with the history common in general paralysis with notions, for instance, that their bowels are closed; in some there is an idea that the body is wasted or has become very small, so that the term "micromania" has been invented to describe the feeling as contrasted with "megalomania," the feeling of greatness and power. Beside melancholic symptoms we may have any form of mental disorder accompanying general paralysis, and a good practical aid to diagnosis is the fact, that however depressed such patients may be in the earlier stages of the disease, they become fat and weak in mind in the second stage, even though they may still talk of their miseries. There is inconsistency between their bodily and mental states.

This second stage of general paralysis is further characterised by certain physical symptoms, which must be carefully looked for. The earliest of them is commonly a defect of articulation, an indistinctness or *thickness of speech*, or a hesitation in the middle of a sentence, or a tendency to substitute for the proper word another of which some letters are the same. The tongue, when protruded, is *tremulous*, that is, it not merely shows a fibrillary tremor—which is often observed in persons who are in perfect health, if they are nervous or anxious—but it oscillates irregularly to and fro, and cannot be held quiet. Sometimes the lips are seen to quiver, as they do in persons about to burst into tears. Mr Austin also says that the mouth is generally closed, that the lips are compressed, and that the upper one is straight, its natural curves being obliterated. The *pupils* are almost always unequal, and they are sluggish. In some cases the *gait* becomes altered, the legs are not lifted properly, the patient walks stiffly, or straddles, or shambles along, and stumbles over any obstacle in his path. The movements of the hands and arms may also be impaired; Griesinger speaks of them as being "stiff;" objects are grasped convulsively, and afterwards suddenly allowed to fall. Dr Mackenzie Bacon has pointed out that an important indication of the disease at this stage is an alteration in the character of the *handwriting;* it becomes tremulous and uncertain; sometimes, too, words are omitted from the sentences which the patient writes, or the same sentence may be written over and over again; or the whole may be an incoherent jumble. All these, however, are indications of mental rather than of bodily failure.

In almost all cases one or more "fits" occur during this period of the disease. These may be of various kinds; the patient may become pro-

foundly comatose, with complete paralysis and anæsthesia, and abolition of reflex movements, and in such an attack he may die. Or he may be less deeply insensible, with loss of power affecting one side only. The paralysis is often very transitory, disappearing in a few hours. Mr Austin was impressed with the belief that seizures of this kind were often direct results of the accumulation of scybala in the large intestine, and that the administration of purgatives and enemata was very efficacious in removing the hemiplegia. Or the fits may be more or less distinctly epileptiform, and these, again, are often directly fatal, as in the case of a man, aged thirty-seven, who died in Guy's Hospital in 1855. Lastly, the patient may be attacked with convulsions alone without becoming insensible, or even falling to the ground.

Persons affected with general paralysis in its second stage are also exceedingly liable to paroxysms of rage and fury, surpassing in violence those that occur in any other form of insanity, exeept, indeed, those which are believed to bear a more or less close relation to epilepsy. Even in an asylum such patients cause unusual anxiety and trouble to those responsible for their safety, and all writers are agreed that it is scarcely ever justifiable to allow them to remain in the hands of friends or relations at this period of the disease. Dr Blandford remarks that these are the persons who sometimes get their ribs broken by attendants before they can be mastered. They may make the most desperate efforts to escape; or they may tear up their bedding and clothes and go about naked. They are often horribly filthy in their habits.

But after a variable period, a week, or a month, or a longer time, these violent symptoms commonly pass off. The patient's condition may even improve so much that he is able to leave the asylum. The disease is then sometimes said to be cured, but all writers are agreed that permanent complete recovery is the rarest possible event, and almost all of them deny that it ever occurs. Dr Blandford says that he has known some persons affected with general paralysis who were able to live with their families, to spend their money without extravagance, and to write long letters without mistakes, detailing their travels and amusements; but he adds that he has not met with one case in which the patient was capable of work or business. The friends and relations of such persons notice a childishness and slowness of intelligence in them, and if they attempt to resume their former occupations they break down and have again to be placed in confinement. The only exception to this rule that I have met with in my reading is a case related by Schüle ('Allg. Zeitschrift f. Psychiatrie,' xxxii). There are, indeed, many cases in which no improvement takes place.

In the *third stage*, the patient's mental condition gradually passes into one of dementia, his bodily state into one of complete paralysis. His notions of magnificence may continue a little longer, but his understanding and memory soon become altogether destroyed. His speech cannot now be understood; his power of swallowing is very greatly impaired. His hands tremble so that he can scarcely hold anything in them; and it is as much as he can do to shuffle about the garden with the aid of an attendant. Sometimes he sits and grinds his teeth for hours together, making a horrible noise.

This state of affairs leads up to a fatal termination which is very seldom long delayed. Writers are not entirely agreed as to the average duration of the disease, but all say that it is short. According to Hitzig the majority

of patients die in from fifteen to thirty months after their admission into an asylum. Calmeil and Griesinger speak of the ordinary course of general paralysis as coming to an end within a period of from several months to three years. But the last-named writer alludes to exceptional cases, in which life was sustained for as long as ten years; and Dr Blandford mentions particularly the case of a baronet of large fortune, on whom a commission of lunacy was held in 1858, when he was suffering from this form of insanity, and who was alive in 1870. This observer says that in his experience the average duration of the disease has been considerably longer than that stated by Griesinger, the reason being that his patients have all been in a position to have the best food and nursing.

Indeed, the immediate cause of death is commonly something which may almost be termed accidental. One way in which it is often brought about is by choking; the patient goes on filling his mouth without swallowing the food, until it gets into the larynx and trachea, or at least fills up the pharynx so as to obstruct the entrance of air. Persons suffering under general paralysis should always have their meat very finely minced, and an attendant should be present at meal-times. Another way in which death occurs is by the supervention of a low bronchitis or pneumonia; if overlooked at its commencement this may destroy life in a few hours. I know of one case in which the patient, a nobleman, had been for his usual drive in the afternoon, but in the evening was noticed to be out of sorts; on auscultation it was found that he had pneumonia, and next morning he died. Dr Blandford remarks that persons affected with general paralysis are always more feeble in cold weather; during the heat of summer they may regain strength to a surprising extent, but with the first frosts they fall back, and are apt to sink. Bedsores sometimes seem to bring about the fatal termination.

Anatomy.—I have already more than once insisted on the fact that general paralysis of the insane differs from the psychoses in being constantly attended with morbid changes in the nervous centres. This, indeed, is admitted by all observers, but unfortunately, when the question is that of defining accurately their nature, and of distinguishing them from those which occur under other conditions, there is by no means the same agreement. Some writers have laid much stress on the fact that the pia mater is often abnormally adherent to the convolutions, so that the cortical substance becomes torn and assumes a ragged appearance when one attempts to strip off the membrane. But in other cases there is an excess of fluid beneath the arachnoid, and the pia mater can then be removed even more easily than from the healthy brain. Thickening of the arachnoid has now and then been noted; pachymeningitis and hæmatoma of the dura mater, and increased density and thickness of the calvaria. The absolute weight of the brain has been said to be diminished; the ventricles are described as being of unusual size, and their ependyma as being abnormally thick; the centrum ovale is said to be peculiarly flaccid when exposed by the knife.

Some of the earlier observers, including Calmeil, described the cortical substance of the brain as of a peculiar violet-red colour; Westphal admits that it sometimes presents this appearance, but he says that in other cases it looks remarkably pale and faded. And, as he remarks, these variations probably depend simply on the mode of death.

Again, the ganglionic cells of the brain have been found affected with pigmentary or fatty degeneration. Hitzig says that this change is almost

always present in a very large number of the cells ; but he goes on to quote Westphal as having stated that similar appearances are observed in other cerebral diseases, and even under normal conditions, so that he is obliged to add that it is only the extent of the degeneration that can be regarded as peculiar to general paralysis. Less commonly, he says, cells are seen which are swollen out and sclerosed ; this seems to be in accordance with an observation of Dr Major's, who in one case ('West Riding Asylum Rep.,' vols. iii and iv) discovered what he describes as a hypertrophy of certain cells, these being abnormally large and furnished with an excessive number of branches. Some have thought that the neuroglia is increased both in the grey and in the white substance. In the latter, patches of degeneration have been observed, of the kind termed miliary sclerosis.

In one case Dr Lockhart Clarke found the white substance of the convolutions full of little cavities, of round, oval, fusiform or crescentic shape, and varying in size from that of a small pea or barleycorn to that of a grain of sand, so that the cut surface looked like Gruyère cheese, or crumb of bread. These vacuoles were doubtless analogous to those which have been described by Dr Dickinson in other diseases. Most of them are empty, but some contained the remains of vessels mixed with granules of hæmatoidin. Probably they all were originally perivascular canals.* Changes in the blood-vessels have been described by some observers as important features in the morbid histology of general paralysis. The small arteries become dilated and tortuous, aud present twists or kinks in their course. Their nuclei and those in the walls of the capillaries are found to have undergone proliferation. Granules of hæmatoidin may be scattered upon their exterior, and they may be affected with fatty degeneration or calcification.

Moreover, other parts of the nervous system, as well as the brain, present morbid changes. The state of the spinal cord has been especially investigated by Westphal, and he has found that its posterior and lateral columns are often extensively altered. In the former the appearances are generally those of grey degeneration, exactly like that which constantly occurs in locomotor ataxy. We shall presently have to discuss the relations between the two diseases. In the lateral columns the appearances are rather those of chronic myelitis. It is not to be supposed that such changes arise by the extension downwards of a morbid process which had commenced in the encephalon, for Westphal could not trace the affection of the posterior columns above the commencement of the fourth ventricle, nor that of the lateral columns beyond the lower end of the crura cerebri. They must, therefore, be regarded as of independent origin. Indeed, it would appear that even the peripheral nerves fail to retain their normal structure. At least, the sciatic nerves are said by Dr Lewis ('West Riding Reports,' vol. v) to be smaller and softer than natural, and less rounded in form. And under the microscope he was able to detect atrophy of the nerve-tubules, with overgrowth of the connective-tissue elements. Again, Bonnet and Poincaré have pointed out that the great sympathetic ganglia, especially in the cervical region, constantly present morbid changes. The nerve-cells in them are sclerosed and pigmented ; they may even undergo destruction to a considerable extent, their place being taken by adipose and connective-tissue cells. The discoverers of these appearances believe that they constitute the

* See some cases of this remarkable condition of the brain described by Drs Savage and Hale White, in the 'Pathological Transactions' (vol. xxxiv, 1883).

starting-point of the disease, and that all its phenomena depend upon the resulting vaso-motor disturbances. But it seems most unlikely for this conclusion to be correct. The changes in question rather afford a further illustration of the fact that in general paralysis the nervous structures throughout the body simultaneously undergo degeneration.

Diagnosis.—This must be considered from two points of view. In some cases it is a question between general paralysis and the psychoses; in other cases between it and the various forms of paralysis dependent upon chronic alcoholism, brain-wasting, or other diffused changes in the nervous centres. It is not generally difficult to say whether a patient is suffering from general paralysis or from mania, even at an early stage. But mistakes have sometimes been committed, from its being supposed that the presence of large delusions is characteristic of the former disease. Thus Dr Blandford mentions the case of a gentleman who wanted to make a tunnel through the earth to the antipodes, and who thought that people might live a thousand years if they would bathe in beef-tea and beer, and that he should be able to pay off all the mortgages on his estate by assembling 10,000 persons in his park, having them photographed, and selling the photographs at five pounds apiece. But it was clear that he was not labouring under general paralysis, for he did not stutter, his memory was perfect, and (above all) he had had a similar attack some years before. In cases of this kind a correct diagnosis is of the greatest importance, because the prognosis depends upon it. The opinion was given that the case was one of mania and that it might pass off; and the sequel proved the correctness of this judgment.

The exceptional cases in which general paralysis occurs in persons at an advanced age may be very difficult to distinguish from those of senile dementia, unless, indeed, the more characteristic symptoms of the former disease are well pronounced. The childish nature of the delusions in those who are suffering from senile dementia commonly lends aid to the diagnosis; that complaint is slower and more uniform in its course; it is less apt to be complicated with epileptiform fits, and any apoplectic attacks that may occur are much more likely to be followed by permanent hemiplegia.

Chronic alcoholism sometimes gives rise to a chain of symptoms so like those of the less marked forms of general paralysis that it may be impossible to make a diagnosis between the two diseases until one has watched the case for a little time. Dr Blandford says that the muscular weakness does not in alcoholism extend to the muscles of articulation, but this is inconsistent with the statements of most other authorities.

Lastly, there may be a difficulty in distinguishing general paralysis from some affections of the spinal cord. There are cases in which an impairment of muscular power shows itself some months before any psychical symptoms develop themselves. It has even been a question whether the disease may not sometimes run its course without being attended at any time with impairment of the mental powers. This question is answered in the negative by alienist physicians; but one may fairly reply that those observers would have no chance of seeing such cases, supposing them to occur. It is in the wards of a general hospital that they must be looked for. But, so far as I am aware, there is no such case on record. Dr Wilks has published in the 'Guy's Hospital Reports' (xvi, p. 194) at least one instance in which the patient's mind was unaffected while he was under his care, the paralytic symptoms being well marked. But he had only been ill nine or ten months,

and I think that in all probability the mental symptoms afterwards developed themselves.

There is sometimes a special difficulty in distinguishing general paralysis in its early stage from tabes dorsalis. Dr Wilks relates that two physicians—one an authority with regard to the former disease, the other with regard to the latter—had the same case shown to them, and that each of them pronounced it to be an example of the malady with which he was the more familiar. The explanation seems to lie in the fact that the two diseases are not infrequently present in the same individual at the same time. I have already quoted Westphal's observations as to the occurrence of grey degeneration of the posterior columns of the cord in cases of general paralysis; and that writer expressly states that they had presented the ordinary symptoms of locomotor ataxy. As far back as 1862 Baillarger published clinical reports of five cases of a similar kind.

Dr Bristowe has published an important lecture on the occasional difficulties of diagnosis between General Paralysis and Insular Sclerosis ('Brit. Med. Journ.,' Jan. 1, 1887).

Treatment.—With regard to this there is unfortunately not very much to be said. I have already stated that in the early stage of the disease confinement in an asylum is absolutely necessary. It is, indeed, a question whether it might not sometimes run a more favourable course if the patient were secluded at an earlier period than is usually the case. His friends and relations are but too apt to insist on his having amusement, to take him to the seaside, or even to make him travel about from place to place; whereas what his brain really needs is absolute rest. Among medicinal agents Dr Blandford speaks very highly of tincture of digitalis in doses of ℥xv to ℥xxx every four hours. He says that it often soothes such patients wonderfully and restores them from a state of noisy turbulence to one of comparative rationality. According to Hitzig the application of galvanism to the medulla oblongata is sometimes of marked temporary utility; he also speaks favourably of iodide of potassium. The bichloride of mercury has been largely used, but without any good result. Dr Blandford lays stress upon the importance of withholding stimulants, such as brandy, while there is excitement; but he says that opium and morphia and chloral are often useful both in the early, and still more in the later stages of the disease. The same writer insists on the value of tonics when the more acute symptoms have passed off, the tinctura ferri perchloridi and the other preparations of iron being particularly serviceable. Dr Crichton Browne published two cases in which the extract of Calabar bean in doses of a quarter to a third of a grain, continued for nine or twelve months, appeared to cure the disease, although it was in an advanced stage ('Journ. of Mental Science,' 1875).

Other forms of atrophy of the brain.—In sharp contrast with the definite clinical course of General Paralysis, which disease is chiefly seen in asylums for the insane, there occurs in ordinary medical practice a precisely similar atrophy of the brain, attended with the greatest possible variety of symptoms, or even with no symptoms at all. This affection has been very generally ignored, although Dr Wilks published some observations in reference to it in the 'Journal of Mental Science' for 1864. But that it is not very infrequent is evident from notes of no less than fifty cases in which it has been found in the *post-mortem* room of Guy's Hospital within the last

twenty years; and that number might be considerably augmented, for only the more striking examples were selected.

The wasting of the cerebral substance is characterised, not only by loss of weight, but also by an obvious shrinking. When the dura mater is turned aside, after the calvaria has been removed, the surface of the hemispheres may appear to be covered with a gelatinous substance, which, however, is really an accumulation of serous fluid in the meshes of the pia mater, and runs out as soon as the arachnoid is punctured, leaving the membranes collapsed and wrinkled. The convolutions are small, and they are separated by deep, broad sulci; the ventricles may be wide; the ependyma is often granular; the choroid plexuses have undergone more or less of cystic degeneration. The cerebral substance itself is in some cases firm and natural looking. But in other instances it presents patches of softening, either thickly scattered throughout the white matter beneath the convolutions or limited to the basal ganglia. In an old man of seventy-six, whose body was examined in 1876, the cut surface of the hemispheres showed smooth-walled cavities round the vessels, especially in the neighbourhood of the grey matter, so that it had an appearance like that of Gruyère cheese—exactly such as was described by Lockhart Clarke in cases of general paralysis (p. 668). When morbid changes of this kind are present, the minute arteries are, as a rule, diseased. Not unfrequently they are so thickened and even calcified, that their cut ends stick up as if they were so many bristles embedded in the brain. The membranes, too, are often thickened and opaque, especially over the hemispheres; and the ependyma of the ventricles is granular. It would be difficult to deny that these last appearances are indicative of chronic inflammatory change, rather than of mere atrophy; but they certainly occur in cases in which there is no other evidence of inflammation.

The causes of atrophy of the brain vary in different cases. Sometimes it seems to be a *senile change*, and it may then give rise to no obvious symptoms; the old man whose hemispheres resembled Gruyère cheese was said to have been perfectly clear in his intellect. In other persons of advanced years, however, there is a more or less marked failure of both mind and body. They are eccentric or odd in manner, or even demented. Of one patient, a woman, aged sixty-eight, who was brought to the hospital for a fractured thigh, it is noted that she got out of bed, splint and all, and tried to walk about, with the result that the limb became gangrenous. Another, a man, aged seventy-four, who was taken in for bronchitis, showed no special cerebral symptoms, except that he passed his evacuations under him. Others again have been admitted for injuries due to attempts at suicide: a woman had thrown herself out of the window and fractured her spine, and a man had cut his throat. Probably many cases of this kind are undistinguishable from those of ordinary insanity, in which the brain remains perfect, so far as our means of investigation enable us to determine.

The affection is not confined to old people; cases occur in persons between forty and fifty years of age. Thus in 1867 there died in Guy's Hospital a man, aged forty-nine, who had been an accountant, but who for some time had been unable to do his work. His memory had failed him, his speech had become uncertain, he wrote badly, and he spelt his entries in a cheque-book all wrong. Afterwards, however, his writing was correct, but he formed his words very slowly. When speaking he would lose the thread of his discourse, and would vainly clasp his head with his

hands in search of what he wanted to say. For two or three days before his death he lay in a doze. At the autopsy patches of brown discolouration, breaking down in their centres, were found in each corpus striatum; the cerebral arteries were very rigid and calcareous, and the kidneys were granular.

In some cases of this kind the disease advances to a fatal termination within a few months of its commencement; its diagnosis from other organic affections of the brain may then be impossible. There is often more or less complete paralysis of some or all of the limbs; sometimes convulsive movements or jactitations are present; sometimes epileptiform seizures occur. Headache appears not to be generally a marked symptom; but in one patient it was exceedingly severe for the last six years of his life, and continuous for three months at a time.

Cerebral atrophy is often the result of chronic *alcoholic poisoning*. A large proportion of those who die of delirium tremens have wasted brains; but that disease cannot be described as being merely an effect of the morbid change in question, since in some cases the brain is found to be perfectly healthy.

Yet another cause of cerebral atrophy is *poisoning by lead*. In 1863 a compositor, aged thirty-four, died in Guy's Hospital who had been admitted for colic, and who afterwards became almost totally paralysed; the convolutions of the brain were shrunken, the sulci were deep and contained an excess of fluid, the ventricles were unduly large. Chronic plumbism, as is well known, sometimes gives rise to epileptiform convulsions, which may be repeated at intervals, and followed by delirium and by a fatal coma. In cases of this kind the brain is commonly found wasted.

In a patient of the editor's, however, a woman, aged thirty-five, who died after eclampsia, obstinate vomiting, and other symptoms ascribed to chronic lead-poisoning, the brain and membranes after death were found perfectly normal, and weighing 42 ounces. No trace of lead could be found here, although it was present abundantly in the liver, spleen, and intestines. The patient had worked in a white lead factory and showed the characteristic blue line, colic, and dropped wrist.

INFLAMMATION OF THE MEMBRANES OF THE BRAIN

TUBERCULAR MENINGITIS—*History—Morbid anatomy—Relation of the tubercles to the inflammation—Their occasional absence in basal meningitis—Ætiology—Onset—Course—Later and final stages—Tubercular meningitis in adults—Diagnosis—Prognosis.*

EPIDEMIC MENINGITIS—*History—Morbid Anatomy—Symptoms and course—Varieties, complications, and sequelæ—Ætiology—Diagnosis.*

ACUTE NON-TUBERCULAR MENINGITIS—*Anatomy—Varied causes—Secondary and idiopathic—Symptoms—Diagnosis—Prognosis.*

Treatment of meningitis generally.

Adhesive thrombosis of cerebral sinuses.—Hæmatoma of dura mater.

Among the most frequent and the most fatal of all cerebral diseases are those which depend on inflammation of the membranes of the brain. Their study has been greatly simplified of late years. Until recently, it was usual to describe separately "acute inflammation of the dura mater," "arachnitis," and "meningitis" seated in the pia mater. Now it is true that various diseases of the bones of the skull—such as, for instance, caries of the temporal—may give rise to ulceration or sloughing of the corresponding part of the dura mater. But that local affection requires no special notice; it is only preliminary to the development of a diffused inflammation of the other membranes or of an abscess within the brain itself; and upon these conditions all the clinical and pathological interest of the case is centred. Again, there is not any disease that can be said to be strictly "arachnitis." Pus or purulent lymph is sometimes found in large quantity within the arachnoid cavity; and its presence or absence in that space constitutes one of the distinctions between two different forms of meningitis. But the limitation of suppuration to a supposed serous sac, without implication of the pia mater, is not, I believe, known even to the surgeon, as a result of any kind of injury to the skull; and certainly the physician never meets with such an affection. If the arachnoid were perfectly analogous with the pleura or the peritoneum, we should doubtless expect it to be liable to inflammatory processes attended with the exudation of lymph and serum, and resembling those that occur so commonly in the other great serous cavities. But the modern anatomical doctrine is that no such analogy exists; the "parietal layer" is now regarded as the endothelial lining of the dura mater; and the "visceral layer" as nothing but an outermost condensed stratum of the pia mater. The subdural space—as the supposed arachnoid cavity is now called—is believed to be continued outwards along the optic nerve, just like that which is termed the sub-arachnoid or "subpial" space, whereas formerly it was naturally supposed to be cut off by a reflexion of the arachnoid membrane.

Thus, the diseases now to be described are, all of them, forms of *menin-*

gitis, inflammation of the pia mater, or leptomeningitis. They all are seated mainly in the loose tissue of the pia mater, over which they diffuse themselves more or less widely; but they differ much in their intensity, and still more in their causes. One of them is attended with the development of tubercles; another occurs epidemically, and ought in strictness to be placed with the specific fevers rather than with local lesions of the brain; a third is made up of a group of affections themselves referable to a variety of conditions. The most common of all these disorders, at least in this country, is "tubercular meningitis;" it is, indeed, absolutely the most frequent of all organic diseases of the nervous centres. I shall therefore describe it first; and afterwards I shall take up, in succession, the "epidemic" and that which may be termed the "simple" form of meningitis.

TUBERCULAR MENINGITIS.*—It is a curious circumstance that although this disease has been known for more than a century, its real pathology has been recognised only during the last fifty years. The earliest detailed account of its symptoms was published in 1768 by Dr Robert Whytt (a man of great eminence in his day in Edinburgh) under the name of dropsy of the brain; and in the early part of the present century it became generally known as "acute hydrocephalus"—a designation which was retained by Sir Thomas Watson as recently as 1857 as being "so established, both among medical men and with the public, that he could not venture to propose any change." He was, however, well aware of the fact, first pointed out by Papavoine in 1830, that the essential morbid change is in reality the presence of tubercles in the membranes, and that the fluid within the ventricles is of quite subordinate importance. And as inflammatory products also are generally found in large quantity, the name of tubercular meningitis, originally suggested by Brechiteau, in 1814, is obviously appropriate, and has now met with universal acceptance.

Morbid anatomy.—The appearances found after death vary considerably in different cases, and in different parts of the brain. As a rule, the most marked lesions are found at the base. The diamond-shaped space bounded by the optic tracts and the crura cerebri is filled with gelatinous or puriform lymph, and a similar material envelops the great arteries arising from the circle of Willis and their branches. The inflammatory process may extend along the Sylvian fissures until it occupies a great part of the convex surface of the hemisphere. Generally, however, the membranes covering the convolutions show no obvious changes; their surface may be dry and the sulci reduced in size; but these are merely effects of pressure. A large quantity of lymph is often spread out over the pons and bulb, and it may be traceable in the subarachnoid space as far as the lower end of the spinal cord. Almost invariably there is a yellowish patch upon the upper surface of the cerebellum, close to the opening of the veins of Galen; and sometimes the velum interpositum and choroid plexuses are infiltrated and thickened. The fluid within the ventricles is increased in quantity and is generally rather turbid. Its specific gravity may be raised to 1010; it yields a more or less distinct coagulum on boiling, and exhibits leucocytes under the microscope. The ependyma is often granular. The adjacent parts of the brain are usually much softened. The fornix and septum lucidum may be almost diffluent, and even the great basal ganglia may have their consistence so

* *Synonyms.*—Acute Hydrocephalus—Basal meningitis—Idiopathic infantile cerebro-meningitis—Leptomeningitis tuberculosa.

much reduced by this white softening that they fall into a shapeless pulp as soon as their removal from the skull deprives them of support.

These changes, however, are common to other forms of meningitis as well as that now under consideration. What is special to the latter is the presence of more or less numerous tubercles. If there are but few of them they are most easily recognised in the Sylvian fissures or in the folds of pia mater dipping into the sulci; they then appear as minute grey dots, adherent to the smaller arteries or to delicate filaments of connective tissue. When they are abundant, they may become fused together so as to ensheath an artery in a granular mass. Some are generally found caseating, and their opaque yellowish colour enables them to be more easily recognised. Cheesy aggregations of considerable size may be found embedded in puriform lymph. If the tubercles are present in very large numbers, they may be seen thickly scattered as milk-white spots beneath the visceral arachnoid, especially on the under surfaces of the cerebral lobes. They may also be developed in the form of very minute transparent granules upon the inner aspect of the dura mater lining the fossæ of the skull; and (according to Huguenin) between the two layers of that membrane, close to small twigs of the middle meningeal artery. In the vertebral canal they are found both on the smooth side of the spinal dura mater and on the visceral arachnoid. Within the ventricles I believe that they occur only in the choroid plexuses. They sometimes grow from the summits of the processes of the pia mater which pass down between the convolutions, appearing as whitish-yellow streaks along the small arterial branches within the cineritious substance of the cortex. Dr Gee mentions a case in which the capillary vessels throughout the whole of one hemisphere were everywhere studded with miliary tubercles, which remained after the softened cerebral substance had been all washed away.

In one instance I found the right hemisphere affected on its convexity with meningitis which appeared to be clearly associated with the presence of tubercles, but the left side of the brain and its base seemed to be entirely free. Huguenin describes two similar cases, in each of which the affection was limited to the territory of one of the Sylvian arteries.

The meninges covering the bulb and cord are often affected as well as those of the brain.

Pathology.—The histological characters of tubercles have already been given at p. 81; and in discussing the theory of acute tuberculosis I have advanced the reasons which lead me to think that the disease is not the result of any infective process, nor due to the absorption of matter from caseous foci, as is believed by so many modern pathologists.

Even if one leaves out of consideration the scrofulous glands and similar affections, to which those observers attach so much importance, one still finds that tubercular meningitis scarcely ever occurs as an independent malady. In almost every instance it is either secondary to some chronic tubercular disease, for which the patient is already under treatment or else it is (as Dr Gee says) "merely a fragment" of a general acute tuberculosis. Not rarely, it is both consecutive to another chronic lesion and associated with the simultaneous development of recent tubercles throughout the body generally.

Of one hundred and twenty-four cases of tubercular meningitis that have occurred in succession at Guy's Hospital since 1856, sixty-five were in persons under the age of twenty; fifty-nine in persons between

the ages of twenty-one and sixty. Among the former there were thirteen in which the cerebral affection was secondary to hip-joint disease or spinal disease, or some other malady capable of clinical recognition; among the latter there were twenty-eight such cases, mostly of pulmonary phthisis. In eighty-six of the one hundred and twenty-four cases there were miliary tubercles in the viscera or in the serous membranes, sometimes, indeed, in very small numbers, but often in such abundance that the lungs or other organs were said to be "stuffed" with them. On the other hand, there are a good many instances in which the brain alone was examined, or in which the state of other parts is not mentioned. In only nine or ten of the whole number is it distinctly stated that recent tubercles were present in the pia mater and nowhere else.

Another question—which is of more importance in reference to tubercular meningitis than to any other form of acute tuberculosis—concerns the relation of the tubercles to the lymph and other products of common inflammation that are so constantly found associated with them. The opinion expressed by Huguenin (in 'Ziemssen's Cyclopædia') is that the miliary granulations are first developed, and that the pia mater tolerates their presence for a time, but that they afterwards excite a process of reaction which passes on into inflammation. I believe that Rilliet was the first to propound this doctrine, and he went so far as to say that the so-called prodroma of the disease were effects of the actual "deposition" of the tubercular material. But, as Wilks and Moxon have pointed out, if such a view were correct, one ought sometimes to discover meningeal tubercles in small quantities, unmixed with inflammatory products, in those cases in which acute tuberculosis destroys life by invading the lungs or other organs; and to find it unattended with the characteristic cerebral symptoms. In this, they say, they have always failed, although they carefully examined the membranes in many cases. Moreover, inflammatory changes are always present where miliary tubercles are found in large numbers in the lungs; but it is certain that the development of the tubercles themselves is in such cases the direct cause of the patient's illness and death. On the other hand, one sometimes finds tubercles in the pia mater unattended with the exudation of lymph or pus in any appreciable amount. But I believe that in such cases symptoms resembling those of tubercular meningitis have always been observed during life.

I have met with two instances of this kind. One occurred in a woman, aged thirty-two, who was admitted under the obstetric physician for ulceration of the os uteri and for an abdominal tumour that turned out to be the omentum indurated by tuberculous matter. She was attacked by hemiplegia, coma, ptosis, and delirium, and died in seven days. In making the autopsy, I found that the brain and its membranes looked healthy, except for the presence of a single minute granule on a fold dipping into one of the sulci, and for that of a little filmy material round one Sylvian artery which was absent on the opposite side. So slight was this change, that if it had been observable on both sides I should certainly have passed it by as unworthy of further investigation. But the microscope showed that even the apparently healthy artery which I had set apart for the sake of comparison had a distinct growth of tubercle about it; and the one little white grain in the pia mater was actually caseating in the centre, its periphery being made up of lymphoid tissue. The case shows that by minute investi-

gation it is sometimes possible to trace the cause of fatal cerebral symptoms to a lesion that might readily be overlooked.

The other instance is that of a man, aged fifty, who became suddenly unconscious on May 2nd, 1876, and was brought to the hospital in a state of coma, with stertor and right hemiplegia. He afterwards partially recovered, and on May 5th he was sensible enough to answer questions that were put to him; but in the following night he was attacked with another fit, of which he died in twelve hours. I found tubercles thickly scattered about the Sylvian arteries and in the adjacent parts of the pia mater, but without any lymph being present; both lungs contained many grey clusters. In that case, indeed, it is probable that the tubercles had been formed more slowly than usual, for it was stated that a fit had occurred as far back as twenty-two days before the man's death, and that afterwards he was always drowsy and stupid. It ought, perhaps, to be added that he was a gouty subject, and had Bright's disease of the kidneys, so that one was obliged to take into consideration the question of uræmia; but I came to the conclusion that the meningeal tubercles were the cause of his convulsive seizures, believing that tubercles are never found in the subarachnoid tissues without having given rise to some cerebral symptoms.

About a year later this very point was raised in a trial for murder. Harriet Staunton, the wife of one of the prisoners, had died in a state of neglect and emaciation, which led to suspicions that she had been starved or poisoned. The medical men who made the *post-mortem* examination expressed it as their opinion that death had been due to deprivation of food, and yet stated that they had discovered in the membranes of the brain bodies which they believed to be tubercles, but to which they attached no importance. What were the exact appearances must remain uncertain, for one report spoke of "small patches of rough, millet-seed like deposit in the meshes of the pia mater," and another of "a small, recent patch of tubercular deposit upon the arachnoid membrane on the upper part of one hemisphere, about the size of a fourpenny piece." The husband and three other persons were convicted of murder. But before the time fixed for their execution the leading pathologists of London addressed a memorial to the Home Secretary and the lives of the prisoners were spared. If there really were tubercles in the meninges, their presence ought certainly to have been regarded as an indication that the immediate cause of the woman's death was disease and not poison.

The reader may perhaps be disposed to doubt whether tubercles that are only discovered by the aid of the microscope really possess the importance attributed to them in the last paragraph; but, just as in a case of concussion of the brain the presence of obvious ecchymoses of one or two convolutions (which cannot be regarded as themselves the cause of the fatal symptoms) is nevertheless of the highest significance as showing that the injury was sufficiently severe to have produced extensive though invisible damage to the texture of the organ—so it would seem that the development of even the smallest tubercles in the pia mater is attended with changes in the cerebral tissues that are incompatible with the maintenance of life, although our knowledge of those changes themselves is as yet very imperfect. Rindfleisch speaks of the superficial layer of the cortex—patches of which often remain sticking to the pia mater when it is stripped off—as infiltrated with leucocytes, and Huguenin says that inflammatory products may even be found in the white substance of the hemisphere. Indeed, some one part of

the brain is now and then found in a state of well-marked yellow or red softening. I have notes of five instances of this that have occurred at Guy's Hospital, and others are mentioned by Rindfleisch and Huguenin. Sometimes the softened part has been the island of Reil on one side, sometimes the temporo-sphenoidal lobe, or the front of a lateral half of the brain. In one case, examined by Dr Goodhart, no granule-masses could be detected in the softened parts, but "the tissue was very fatty and granular, and the nerve-fibres seemed to have undergone destruction, scarcely any of them being visible." The "white softening" of the central parts described at p. 676 appears likewise to be attended with a mere disintegration of the neuroglia and of the nerve-fibres, for no exudation cells are discoverable. I may take this opportunity of remarking that in some cases no ventricular effusion occurs, and that the fornix and septum lucidum then retain their normal consistence. Certain pathologists have therefore supposed that white softening is a mere result of *post-mortem* maceration of the cerebral substance, but such an opinion is untenable.

Basal meningitis without tubercles.—In cases in which the symptoms and course have been altogether like those of tubercular meningitis, the membranes at the base of the brain and in the Sylvian fissures are sometimes found after death with well-marked signs of inflammation, but no evident tubercles. Six or seven cases of this kind are to be found in the records of Guy's Hospital, but in all of them, with one exception, the lungs (and often other viscera as well) contained miliary tubercles; in the one exceptional instance the bronchial glands were caseous. A sufficiently careful microscopical examination might perhaps have cleared up the difficulty, but even without this the changes in other parts seem to me to be conclusive as to the real nature of the disease. Such cases are exactly analogous with those of so-called "pneumonic phthisis," in which characteristic tubercular lesions are found in the larynx, intestine, or elsewhere.

There is, however, a form of acute basal meningitis in which neither tubercles nor scrofulous changes of any kind are to be found in any part of the body. This disease is described by Huguenin as *Leptomeningitis infantum.* He relates as a typical case that of a female child, aged eleven months, who died in the fourth day of an attack of measles, attended with convulsions and other cerebral symptoms. Flattening of the convolutions, injection of the choroid plexuses, distension of the ventricles and softening of the central parts were the only obvious morbid appearances, and I must confess that these would not have deterred me from attributing the cerebral symptoms to the measles; but the microscope showed that leucocytes were present everywhere in the pia mater, especially at the base, though not in such numbers as to render the membrane cloudy or opaque.

A striking instance occurred at Guy's Hospital in 1859, when I was clinical clerk to the late Dr G. H. Barlow. A boy, aged nine and a half years, died after an illness of twelve days' duration, which began with intense headache and ran its course with convulsions, grinding of the teeth, strabismus, and coma. That the disease was tubercular meningitis was doubted by no one who saw the child, and when the skull was opened the brain looked flattened, as if by effusion; but, except that its tissue was soft and that there seemed to be a slight increase of the fluid in the ventricles, no morbid changes were discovered. There were no tubercles in other organs. The real pathology of such cases appears to me to be still doubtful, but they make it necessary for us to be cautious in asserting positively

that a patient is suffering from tubercular meningitis, and perhaps they should especially deter one from giving an absolutely unfavourable prognosis.

Ætiology.—The causes of tubercular meningitis are those of scrofulous affections in general. Impure air, want of exercise, scanty food, are among the chief of them. An inherited predisposition is an important factor in its ætiology; it often attacks in succession several children of the same parents at about the same ages. When this is the case one is apt to suppose that there must be a special morbid susceptibility of the nervous centres, but it is to be borne in mind that, although clinically the cerebral symptoms mask all others, yet the disease is almost always a general tuberculosis. This fact also seems to render it very doubtful whether emotional excitement, over-study, mental shocks, alcoholic intoxication, or blows upon the head can be capable of giving rise to the disease; at least it would seem that they can only act indirectly by lowering the general health, and not directly by disturbing the functions of the brain. Huguenin alludes to two cases in which intense emotional depression was followed by tubercular meningitis; in one there was an interval of fourteen days before the complaint set in. The patient had before enjoyed tolerably good health, but at the *post-mortem* examination it was found that there was latent pulmonary phthisis of old date, and that the lungs also contained miliary tubercles. Several years ago a little boy who was in one of the surgical wards at Guy's Hospital suffering under disease of the hip-joint was placed under chloroform; soon afterwards he was seized with sickness, which caused no alarm, but next day he was very ill, and he at length became comatose and died at the end of a fortnight; tubercular meningitis was found to be the cause of death. This was no doubt an accidental coincidence, and so was another case, that of a policeman, who attributed his illness to over-fatigue in attending a review in Hyde Park in June, 1860, but who afterwards admitted that he had previously been complaining of pain in the head. Three children have died in Guy's Hospital in whom the exciting cause of the disease has been supposed to be a blow or fall upon the head sustained from one to three months before. Thus, unless I am wrong in rejecting the doctrine which would refer tubercular meningitis to infection of the blood by absorption of caseous matters, I can only conclude that we are as yet ignorant of its having any special exciting cause.

Sex and age.—It is a curious fact that the disease is much more frequent in males than in females. At Guy's Hospital the proportion has been as eighty to thirty-seven, and it does not seem to have varied very much at different periods of life, although Huguenin says that below fifteen years of age the preponderance of males is much more marked than in adults.

The relative frequency with which persons of different ages are attacked is not yet accurately known. There are no hospitals to which children and adults are brought in numbers corresponding with their ratios to the population as a whole, and until *post-mortem* examinations become universal the Registrar-General's 'Reports' will fail to do justice to the liability of grown-up persons to tubercular meningitis. It is stated by all writers to be much less common in infants under two years than in older children, but Guersant met with one case in an infant only six weeks old. Three cases have occurred since 1854 at Guy's Hospital in infants aged six months, ten months, and one year respectively. Between two and

four years there have been fourteen cases; between five and seven and a half, nine cases; between eight and ten, twelve cases; between eleven and fifteen, eleven cases; between sixteen and twenty, sixteen cases; altogether sixty-five patients under twenty years old. Between one and twenty and thirty there have been thirty-one cases; between thirty-one and forty, fourteen cases; between forty-one and fifty, eleven cases; between fifty-one and fifty-six, three cases; altogether fifty-nine patients above twenty years of age.

Clinical course.—The symptoms of tubercular meningitis are not essentially different in patients at different ages, but as the best writers have based their descriptions mainly upon observations of cases occurring at an early period of life, and as such cases present certain minor clinical peculiarities, we will begin with the disease as it is seen in *children.*

Premonitory stage.—In the first place, before any definite indications of cerebral mischief develop themselves, a general failure of health is often observed, which may last for several weeks or even for two or three months. The symptoms which are manifested during this period are termed "premonitory" or "prodromal." In the well-known work of Rilliet and Barthez on the diseases of children, an admirable sketch is given, which has afforded materials for all subsequent writers. Probably these symptoms may be due to two different causes, sometimes to a scanty early formation of tubercles in the lungs or other organs, which is afterwards followed by a more abundant crop; sometimes to the slow progress of a chronic scrofulous affection of the mesenteric or bronchial glands. Foremost among them is emaciation; the limbs waste and lose their roundness, the ribs and the bony processes stick out beneath the skin, the muscles feel soft, the skin is lax and flabby. The cheeks often retain their plumpness of outline, so that the loss of flesh is first observed by the nurse who dresses and undresses the child; but the face becomes pale, the eyes are dull, and there is a want of animation in the countenance. The appetite is diminished or capricious. The bowels are disordered, being generally constipated, but with intervals of diarrhœa; the evacuations may be pale and offensive. The disposition and the temper often become changed. The child is dull, apathetic, and slow in its movements; it is easily fatigued by lessons, and quickly tired of toys. It may be fretful and peevish, or it may exhibit a morbid tenderness and affection, breaking off in the midst of a game to throw itself into the mother's arms, and bursting into tears if she should check the unexpected display of emotion. Headache is sometimes present, but more frequently the complaint is rather of being sleepy and tired and wanting to lie down. At night it is restless, lying with the eyes half closed, rousing at the slightest noise, and being unable to sleep with a candle in the room; or it may grind its teeth, and start or cry out with alarm at its dreams. Whether fever is commonly present is doubtful. Dr Gee speaks of feverishness in the evening, but he adds that he knows of no thermometric observations, and the most careful mother often fails to observe any unusual thirst or heat of skin until the premonitory period has passed. He suggests that the presence of a remittent elevation of temperature may perhaps serve to indicate that the formation of tubercles has actually commenced, and that the child is not merely in a state of depressed health such as might be antecedent to their development; but the difficulty seems to me to be that trifling disorders of the stomach and bowels so easily excite feverishness in children.

Premonitory symptoms do not always occur, but Dr Gee says that among twenty-six cases collected by him there were only two in which they were not noticed. Their duration is very variable, sometimes not more than a fortnight but generally longer, and now and then even as much as four or six months. They may subside, and the child's health appear to improve, before the disease breaks out in a characteristic form. What is peculiar to the actual "invasion" of tubercular meningitis is that its further course is limited. In marked contrast to the uncertain length of the prodroma, I believe that life is never prolonged much beyond twenty-one days from the occurrence of the earliest invasion-symptom. The only apparent exception to this rule that I know of is in cases in which one or more solitary tubercles (of the kind described at p. 605) had been developing themselves in the brain before the commencement of the meningeal affection.

Onset.—The symptom that first excites serious alarm is most often the occurrence of repeated *vomiting*. Sometimes the child is sick only when it takes food, sometimes it brings up bilious matters even though it may have swallowed nothing. The sickness generally lasts for two or three days only, but it may go on for a week. If it once ceases for twenty-four hours it seldom returns. In some cases there is no vomiting for the first day or two; now and then it is altogether absent. The symptom which then often ushers in the disease is a *convulsion*, more or less completely epileptiform in character. In one instance mentioned by Dr Gee there was general rigidity which recurred several times; in another the attack took the form of temporary unconsciousness. In a single one of his cases (twenty-five in number) the invasion was marked neither by vomiting nor by convulsions, but by a rather sudden increase of the headache, drowsiness, and wasting which had existed during the premonitory stage.

Severe *headache* is now almost invariably present; the child keeps its hands pressed against the forehead, or may go on rubbing the scalp, first in one place, then in another. From time to time, when exacerbations occur, he perhaps calls out "Oh, my head!" Trousseau lays stress on a peculiar inarticulate "hydrocephalic cry," which is sometimes heard from the very beginning of the disease, sometimes only towards the end; he describes it as a single, sharp, loud sound, like that of a person exposed to some sudden danger; it may be repeated every hour, or every five minutes, for several days together. Rilliet, on the other hand, declares that this symptom is neither frequent nor special to meningitis, and Dr Gee agrees with him. For several days there may be no marked impairment of the intelligence; the child perhaps continues to talk rationally, but his answers may be rather slow and his memory and power of perception somewhat impaired. He is apt to lie in a drowsy state, half asleep, with his eyes staring vacantly, and then he may go on talking senselessly to himself, or repeating some particular phrase over and over again, or singing, whistling, and shouting.

The *pupils* are often sluggish, and very commonly one of them is larger than the other. A tendency to squint is another early symptom of very frequent occurrence. Trousseau relates one case in which there was transient unilateral hemiopia; the child was sitting near a window when it called out, "Oh mamma look at that little boy, he has only half a blouse and half a face!"

Such are, as a rule, the symptoms of tubercular meningitis for perhaps eight days after its invasion. During this period there is more or less fever,

the evening temperature rising perhaps to 102° or 103°, while in the mornings it may be 101°, or 100°, or even not above normal. The pulse may be a little quickened, or natural, or slower than natural; sometimes it is irregular, being easily made more rapid by the slightest excitement, or even becoming so without any obvious cause. The frequency of the respirations is generally but little altered. Constipation of the bowels is almost always present. The tongue may be furred, but it is sometimes almost clean.

Later stage.—When about eight days have passed, the condition of the little patient undergoes a change, which now and then appears to be almost sudden, but more often takes place gradually; and its most striking feature is a loss of consciousness; and the period which follows has been called the "stage of pressure." The child now ceases to take notice of anything that goes on in the room. It commonly lies on one side, curled up with the knees drawn close to the abdomen, and the hands folded over the pudenda. Sometimes the head is drawn backwards and the muscles of the nape of the neck may then be felt to be hard and rigid. It may keep grinding its teeth every few minutes, making a noise which has a most disagreeable jarring effect upon one's ears. The pupils now become dilated and insensible. One or more of the cranial nerves may be paralysed; the third, for instance, so that the eyelid drops; or more rarely the facial nerve. There may be loss of power in the limbs. Tickling the soles of the feet may cause only one leg to be drawn up, the other remaining extended. The evacuations are often passed into the bed without the patient's knowledge.

The order in which the various symptoms make their appearance is uncertain; some of them may commence during the earlier period of the disease, before coma sets in. This is particularly the case with certain changes in the patient's aspect which have now to be mentioned. One is the presence of a frown upon the brows, and deep lines often seem to be drawn around the nose and mouth. There is generally flushing of one cheek, sometimes of both of them; or the whole of the face may be suffused with blood. If the countenance is pale, it may perhaps become reddened when the child is disturbed, or when anything is given to it to drink. So, also, any part of the body which has been pressed upon shows a marked injection of its capillary blood-vessels. A particular instance of this was made into a leading symptom of tubercular meningitis by Trousseau, under the name of the *tache cérébrale*. He pointed out that if one draws one's finger-nail gently over the patient's thigh, or abdomen, or face, a bright red line is produced, and that this differs from the effect of an equally slight scratch in a healthy person, by appearing earlier (within thirty seconds), by lasting longer (eight, ten, or fifteen minutes), and by being broader and of a deeper colour. With regard to the diagnostic value of this sign, we shall have some remarks to make further on (p. 687).

The ophthalmoscope may show ischæmia of the optic discs, or descending neuro-retinitis, or both in succession. Dr Allbutt found some affection of the retina in twenty-nine out of thirty-eight cases examined by him,

Another symptom which may be observed in most cases at this period is retraction of the abdominal walls. Vomiting is generally absent, and the bowels may still remain obstinately constipated; but for some unknown reason the intestines no longer contain the usual quantity of gas; and the belly therefore becomes deeply hollowed, or (to use a common expression) "boat-shaped," the rib-cartilages, the iliac crests, and the pubic symphysis

appearing unduly prominent. The tongue may now be red and dry, but it often still remains moist. The temperature seldom rises above 101°; Dr Gee remarks that it may for days together remain at between 96° and 98°. Thus, if the case is advanced when the child is first brought to one, the disease may appear altogether non-febrile. The pulse during this period is generally infrequent, 60 or even 50 per minute; the number of its beats may each day be less than before; it is still apt to be irregular and unequal in force. A similar irregularity and inequality of the respiratory movements are very commonly present; the child perhaps breathes rapidly three or four times in succession, and then the chest may remain motionless for some little time. Trousseau laid great stress on this symptom, and has recorded a case in which the breath was held for as long as fifty-seven seconds. Typical "Cheyne-Stokes respiration" is sometimes observed.

Final stage.—The "stage of pressure" may continue with but little alteration until it terminates in the patient's death; this being perhaps immediately preceded by a convulsive seizure. But in certain cases the symptoms during the last twenty-four or forty-eight hours are to some extent peculiar. One remarkable change is that there may be a brief return of consciousness for a short time before the end. I once saw a striking instance of this myself. Dr West relates how a girl, aged seven, who had been in a state of stupor for six days, and profoundly comatose for two days, became conscious, swallowed some drink, spoke sensibly, and said she knew her father; in the course of an hour and a half, however, she became worse again, and a little later she died. The pulse often becomes rapid during the last two or three days; and as Dr Gee points out, the temperature may steadily rise until it is above 107°. But in some cases the beats of the heart remain infrequent up to the time of death; and the temperature even in the rectum may fall, until it is very low indeed. In one case, three days before the fatal termination, the thermometer registered 97·8° and 96°; next day the highest temperature was 96·2°, the lowest 93°; the day after they were 82·8° and 82·1° respectively; and on the day of death 80·5° and 79·4°. In other cases the face and limbs are livid and cold, and covered with a clammy sweat, while the thermometer shows that fever is still present. Towards the last a peculiar fœtid earthy smell is often perceptible, which I do not remember to have noticed in any other disease. I am surprised to find that Rilliet and Barthez, after alluding to Whytt having mentioned this symptom, go on to say that they have not themselves recognised it.

Symptoms in adults.—The onset is comparatively seldom preceded by marked prodromal symptoms. As I have already observed, it is often secondary to phthisis, and, when this is the case, the pulmonary affection is of course rightly regarded as the cause of any general failure of health that may have been noticed. Then, again, few grown-up persons are watched as carefully as children are by their parents and nurses; and in adult life loss of flesh may be due to so many other conditions that tubercular meningitis is very unlikely to be thought of until its symptoms are actually developed. But apart from all these considerations, I must state it as my impression that most of the patients whom I have seen have been fairly well-nourished at the time of death. I have no recollection of having ever observed a very marked degree of wasting, unless when it was obviously referable to some co-existing visceral disease; and in the case of Harriet Staunton, already referred to at p. 678, I should have been reluctant to regard the extreme

emaciation, which must have been going on for several weeks, as dependent upon the presence of meningeal tubercles.

The actual invasion is, I believe, far less often ushered in suddenly by vomiting in adults than in children. Dr Gee, indeed, speaks of vomiting as an early symptom, occurring in almost every instance; but neither of the last two patients whom I saw in consultation had any sickness, at least up to a time when other characteristic symptoms had developed themselves. What particularly impressed the fact on my mind was that in each case the medical man in attendance had been led to a diagnosis of enteric fever by finding that the stomach did not reject its contents. Again, I do not find that in a grown-up person an epileptiform seizure is of frequent occurrence as a mere invasion-symptom, followed by the slow development of the disease in regular stages. In adults the disease seems generally to begin very gradually and insidiously. On the other hand, we have had at Guy's Hospital more than one case in which the sudden occurrence of convulsions has been the precursor of a fatal termination within a day or two, and in which an autopsy has shown that the disease was tubercular meningitis. It is not at all uncommon for death to take place after an illness of twenty-four or forty-eight hours only. In 1868 a man, aged thirty-two, a patient of Dr Wilks, died within two days of having been about his business, as a draper; on admission he was so restless that he had to be held down in bed, but he quickly became comatose. In one instance there was violent delirium as late as two days before death.

In some cases of tubercular meningitis, in adults, the first symptom is well-marked local paralysis; I do not know that the same thing ever occurs in children. In 1871 a woman, discharged from a surgical ward because she had advanced phthisis, came the same morning to the taking-in room to seek readmission as a medical patient. No symptoms of cerebral disease had been observed, but I found that she had paralysis of the left facial nerve, an affection which must have developed itself within a few hours. I therefore took her into the Clinical Ward; she seemed rather stupid, but was intelligent enough to be able to tell us that she could not hear the ticking of a watch until it was placed in contact with the left ear, whereas on the right side her hearing was perfect. However, she quickly became drowsy, and then comatose and unable to swallow; and at the end of six days she died. At the autopsy I failed to discover any special affection of the facial nerve, either in the petrous bone, or elsewhere. So far as I know, Huguenin is the only observer who has attempted to trace the affections of special cranial nerves occurring in tubercular meningitis to definite local changes. In one instance, in which the face was paralysed, he found the portio dura thinned, of a yellow colour, and covered with a large quantity of pus; cellular elements were present in numbers in the nerve-sheath, but the fibres appeared to be intact. In another similar case, however, no change could be found in the nerve. In one instance, in which there had been convergent strabismus, the abducens was greatly diseased, diffuse suppuration having occurred between its fibres. Similar appearances were twice discovered in the third nerve, when the muscles supplied by it had been paralysed during life; but in some other cases, in which the same symptoms had been incompletely developed, the nerve seemed to be normal.

Lastly, tubercular meningitis in adults may have hemiplegia for its earliest, and even for its principal, symptom. Three instances of this have been observed at Guy's Hospital within the last few years. One patient

was a man, aged thirty-three, who was admitted on account of an abdominal tumour which proved afterwards to be a tuberculous omentum. Some days before his death he was attacked with loss of power in the left arm and leg; he was sensible to the last. In the other two cases the right limbs were affected, and, as the superficial seat of the lesion might have led one to anticipate, the paralysis was accompanied by well-marked aphasia. One patient was a woman, aged twenty-six, who came under Dr Wilks's care in 1867; I well remember that, until the real nature of the affection was revealed at the autopsy, there was not the slightest suspicion of its being due to any cause other than some disease of the Sylvian artery. The third, also a woman, aged forty-one, had been attending as an out-patient for phthisis, when she was seized with right hemiplegia and loss of speech; afterwards she became semi-delirious and her paralysis changed sides. In none of these three cases was any well-marked change found in Broca's convolution or in the adjacent parts of the brain; but in a boy, aged nine, in whom right hemiplegia and aphasia were combined with the more ordinary symptoms of tubercular meningitis, there is said to have been red softening of the left third frontal, and of the inner ends of the two left ascending (or central) gyri. Huguenin relates three instances in which paralysis of the right limbs and loss of speech were the chief symptoms; in two of them many tubercles were present in the left Sylvian fissure, but none in the opposite one, and in the third case the pia mater in the left fissure was more thickened than anywhere else. In one of the cases in which there was red softening of the left island of Reil and of Broca's convolution, the first symptom after headache was speechlessness, but this lasted only about seven minutes, and for two days afterwards the patient (a man, aged fifty) was able to go to business as usual. One can only suppose that the loss of speech was due to functional disturbance of the brain; or, in other words, that it was a neurosis, and analogous to what has been observed in other cases, referred to at p. 584.

Huguenin raises the not very important question whether it is generally possible from the symptoms to determine the presence of ventricular effusion, or the extent to which tubercles are developed on the convexity of the brain and at the base respectively. His statements and the observations which have been made at Guy's Hospital seem to show that when there are no inflammatory changes in addition to the tubercles there is apt to be no persistent coma. Violent delirium is sometimes associated with the presence of very numerous tubercles between the convolutions of the upper surface of the brain. Choked discs are probably an indication of a general increase of pressure within the cranial cavity, whereas neuro-retinitis points more definitely to the existence of an inflammatory process at the base of the brain, in the neighbourhood of the optic tracts or nerves. In one case in which there was impairment of power in all four limbs, Huguenin found changes in the superficial layers of the crura cerebri. The extension of meningitis to the spinal canal does not seem to give rise to any very marked special symptoms. It probably gives rise to a rigidity of the muscles of the neck, which (in children at any rate) is very common, for this is one of the chief symptoms of an epidemic form of meningitis which until recently was supposed to be peculiar in affecting the spinal as well as the cerebral membranes; and sometimes the occurrence of painful cramps or spasms in the limbs may be attributed to the same cause.

Diagnosis.—This may be very easy or very difficult, either in children

or adults. During the first few days of a case which is to last two or three weeks one is often unable to speak positively, but there is seldom much uncertainty when its fatal character is once fully developed.

All writers lay stress on the importance of distinguishing it from *enteric fever*, and the fact is indisputable that many cases, which for a week or ten days are supposed to be examples of that disease, afterwards become attended with such well-marked cerebral symptoms that they are forthwith transferred to the category of tubercular meningitis. Thus Rilliet and Barthez describe a special form as having a *début typhoide*. But although to the clinical observer the meningeal affection thus masks all the other features of the case, yet the pathologist almost always finds it to be only a part of general miliary tuberculosis, which in reality constituted the patient's illness from its commencement, and to which the early febrile symptoms were due. In our reports at Guy's Hospital I find only two recorded instances in which the first diagnosis was that of fever, and in which tubercles were found in the meninges and nowhere else. Sometimes, indeed, the opposite error is committed, enteric fever being attended with strabismus and irregularity of pupils, vomiting, and constipation, as well as with headache, delirium, and coma, so as to be taken for meningitis. So far as I know the *tache cérébrale* of Trousseau affords very little help in the diagnosis of cases that would otherwise be doubtful. Dr Gee says that in tubercular meningitis he has often been unable to bring it out except by using an unfairly hard stroke, such as would cause reddening of the skin in any child; and I have seen instances in which this symptom has been present, but in which a review of the whole course of the disease, after the recovery of the patient, has led to the conclusion that it was enteric fever. The clinical value of ophthalmoscopic changes in the optic discs is still somewhat doubtful. It is certain that a normal state of the retinæ is no proof of the absence of tubercular meningitis, but I believe that the time has not yet arrived for a dogmatic expression of opinion as to the positive significance of ischæmia (or even of retinitis) as between that disease and some less severe affection of the brain, such as might be attended with great vascular congestion of its tissue. One appearance, indeed, is conclusive, namely, the presence of tubercles in the choroid. It is true that they belong, not to the meningeal affection itself, but rather to a general acute tuberculosis, but this fact in no degree diminishes their diagnostic importance. For, on the one hand, the membranes of the brain are very seldom the sole seat of miliary tubercles, and, on the other hand, whenever there are any tubercles in the membranes the case always assumes clinically the aspect of a cerebral disease, even though they may be infinitely more numerous elsewhere. In fact, in all cases of suspected tubercular meningitis one should carefully search the lungs and other parts for the very slight indications of acute tuberculosis which they sometimes yield; one ought even to examine the testes, the lymphatic glands, and other organs, for chronic lesions of the same nature.

Another morbid state, that formerly used often to be mistaken for tubercular meningitis, is one which is of frequent occurrence in young children, and which has been rendered classical by the descriptions given of it by Marshall Hall (1825), Abercrombie (1828), and Gooch (1829). The first of these writers called it "*the hydrocephaloid disease*;" Sir Thomas Watson gave it the name of "spurious hydrocephalus;" and Dr Gooch stated that he had "invariably found it attributed to and treated as, con-

gestion or inflammation of the brain." But at the present day I do not think that a skilled practitioner is likely to fall into the error of diagnosis which fifty years ago was so frequent; and at any rate the mistake is not now likely to be attended with any serious results. Among the chief causes of the affection was one to which little children are no longer liable; namely, the free withdrawal of blood, under medical authority, in spite of existing depression of the vital process. A case related by Dr Hall is that of a girl, aged two and three quarter years, who had had sixteen leeches applied for an attack of influenza; and he states that all his patients were in a state of exhaustion before they were attacked by the cerebral symptoms; many of them had had protracted diarrhœa after weaning. On the other hand, Dr Gooch says that in most of his cases there had been no previous illness; but perhaps he was hardly keen enough in the appreciation of the gradual effects of insufficient or improper food. The child's aspect is characteristic; it lies on its nurse's lap, unwilling to raise its head, drowsy or even comatose with sunken, half-closed eyes, dilated insensible pupils, irregular and sighing respiration. The face is pale, and the skin cold. Dr Gooch, indeed, speaks of a slight and transient flush, as being sometimes present, and Dr Hall, of an early stage is which the little patient is irritable, restless, feverish; but such symptoms very seldom, if ever, last long enough to mislead a careful observer into the diagnosis of meningitis. Depression of the fontanelle in an important symptom of exhaustion in children, although it seems formerly to have escaped notice. The proper treatment is to give ammonia and brandy, but above all, to take care that suitable food is supplied. For an infant recently weaned a wet nurse should generally be procured; and a return to the breast is often advisable, even when the child is some months old and has long been fed with the bottle. At least, it ought to have asses' milk, or goats' milk, if cows' milk should appear not to be readily digested. To prescribe leeches and calomel would, of course, be fatal; but this kind of practice is now obsolete in tubercular meningitis,

At Guy's Hospital we have had cases which during life were supposed to be examples of *mania*, *delirium tremens*, or *epilepsy*, and in which the disease has turned out to be tubercular meningitis. Huguenin remarks upon the possibility of mistaking the latter disease for *uræmia*, when a chronic renal affection, in reality tuberculous, is attributed to morbus Brightii. A curious case occurred some years ago at Guy's, that of a painter, who came saying that he had "lead colic," and that his bowels had not been open for a fortnight. In the evening, after his admission, he had convulsions and became insensible, and on the morrow he died. There was strumous disease of two of the lower dorsal vertebræ, with a large abscess in front of the spine; and the membranes of the brain and cord showed the characteristic appearance of tubercular meningitis. The same disease has been found on *post-mortem* examination in two cases of boys admitted into the surgical wards for symptoms supposed due to vesical calculi; no cause could be discovered for the irritability of bladder from which they had been suffering before any obvious signs of cerebral mischief appeared. A very similar case occurred in a woman who was taken into a medical ward for trouble with her bladder; but in addition to the affection of the membranes of the brain there was a tumour in the spinal cord, and this may possibly have given rise to the vesical symptoms.

The diagnosis between tubercular meningitis and other *organic affections*

of the brain is of comparatively little practical importance. The prognosis is scarcely, if at all, affected by it; and there are very few cases in which the treatment would be different. The points which distinguish this kind of inflammation of the membranes of the brain from the other kinds will be indicated hereafter when they have been described. I believe that many cases have been recorded as examples of sporadic "cerebro-spinal fever" in England, which have really been tubercular. Particular cases of abscess or tumour of the brain may sometimes resemble those of meningitis very closely; but it can hardly be said that a mistake in this direction is of serious consequence. The single exception is that in grown-up patients one must always be alive to the possibility that syphilis may be the cause of the most varied cerebral symptoms.

In adults, however, the most serious error of all is to mistake tubercular meningitis for *hysteria*. I shall never forget a case which I saw many years ago, in a clergyman's daughter who had been unfortunate in love, and was attacked with delirium; until within a few hours of her death, no one was able to speak confidently as to the nature of the disease. Two similar instances have occurred at Guy's Hospital. One patient, a woman, aged twenty-five, had long been "odd in her mind and scarcely to be trusted;" she was attending as an out-patient, with hysterical symptoms, when she sought admission and died three days later. There were several yellow tubercles, of the size of peas, in the brain, as well as the usual appearances of tubercular meningitis.

Prognosis.—The reason why it is of so great consequence that one should not mistake tubercular meningitis for hysteria or enteric fever is that the prognosis of these affections is so different. Tubercular meningitis is one of the most fatal of all diseases. Rilliet has indeed recorded an instance of recovery, in which the child died five and a half years afterwards of a second attack, and at the autopsy the remains of the former mischief at the base of the brain were clearly recognised; and a similar case is mentioned by Trousseau.* Among all the *post-mortem* examinations made by the author he only twice found the membranes on the under surface of the encephalon matted together in such a way as to suggest that inflammation had formerly occurred there. In neither instance was any caseous matter present; so that, if meningitis was really the cause of the adhesions, it may probably have been a simple, rather than the tubercular, form of the affection.

However, it is by no means a very rare circumstance for recovery to take place in cases in which tubercular meningitis had been diagnosed more or less positively, and for the nature of the disease to remain uncertain, as it must necessarily remain in such cases, unless an accident should lead to an autopsy being made at a future time. Most observers are disposed to think that these are generally instances of enteric fever complicated by cerebral symptoms. But it is surely fair to remark that in some fatal cases the usual morbid changes are not discoverable, while the absence of tubercles in the other organs deprives us of all warrant for the supposition that the microscope might reveal their presence in the membranes, even in the most minute quantity. I would not venture to assert that the disease is then merely a neurosis. But even if some as yet unrecognised lesion should hereafter be demonstrated, one might at least suppose that it need not necessarily prove fatal. How closely a case which terminates in recovery may resemble tuber-

* The late Dr Carrington recorded a similar and most conclusive case.

cular meningitis is strikingly illustrated by a clinical history, which we owe to Dr West. A child, aged three years and a half, a member of a phthisical family, was attacked by a disease which ran the ordinary course of acute hydrocephalus, unchecked by the customary treatment. Convulsions took place, coma succeeded them, deglutition was very difficult, the pupils were dilated and almost motionless, the pulse was very feeble and very frequent, and everything portended a speedy death. A younger brother had died a year before of the same disease. Food was still given as the power of swallowing was not entirely lost, and ammonia and ether were administered, and after a time quinine. For days the child remained unconscious, but at length she began to raise her hands to steady the cup that was put to her lips. Next she recovered her sight, after some weeks she became able to speak, and after many months she began to walk with a tottering step. Three years afterwards, although her intellect was not defective, she still had a vacant smile, and had never regained flesh, nor recovered the look of health; her gait also remained unsteady. Whatever the real nature of this case may have been, one cannot be wrong in deducing from it the necessity of being very cautious in asserting that it is impossible for a patient to get over an attack of what appears to be tubercular meningitis. However confident we may be in our diagnosis, and however threatening may be the symptoms, our opinion should be given with some reserve.

The *treatment* of tubercular meningitis will be discussed when the simple forms of the disease have been described.

Epidemic Meningitis.*—From the earliest years of the present century there have been recorded, from time to time, in various parts of the northern hemisphere, epidemics of a disease characterised anatomically by inflammation of the membranes of the brain and cord, and clinically by fever, various eruptions, and a number of cerebral and spinal symptoms, especially rigidity of the neck, or of the whole vertebral column. So striking is the symptom last mentioned that in Germany it has given to the affection the popular names of "Genickkrampf" and "Nackenstarre." In medical works it has been called "epidemic cerebro-spinal meningitis." But the epithet "cerebro-spinal" is likely to encourage the incorrect notion that an extension of inflammatory process to the membranes of the cord is more or less distinctive of it as compared with tubercular meningitis.

History.—The first well-ascertained epidemic of this disease seems to have been in 1805 at Geneva. In 1806 it appeared in the United States and continued to prevail there for ten years. During this time, and indeed throughout the past half of the century, it was observed in different towns of France and of Italy, in Algeria, Spain, Denmark, &c. In 1854 and for seven years afterwards it raged in Sweden, destroying more than 4000 persons in that country. From 1861 to 1864 it showed itself in various parts of the United States. In 1863 it broke out in Germany; the north-eastern provinces of Prussia were the first to suffer from it; but within the next year or two it appeared in Erlangen, in Nuremburg, and in other South German towns, and in the country districts of Franconia. From that time it has never ceased to show itself at intervals of a few months or longer, now in one part of the German Empire, now in another. Writing in 1874, Ziemssen said that it seemed to be naturalised.

The British islands have hitherto been remarkably free from this disease.

* *Synonyms.*—Cerebro-spinal Meningitis—Cerebro-spinal Fever.

In 1846 it appeared in many of the workhouses of Ireland; and in 1866–68 a very fatal type of it prevailed in Dublin, and to some extent in other parts of the country. Scotland, I believe, has been altogether spared by it; and in England only a few isolated and small epidemics have been observed in certain provincial towns and villages. Its occurrence in London has not yet, I think, been satisfactorily established. A few cases have from time to time been recorded as sporadic examples of it by writers who seem to have thought that the fact that a meningitis was cerebro-spinal was of itself sufficient to justify a presumption that it was related to the epidemic disease. But now that we know that all forms of inflammation of the base of the brain are apt to extend to the cord, this conclusion is of course untenable. And the symptoms observed in the various cases in question have not seemed to me to support such a hypothesis.

Anatomy.—The morbid anatomy of epidemic meningitis has, in fact, little or nothing to distinguish it from that of the sporadic form of the disease. Pus and lymph are found both at the base and on the convexity of the brain, especially between the pons and the chiasma, along the large vessels, and in the various depressions and furrows on its surface. It rarely happens that the hemispheres are uniformly covered by it. In the spinal canal the exudation is generally most abundant at the lower part, and on the posterior surface of the cord, having perhaps accumulated there by gravitation. The ventricles of the brain mostly contain turbid serum or pus; the choroid plexuses and even the whole of the ependyma may be coated with puriform lymph; in one case Fronmüller is said to have found the central canal of the cord dilated and full of pus. Punctiform hæmorrhages or small spots of softening may be seen in the cerebral substance; or it may be simply œdematous and flattened. The cord presents similar changes, but less marked.

Among the appearances presented by other organs are congestion and œdema of the lungs (especially of their lower lobes), engorgement of the liver and spleen (the spleen, however, is not very often much enlarged), a relaxed state of the heart, congestion of the kidneys with fatty epithelium and fibrinous casts in the tubes, and a fine granular degeneration of the fibres of the voluntary muscles in general, and especially of those which lie along the spine. Rigor mortis is said to be of long duration. The cadaveric stainings of the surface appear early, and are not always limited to the dependent parts of the body.

The pericardium and the pleuræ have sometimes been found ecchymosed, or even lined with puriform exudation. Von Ziemssen once saw the large intestine inflamed as in dysentery. The joints often contain pus, and sometimes there are scattered abscesses in the connective tissues and muscles. All these changes must be regarded as complications dependent on a secondary blood-poisoning.

Course and symptoms.—As a rule cerebro-spinal fever sets in suddenly the patient may be at work, or (if a child) at play or at school, when he is seized with shivering and violent pain in the head, and feels so ill that he is obliged to go to bed at once. But sometimes—among Ziemssen's cases in five out of forty-three—there are slight premonitory symptoms, consisting of headache, malaise, nausea, loss of appetite and wandering pains. In several instances these passed off, and there was an interval, in which the patient felt perfectly well, before the disease began with its usual violence.

Vomiting is almost always an early symptom, being repeated whenever an attempt is made to sit up. After a day or two it generally ceases, but

the headache mostly continues throughout the whole course of the disease, although it may sometimes subside for a time. It varies in character and in seat, being sometimes frontal, sometimes occipital, sometimes diffused over the whole of the head. Giddiness is often present with it. In cases of moderate severity the patient lies in a state of stupor, tossing restlessly about, but rousing when spoken to and trying to answer. He is often very irritable to light and sound. The pupils may at first be normal or contracted; ultimately they become dilated. In the more dangerous cases he quickly becomes delirious, with or without convulsions, or passes into a condition of insensibility. Even when he is deeply comatose he often still feels the pain in the head, and groans or cries out, or grasps the temples between his hands. Aphasia, hemiplegia, and other paralytic symptoms of this cephalic group are sometimes observed; but in the most severe cases of all their presence can hardly be determined.

The *muscular rigidity* of the neck already referred to is scarcely ever entirely absent, but is not commonly a marked symptom during the first day or two. It varies in degree from a slight stiffness, noticed only when an attempt is made to bend the head forwards up to a forcible retraction, bringing the occiput almost to a right angle with the spine. Dr Burdon Sanderson has suggested that it is due to a half voluntary effort on the part of the patient for the relief of pain in the muscles; but it may be present when there is not pain at all, either in the neck or in the back. It would seem, however, that the muscles below the occipital bone do not feel as hard as might be expected; for Sanderson could not detect any tightness of them so long as the head was thrown back, and Ziemssen probably means little more when he says that the tetanic spasm is "limited to the deeper muscles, the trapezii almost invariably escaping." In about half the cases this symptom is accompanied by a contraction of the extensor muscles of the dorsal and the lumbar vertebræ. Sometimes the back is arched so as to be in a state of opisthotonos, but more often it is straightened into what Ziemssen calls *orthotonus*. If an attempt is made to raise the patient, he either slips down to the foot of the bed without bending his back at all, or allows his body to be lifted a very little way, and at the cost of so much pain that he is very soon put back into the recumbent posture. Almost always he lies on one side with his knees drawn up. He may then be nearly free from pain in the back, but sometimes its intensity is hardly at all affected by the position of the body; it is apt to be particularly severe in the sacrum. Pains in the limbs, and especially in the legs, are often complained of; some writers lay stress on the frequency of an acute pain in the knee. The joints may become hot, red, and painful as is the case in other spinal affections. An extreme cutaneous hyperæsthesia is another common symptom; the patient, though he may be comatose, will perhaps scream out at the slightest touch, or even if his bed is shaken. Tetanic rigidity of the limbs is seldom present, and so also trismus is of infrequent occurrence.

The degree of *fever* in epidemic meningitis is exceedingly variable, and its course is very irregular, so that even those observers who lay most stress on the typical fever-courses of many other specific diseases admit that nothing of the kind can be traced here. The temperature usually ranges from 100° to 103°, but it may fall and remain normal or nearly so for a day or two at a time; sometimes it rises to 105° or 107°, especially towards the last. The rate of the pulse may be natural or slightly increased; it is liable to frequent fluctuations; in bad cases it may be very rapid. The face is generally pale.

The spleen is sometimes, but rarely, found enlarged. As a rule, the abdomen is retracted, but it may be greatly distended. Ziemssen observed several cases in which complaints were made of oppression at the epigastrium, of constriction of the chest, and even of severe paroxysms of dyspnœa. An abundant secretion of urine has been noticed by several German physicians, even when the fever was high; in exceptional cases a small quantity of albumen or of sugar has been present.

An important symptom in this, as compared with other forms of meningitis, is the occurrence of certain *cutaneous eruptions*. Chief among them is herpes of the face. This generally begins on the lips and spreads to the cheek, nose, ear, eyelids; it is often bilateral, it may cover the whole side of the face, and thus it is far more marked than in any other acute disease. It first appears between the third and the sixth day, but fresh outbreaks of it may take place as late as the sixth or the seventh week. It may be seen even on the trunk or on the limbs; those parts, however, are more often the seat of a roseolous or an erythematous rash, an urticaria, or a diffused and extensive purpura. There appears to be nothing specific in the characters of any of these eruptions; they are often mixed together in the same case. Ziemssen lays stress on the symmetry with which they are distributed on opposite sides of the median plane—herpes on each wrist, urticaria on each leg, or petechiæ on each shoulder. Dr Collins, of Dublin, saw purpuric spots suppurate and scab over, so that they finally left pitted cicatrices. Sometimes large patches of hæmorrhage coalesce and give a uniform black appearance to a considerable part of the body.

The organs of *sight and hearing* are affected in many cases of cerebro-spinal fever. There is often an intense conjunctivitis, attended with extreme chemosis. Ulceration of the cornea, irido-choroiditis, optic neuritis, may each of them develop itself, leading to the usual consequences of these several lesions. Vision may be suddenly lost at an early period of the disease; in cases which recover the patient is apt to be left more or less completely blind.

The ears were found by Ziemssen to suffer in eight cases out of forty-two. Pain, tinnitus, impairment of hearing were generally complained of soon after the patient fell ill; they either passed off or ended in a partial or total deafness. Such symptoms sometimes depend upon suppuration of the tympanum, leading to perforation and discharge through the meatus It has been suggested that in other instances they may be direct results of inflammation of the floor of the fourth ventricle, involving the *striæ acusticæ*, or that they may be due to the presence of purulent exudation about the seventh pair of nerves. Ziemssen, however, remarks that he has often found both these lesions in cases in which there was no affection of the auditory functions during life. In certain cases, on the other hand, in which deafness had been present, Heller discovered after death a suppurative process in the labyrinth, besides an infiltration of the portio mollis with pus. It then became a question whether these morbid changes were caused by an extension of mischief from the pia mater, or whether they began simultaneously with the meningitis. Heller was disposed to adopt the former opinion. It is curious that in cases of this kind the portio dura constantly escapes and that facial paralysis is not observed. Severe inflammation of the labyrinth usually leads to an absolute loss of hearing; and in most cases both ears are affected. The consequence is that the patient, if very young, never learns to speak. Even children two or three years old,

who were able to talk before they fell ill with the meningitis, and whose articulation after their recovery was at first tolerably distinct, soon begin to lose the power of speech, and ultimately become unintelligible. Hirsch, indeed, says that there is sometimes an aphasia, as an immediate result of the cerebral affection; but this was never observed by Ziemssen. How important a part may sometimes be played by epidemic meningitis in the production of deaf-mutism is shown by the fact that in 1874 every one of the inmates of an asylum at Bamberg owed the defect to an attack of that disease.

Varieties.—Cerebro-spinal fever has many degrees of severity, so that systematic writers describe several distinct forms. Some cases are called *foudroyant* or fulminant, the patient dying within a few hours from the commencement of his illness. Thus Dr Gordon recorded one instance in Ireland in which the disease ran a fatal course in less than five hours. There was a dark purplish eruption of spots of various sizes and shapes, especially upon the lower limbs. Ziemssen says that among forty-three cases he met with four in which the duration was from twelve to thirty hours. He relates a curious instance of a girl who was attacked one afternoon with headache and vomiting, but who got up in the middle of the following day feeling perfectly well, and went out of doors to fetch some beer; at about 2 o'clock she was again seized with violent headache and fell into convulsions, and at 6.30 she died. This form of the disease is seen chiefly at the beginning of an epidemic. According to Ziemssen, the presence of exudation into the membranes is discoverable only with the aid of a microscope, which reveals an infiltration of cells, principally along the blood-vessels. But in Dr Gordon's case greenish lymph had already been poured out both on the surface and at the base of the brain, and in places along the cord.

On the other hand, there are cases which are termed *abortive*, in which the patient may not be confined to bed for more than a day or two, or may even go on with his work as usual from the beginning to the end of his illness. Ziemssen has recorded in detail three examples of this form of the disease; in each of them headache, a painful stiffness of the neck, and vomiting (or at least nausea) were present; in one there was also rigidity of the upper dorsal vertebræ, and in another herpes and partial deafness were observed. In every instance recovery took place within four or five days. Abortive cases are said to be especially numerous when an epidemic is declining. Their proportionate frequency is very variable. According to Hirsch it sometimes happens that the greater part of the population of a district in which cerebro-spinal fever is prevalent are affected by this mild variety of the disease. Or it may be seen in the adults and the old people, while the severe form is raging among the children. To what extent the morbid changes in the membranes are developed in cases of this kind is as yet altogether unknown.

As a rule, however, the severity of epidemic meningitis is intermediate between the two extremes just mentioned. If it terminates in recovery, it does not begin to subside before the end of a week or a fortnight. There are certain minor modifications of the disease to which brief reference must be made. One is an "intermittent" form, in which there are regular paroxysms of fever, recurring after a quotidian or tertian type, with aggravation of all the other symptoms, the intervals being more or less completely apyretic. There are two ways in which cases of this kind have

been explained; one supposition has been that they depend on the combined action of the marsh-poison and of the specific poison of epidemic meningitis; the other, that the last-named disease is itself really of malarial origin. But it is evident that neither of these opinions is correct, for the intermittent variety has been observed in districts where ague does not occur. Moreover, Ziemssen has shown that when measured by the thermometer the fever is far from exhibiting the regular gradations which are characteristic of that disease. The remissions or intermissions often last over several days, and the temperature chart is most indefinite, and is not at all modified by the administration of quinine. Another so-called variety is the "typhoid." This arises in protracted cases, and is marked by muttering delirium, a dry brown tongue, sordes on the lips, involuntary evacuations, and bedsores.

Several observers have found that during or just after an epidemic of cerebro-spinal fever, meningitis has presented itself with unusual frequency as a complication of other acute diseases. This is especially apt to be the case with acute (croupous) pneumonia; no less than fourteen instances of it are recorded as having occurred in Erlangen between 1866 and 1872. It has also been noticed with pleurisy, acute tonsillitis, and scarlatinal nephritis. Many of the patients recover perfectly well, the headache and stiffness of the neck passing off as the symptoms of the primary malady subside; it may then be said that the meningitis is of the "abortive variety. But not a few such cases terminate fatally; and the usual morbid changes in the membranes are then found at the autopsy.

Relapses are not very uncommon, even in cases of moderate severity, in which the disease began to subside after a week or two of illness. And though the patient's recovery should be uninterrupted, it is often very slow. The headache sometimes continues throughout the convalescence, and may persist for years afterwards, undergoing aggravation when he stoops, or makes any mental or bodily effort. Ziemssen says he has seen several instances in which the spine remained stiff during several weeks.

These symptoms may probably be referred to cicatricial thickening of the pia mater, and this has been actually found present when there has been an opportunity of making an autopsy, at an interval after recovery from epidemic meningitis, in consequence of chronic hydrocephalus having developed itself as a sequela. Recent observations seem to show that this is not an infrequent occurrence. Ziemssen has recorded three instances in which death happened ten weeks, twenty-seven weeks, and thirty weeks respectively from the commencement of the patient's illness; and one in which it took place as late as seven years after. In all of them the membranes were thickened and opaque in different places; and twice cheesy masses were found upon the convexity as well as at the base of the brain. The quantity of fluid in the ventricles was often very great; in the case which proved fatal at the end of thirty weeks—that of a boy, two years old—the thickness of the hemisphere (white and grey matter together) was only about an inch. A sufficient explanation of the occurrence of hydrocephalus under such circumstances may perhaps be found in the persistence of the inflammation of the ependyma which exists during the acute stage of the disease. But, as Ziemssen remarks, the fact that between the meningitis itself and the commencement of the symptoms of the ventricular effusion there is often a clear interval, during which convalescence appears to be going on favourably, tends to support the opinion that the secondary

affection is in some way dependent on the cicatricial changes in the membranes. Niemeyer has suggested that the outflow of blood through the veins of Galen is interfered with by pressure. Dr Collins, of Dublin, in a case which he examined on the sixty-sixth day, found the cerebro-spinal opening between the cerebellum and the bulb occluded, and attributed to this the hydrocephalus, in accordance with the well-known views of Mr Hilton. But in a series of cases recorded by Dr Merkel, of Nuremberg, in vol. i of the 'Deutsches Archiv,' it was noticed that there was always also a large quantity of fluid beneath the arachnoid around the *cauda equina*. This, it is obvious, is altogether inexplicable on the mechanical theories just referred to. And on the whole it seems best to fall back upon the idea of a chronic ependymal inflammation, which indeed is directly supported by the histological investigations of Merkel, who found the ventricular lining and its vessels thickly set with nuclei.

The symptoms which indicate the supervention of hydrocephalus are said by Ziemssen to be chiefly severe headache, and pains in the back and limbs, occurring only in paroxysms, and attended with vomiting, loss of consciousness, convulsions, and the involuntary discharge of fæces and urine. During the intervals, which may last for weeks at a time, the patient may appear to be in good health, mentally and bodily; but very often he is dull and stupid, or he is affected with a general cutaneous hyperæsthesia, or with paralysis or contraction of one or more of the limbs. Progressive emaciation appears to be another marked symptom. Whether it is possible for recovery to take place from the hydrocephalus is as yet uncertain. It is to be noted that failure of memory and weakness of intelligence, when they immediately follow an attack of epidemic meningitis, do not point to the presence of ventricular effusion, and are not of evil omen, since they generally pass off in the course of a few months. And the same thing may be said of various paralytic affections which are now and then observed during convalescence from the disease.

Ætiology.—The causes of epidemic meningitis and its relations to other so-called blood diseases are still very obscure. Those who are attacked by it are comparatively seldom over forty, and generally less than twenty years of age; in some epidemics almost all the cases have been in children under fifteen. But two of Ziemssen's patients were old people aged seventy and seventy-seven respectively. Males appear to be more often affected than females. Ziemssen says that it chiefly seizes upon strong healthy subjects, but he and other observers are agreed that it is apt to be especially prevalent among the poor, who are ill-fed and who live crowded together in dirty, damp, ill-ventilated dwellings. When it occurred in France at the beginning of the century it was often entirely limited to the soldiers in barracks, the civil population in the same towns escaping entirely. In Ireland in 1846 it affected principally the inmates of the workhouses; in the United States it fell with especial severity upon the negroes.

Some of the facts just mentioned would seem to indicate that epidemic meningitis resembles typhus in its distribution, and may therefore be infectious. But of its being contagious in a narrow sense there is no evidence whatever. All observers are agreed that it does not pass from the sick to the healthy directly under ordinary circumstances. Hirsch, however, has collected a series of cases which seem to show that a man going from an infected to a healthy place may sometimes carry with him the germs of the disease, so that not only he himself afterwards falls ill with

it, but others are attacked in their turn. One suggestion is that a contagious principle is given off by the sick, but that it has to undergo some transformation or intermediate stage of its development, possibly in another animal, before it can infect a human being. I find it stated, on the authority of Mr Ferguson, Veterinary Officer to the Privy Council in Ireland, that on each occasion when the disease has prevailed in that country, it has coexisted with an epizootic of the same nature among pigs and dogs.

On the other hand, many observers think that epidemic meningitis is dependent upon some mysterious atmospheric or telluric influence, capable of manifesting itself simultaneously in places far apart from one another. Thus Dr Sanderson reports that in 1865 it broke out on or about January 15th in two districts of the department of Dantzic, distant at least thirty miles from each other. So, again, Stillé insists on its having repeatedly prevailed in Europe and in America in the same years, and on the way in which it has made its appearance within the United States at places hundreds of miles apart. In this respect he compares it with influenza, and defines it as a pandemic disease. But I certainly think he goes too far when he hints that the very small local epidemics which have occurred in England, and even some scattered cases of meningitis that happen to have been recorded in London at a time when cerebro-spinal fever existed on the continent of Europe, may have been due to one and the same widely diffused cause.

I have already mentioned some of the facts which show that marsh miasm is not the cause of epidemic meningitis. Ziemssen further remarks that the districts in which this disease has prevailed have often been dry, sandy elevated plateaus; and Hirsch, that it differs from ague in being especially apt to occur in the winter and spring. Yet another point of distinction is its tendency to attack children rather than adults.

Diagnosis.—This is seldom difficult. At the commencement of an outbreak, however, it would seem that enteric fever may sometimes be mistaken for it. Leyden says that among the German troops before Paris in 1870 a series of cases occurred in which marked rigidity of the neck, severe headache, and hyperæsthesia were present, while the abdomen was flat, the temperature was low, and the bowels were confined. At first it was doubtful whether the disease was not meningitis, but the autopsies showed that it was really typhoid fever, with but slight implication of the intestine. Epidemics of the two diseases may also prevail simultaneously. Leyden has seen this several times; he insists on the facial herpes as distinctive, since it is never observed in enteric fever.

When occurring as a complication of acute pneumonia, epidemic meningitis may be far from easy of diagnosis. Ziemssen remarks that rigidity of the neck is often entirely absent in such cases. In very young children, too, this symptom possesses very little significance even when it is present; and convulsions, coma, and other signs of cerebral disturbance are equally without value. Maurer maintains that a tense projecting fontanelle affords great help, since it proves that the intracranial pressure is excessive, and this is not likely to be the result of simple pneumonia.

Again, if an isolated case of meningitis should occur, it is often impossible to say whether it belongs to the epidemic, the simple, or to the tubercular form. The question may be of the gravest importance, if cerebro-spinal fever should happen to be raging in a neighbouring town or district. It would seem that the spinal symptoms—the painful stiffness of the neck, the

rigidity of the vertebræ, the hyperæsthesia, and pains in the limbs—are generally less marked in the non-epidemic varieties. Tubercular meningitis may often be distinguished by its prodromata, by its gradual onset, by its slow and interrupted course. The presence of a roseolous or purpuric eruption would probably be a conclusive proof that the case was one of cerebro-spinal fever.

The *mortality* of cerebro-spinal fever appears to vary in different epidemics from 30 to 70 per cent.; the mean mortality is estimated at 40 per cent.

In giving a *prognosis* it is important to bear in mind the treacherous character of the disease. A case which at first appears to be of but little severity may afterwards develop dangerous symptoms and prove rapidly fatal; while, on the other hand, patients whose condition had seemed hopeless sometimes recover.

Acute Simple Meningitis.—Some modern writers describe several forms of acute simple inflammation of the membranes of the brain under distinct names, according as the convexity or the base is affected, and according as the disease is more or less distinctly traceable to different causes. And, undoubtedly, a meningitis in which the whole surface of the brain becomes covered with a thick layer of pus is attended with much more violent symptoms and destroys life far more quickly than one which is limited to a particular region, and in which only a small quantity of lymph is effused. But no complete division into separate categories is possible; as indeed one might have expected, from the analogy afforded by the similar affections of other parts.

Anatomy.—When the most intense degree of inflammation has been present, one finds, on turning back the dura mater, that the cerebral convolutions are completely hidden by a green purulent material. This looks as if it were spread out over the hemisphere in the *subdural* space; but by scraping the arachnoid surface one generally discovers that little, if any, of it is really free. Moxon says that even when some of the exudation had appeared to come off upon the edge of the scalpel, he often found only epithelium and detritus on examining it with the microscope. Sometimes, however, a considerable quantity of pus is sub-dural; this, I believe, proves that the disease was due to extension from without. The converse, however, is very far from being true; namely, that meningitis is always attended with exudation into the "arachnoid cavity," when it is set up by such causes as caries of the petrous bone or necrosis of the calvaria. For, on the contrary, in the vast majority of these cases no subdural pus is found; and whenever it is widely diffused over the hemispheres I believe that it is always present in great abundance within the meshes of the pia mater as well.

The investing membrane of the brain, with all its processes into sulci, may in fact be swollen to many times the normal thickness, appearing either soft, green, and gelatinous, or firm, yellow, and felt-like, according to the consistence of the exudation infiltrated into its substance. And from such a condition there may be every gradation, down to a point at which only slight traces of pus can be discovered, along some of the principal vessels, or at which the inflammatory process is limited to the base or even to one Sylvian fissure, or to a single lobe of the cerebellum.

In some of the more severe cases the convolutions themselves are softened. When the pia mater is stripped off it carries with it portions of the cerebral

tissue, leaving a ragged surface behind. Huguenin says that numerous leucocytes may be found throughout all the layers of the cortex, and that suppuration of the brain-substance may actually reach such a point as to give rise to a diffused yellow-grey maceration visible to the naked eye. The vessels often contain but little blood in consequence of the increased pressure which precedes death. The ventricles may either be empty or contain a turbid liquid, or even pus. The choroid plexuses are sometimes infiltrated with inflammatory products.

Some of the sinuses in the dura mater are now and then found plugged with ante-mortem clots, even when the meningitis is not secondary to any disease of the bones which could itself have set up the thrombosis by extension. In a little girl, aged three, for example, who died in Guy's Hospital, and in whom both hemispheres were covered with a thick layer of yellow lymph, each lateral sinus, but especially the left, was filled with a greenish softening material.

How often an acute simple meningitis of the brain spreads to the membranes of the spinal cord it is probably impossible at the present time to say. In the majority of recorded cases the vertebral canal has not been opened, but we have had at Guy's Hospital at least eleven cases in which inflammatory products have been found in greater or less quantity beneath the spinal arachnoid. In none of them was there any reason to believe that the disease was of epidemic origin; in four it was directly caused by fracture of the skull or severe injury to the brain, or arose by extension of mischief from the cranial bones.

Ætiology.—The causes of simple acute meningitis are various. Most frequent among them are *injuries to the head.* These generally come under the notice of the surgeon, but the physician must remember that the disease sometimes follows a blow or fall which may not have produced any external bruise, and about which the patient may say nothing, particularly if he should have been intoxicated at the time. Some years ago a man was admitted into Guy's Hospital for a fractured thigh, caused by his having fallen into a cellar while drunk. He died at the end of five weeks, having been delirious all the time. He was believed to have delirium tremens; nothing was known of any injury to the head; he had been able to get out of bed and stand upright. At the autopsy it was found that there was general acute meningitis affecting the base as well as the surface; a small part of one parietal bone, over an area an inch in diameter, was of a greenish colour, and its diploë was reddened. Both the periosteum and the dura mater seemed quite healthy, but it was thought probable that the bone had been injured at the time of the accident. Another common cause of meningitis is the extension of inflammation from *chronic disease of the skull.* Syphilitic caries or necrosis of the calvaria sometimes kills in that way, and such a result may be altogether unexpected, the patient's progress having apparently been favourable until a very short time before his death. So, again, with lesions of the temporal bone secondary to affections of the ear. Six cases of this kind have been observed at Guy's Hospital within the last few years, a much smaller number than that of the cases in which cerebral abscesses were due to the same cause, and in two or three of them the pathology is perhaps still open to doubt. One patient, a man, aged twenty-two, was said to have had sunstroke seven days before being attacked, and another man, aged twenty, had been struck on the head with a bolster immediately before dangerous cerebral symptoms set in. In each

instance it was clear that the inflammation started from a diseased temporal bone, but there was a question whether an independent "exciting" cause was not in operation. On the other hand, there was the case of a woman-servant, aged twenty-seven, of whose illness no history could be obtained; as no cause for the meningitis of which she died was discoverable, the ears were specially examined by the late Mr Hinton, who found pus in the labyrinth on one side. To this accordingly the disease was attributed, but it must be remembered that in epidemic cerebro-spinal fever inflammation often spreads to the ears from the membranes, and there seems to be no reason why the same thing should not occur in the simple form of the disease.

Numerous other local affections may be mentioned as occasionally giving rise to meningitis. *Local pyæmia* from carbuncle of the face may cause it (cf. *supra*, p. 637). In one case an epithelioma, commencing in the lip, extended with ulceration along the pterygoid region until it passed through the foramen ovale into the interior of the skull. In another, a little girl had necrosis of the upper jaw after measles, and the cavernous sinus was full of a dirty-brown fluid. Or the disease may be set up by suppuration of the eyeball, travelling, in all probability, along the sheath of the optic nerve. Trousseau relates a case in which its starting-point was a chronic affection of the first two cervical vertebræ.

Next in frequency to the cases of meningitis that are traceable to local causes, come those which are secondary to some other acute disease, and which are therefore classified by Huguenin as *metastatic*. It is indeed possible that more careful research may hereafter show that these have themselves a *local* starting-point in some lesion affecting the cerebral vessels.

At Guy's Hospital we have had two cases in which inflammation of the pia mater accompanied an attack of *erysipelas* of the scalp; in one it is noted that the calvaria was discoloured yellow. In four instances it appeared to be part of general *pyæmia;* once there was a grey patch of cerebritis reaching the surface; once pericarditis also was present, so that one might regard the disease as specially attacking the serous membranes if the arachnoid could still be called by that name; once the meningitis was the only evidence of blood-poisoning, but such seemed to be the most probable explanation of its occurrence, as it came on six days after an operation for imperforate anus in a child a year old; once it was associated with an abscess in the lung, and with suppuration in the mediastinal connective tissue and in the substance of one leg, there being, however, no obvious primary lesion. In a single case, inflammation of the cerebral membranes was believed to be a complication of *typhus;* no affection of the vessels in the pia mater was discovered, but the spleen contained infarcts, and there was thrombosis of the renal veins. Four times it seemed to be secondary to acute *pneumonia* or pleurisy; all but one of these cases, however, presented peculiar features. One occurred in a man, aged twenty-two, who had been in the hospital six weeks for pleuro-pneumonia, and was convalescent and able to go into the grounds, when he was again attacked with dyspnœa and high fever, and died in three days; the spinal membranes, as well as those of the brain, were inflamed. Another was in a woman, aged twenty-seven, who had been ill for a week with pleurisy and then was attacked with an acute diphtheritic affection of the fauces, and died a day later, having been very restless towards the last and having one pupil wider than the other; in her, also, the affection was cerebro-spinal. The third case was that of a man, aged twenty, who had acute meningitis involving both hemispheres of the brain, and

pleuro-pneumonia of the lower lobe of the left lung; he was suffering from gonorrhœa, and Dr Wilks was inclined to regard this as the cause of his fatal illness, but it was further said that he had had a blow on the head a few days before he was attacked. In the remaining case the patient was a temperate man, aged fifty-seven, who died, after sixteen days' illness, of acute pleuro-pneumonia affecting the left lower lobe; no cerebral symptoms appear to have been observed beyond delirium before death. There was much recent lymph both at the base of the brain and on the vertex. The only other morbid conditions that were discovered were chronic renal disease (apparently in moderate degree) and hypertrophy of the heart.

Huguenin says that at Zürich inflammation of the cerebral membranes is an exceedingly frequent complication of acute *pneumonia*. He quotes Chvostek as having found it four times in 220 cases in Vienna, and adds that in Zurich the percentage is higher.

Dr Moxon laid special stress on the fact that *syphilis* sometimes seems to be the cause of an acute simple meningitis, and I find five cases of this kind in our records, in addition to those in which inflammation of the membranes was set up by disease of the calvaria. Once a gumma was discovered in the brain-substance. In no instance was any disease noticed in the cerebral arteries, but it seems very probable that such a lesion may hereafter be discovered in similar cases. In 1871 a man, already in the hospital for disease of the aortic valves with regurgitation, died after two days' illness of cerebro-spinal meningitis; no embolism was detected, but the spleen contained infarcts. In 1874 a woman, aged forty-eight, who had been admitted for chronic jaundice caused by biliary calculi, was attacked by a fatal inflammation of the cerebral membranes; this, however, was doubtless consequent upon ulcerative endocarditis of the aortic valves, which was discovered at the autopsy. It is remarkable that I do not find in our records a single case in which Bright's disease appeared to be the only cause of meningitis. Huguenin, however, mentions it as having occurred consecutively to acute pleurisy in a girl, aged fourteen, who had "acute fatty degeneration of the kidneys."

There remain a few cases in which meningitis can be traced to none of the causes mentioned, and must be called *idiopathic*. In our records at Guy's Hospital I find nine cases of this kind. In three of them pus or lymph was spread out in large quantities over the whole surface of the brain, equally over the hemispheres and at the base; in three the amount at the base was much less than upon the convexity: in one the latter region was alone affected, the material being in this instance lymph. Once the under surface of the brain and the subarachnoid space of the spinal cord were all bathed in pus, there being but little upon the hemispheres. In the two remaining cases the distribution of the inflammatory products was the same as in ordinary examples of tubercular meningitis. In every instance the cause of the disease remained a mystery, but sometimes meningitis of the convexity is set up by the action of the sun's rays upon the head. Huguenin speaks of labourers who have been attacked while working bare-headed in the fields, and he gives full details of the case of a sailor who fell ill the day after he had been rowing without anything upon his head in the hottest weather of July, and who died in five days. Another case related by the same writer is that of a student who was foolish enough to spend three hours, from 1 to 4 p.m., during a hot summer's afternoon, paddling about the lake of Zürich, lying flat on his belly on a board. All the exposed

parts of his body became severely sunburnt. At 6 p.m. he was seized with headache, and an hour later with shivering, and all the symptoms of meningitis rapidly developed themselves. His illness, however, terminated in recovery. Guersant met with a similar instance which ended fatally, and was verified by an autopsy, in an infant, aged six months, whose cradle had been left in the sunshine in the middle of a garden. Rilliet and Barthez mention the case of a child who manifested alarming cerebral symptoms after reading a book, the pages of which were exposed to the sun.

Course.—The symptoms of acute simple meningitis bear a general resemblance to those of the tubercular affection; in the typical forms of both there is a stage of "irritation," which is succeeded by a stage of "pressure." But the more severe cases of the one present a peculiarity that is, I believe, without parallel in the other disease, namely, that each of the two periods, although marked by the most characteristic symptoms, may yet terminate within two or three days, or even last no longer than a few hours. Thus an effective contrast can be drawn if one describes several distinct non-tubercular varieties of meningitis, but, as I have already stated, I think that such an arrangement is artificial.

Simple meningitis commonly begins quite suddenly; it has no prodromata. The patient, if an adult, may be seized with a rigor; in children this is less common. Headache is generally present from the first; it may either be referred to the forehead, or affect all parts alike. There may be exacerbations from time to time, in which piercing cries are uttered; the agony appears to be altogether intolerable. Giddiness is frequently an early symptom; the ground seems to give way beneath the feet, and the legs may feel so weak that to stand upright is almost impossible. Vomiting, perhaps, occurs once or oftener. There is an extreme irritability to light and sound. The eyes are brilliant and injected, the face is flushed, the head is hot, the carotids are felt to throb violently. The temperature rises, and may reach 104° by the third day; there may be great complaint of thirst. The pulse is quickened, as a rule, but this is not always the case. Epileptiform convulsions are not uncommon. and sometimes mark the commencement of the disease. The back of the neck is often rigid, so that the patient seems to bore his head into the pillow. Sometimes his limbs are stiff. He generally lies coiled up in bed, anxiously avoiding notice, and most unwilling to be disturbed in any way. His mind may for a time be perfectly clear, but afterwards he becomes delirious. In some cases there is violent maniacal excitement from the very first. Sleep is altogether wanting, or very broken and disturbed. The pupils are, as a rule, contracted; the ophthalmoscope may show either ischæmia or neuro-retinitis.

The second stage of the disease is characterised by stupor, which more or less quickly passes into coma. The pupils are sluggish, or even dilated and insensible; they are often unequal in size. There may be loss of power in the limbs of one side with or without a similar affection of the corresponding half of the face. Sometimes the patient squints, but even when the base is involved one can seldom make out a definite paralysis of any of the cranial nerves; they of course remain unaffected when the inflammation is limited to the convexity of the brain. Epileptiform convulsions may return again and again until one of them proves fatal, or they may be altogether absent, or, again, there may be attacks of spasm confined to certain muscles or to one arm and one leg. The temperature generally remains high, ranging from

102° upwards, but the face is now pale, and the extremities may be cold to the touch and bathed in a profuse sweat. Towards the last the evacuations are passed involuntarily.

The duration of simple meningitis varies with its intensity. Those cases in which the whole surface of the brain, including the convexity, is inflamed scarcely ever last more than a week, and generally terminate within three or four days. When the base alone is affected the patient sometimes lives as long as in the tubercular form of the disease. But this is by no means always the case; he may die in forty-eight hours, and the only discoverable morbid change may be on the under surface of the brain. In such cases, however, there is no successive development of the symptoms in regular order. Sometimes the patient is attacked with epileptiform convulsions and becomes quickly insensible, or he may be violently delirious. In certain cases loss of spirits is the earliest symptom, as was noticed long ago by Abercrombie. There may be little or no pain in the head, and thus the real nature of the disease may be altogether overlooked.

Diagnosis.—Simple inflammation of the membranes of the brain may be difficult to recognise, as we have already seen to be the case with tubercular meningitis; but the diseases most likely to lead one into error are not quite the same. When it is impossible to make out the presence of any of the recognised causes of the affection, and when the symptoms are obscure, one may be unable to distinguish it from alcoholism, mania, or epilepsy. On the other hand, when the patient is known to be suffering from acute pneumonia or erysipelas it may be very hard to determine whether there is more than the ordinary symptomatic delirium. Cases in which alarming indications of cerebral disturbance are traceable to otorrhœa seem to be peculiarly liable to be wrongly interpreted. In 1877 I made an autopsy in the case of a patient of Dr Frederick Taylor's, who had died with what appeared to be clear symptoms of meningitis secondary to disease of the ear, including convergent strabismus, a swollen œdematous condition of both optic discs, delirium, and coma. The only sign of any disease of the encephalon was a blackened state of the arachnoid over a small part of one lobe of the cerebellum, but there was a putrid thrombosis of the lateral sinus and jugular vein, and this had set up numerous pyæmic abscesses in the lungs. In the 'Med. Times and Gazette' for 1877 will be found a precisely similar case, under Dr Wilson Fox, in which the patient had all her limbs flexed, and suffered from headache, photophobia, and hyperæsthesia of the surface, but in which the brain and its membranes were perfectly healthy. It would therefore appear that pyæmia dependent upon thrombosis of a lateral sinus is capable of simulating meningitis rather closely. The point is of the more importance in that the former disease seems not to be always fatal; at least this appears to me to be the most probable interpretation of two cases recorded by Dr Andrew in the 'Med. Times and Gazette' for 1875. One was that of a youth, aged sixteen, who had had a discharge from the ear, and who became drowsy and heavy, with frontal headache, giddiness, sickness, and blurring and œdema of the optic discs. The other occurred in a girl, aged nine, who had also had ear disease, and who was attacked with pain in the head, vomiting, and delirium, so that she screamed and started in her sleep. Meningitis was diagnosed, but each patient recovered after an illness of some weeks' duration, in the course of which there was distinct evidence of pyæmia, one of them having pneumonia, the other having an abscess in the thigh.

Lastly, I believe that there is always an element of uncertainty in regard to the diagnosis of those cases in which the typical symptoms of meningitis develop themselves in regular order, and with such rapidity as to threaten a fatal termination within three or four days, or even still earlier. I have seen more than one instance of this kind, in which the gravest prognosis was given, but in which a complete recovery took place. A case which comes forcibly to my recollection is that of a young lady, the daughter of a medical man. Her education had been rather neglected during childhood, and she had been for some weeks straining all her powers to keep pace with her schoolfellows in her studies. I have unfortunately no notes of her symptoms, but I know that they appeared to point clearly to the presence of meningitis; yet the attack passed off, and she is now in good health. I have cited a case of Huguenin's, in which the diagnosis was adhered to, in spite of the fact that the patient recovered.

Prognosis.—The question as to the possibility of recovery from acute simple meningitis is still more definitely raised by certain cases which come under the notice of ophthalmic or of aural surgeons. In 1866 Mr Hutchinson recorded in the 'Ophthalmic Hospital Reports' a series of cases of children who were brought to him for blindness, which he found to be due to optic neuritis, and nearly all of whom had had a severe illness, attended with delirium and other cerebral symptoms, and supposed to be fever. Dr Allbutt suggests that the disease was really meningitis. This observer also states that he has seen several instances in which a condition of defective mental development (or idiocy as it would be called) has been associated with atrophy of the optic discs, and has in all probability been referable to a long past inflammation of the cerebral membranes. On the other hand, certain cases in which a permanent deafness has resulted from an acute illness have been interpreted in a very different way by Voltolini (who first drew attention to them in 1867) as well as by some other writers. According to Brunner (of Zürich) the affection in question commonly sets in suddenly; the child suffers from fever; it may vomit; its head is hot, it becomes excited and delirious, tossing about in bed and screaming violently; within the first twenty-four hours it becomes partially unconscious, and after two to four days it passes into a state of coma. At the end of another period of two to four days, however, it regains its senses very rapidly; but when it first tries to walk it is found to stagger, and it quickly becomes deaf and (as a consequence) remains dumb for the rest of its life. Voltolini's theory is that the disease in such cases is an acute inflammation of the labyrinth; the cerebral symptoms (as in Ménière's disease) being supposed to be caused in some way by the aural affection. Direct evidence from the *post-mortem* room is as yet altogether wanting. But, as Brunner remarks, the fact that the deafness is constantly bilateral is opposed to such an interpretation. He thinks that there may be some morbid change on the floor of the fourth ventricle, implicating the *striæ acusticæ*. But it seems to me that Voltolini's cases and those of Mr. Hutchinson and Dr Allbutt must be taken together, and in connection with the other cases in which recovery takes place after an illness resembling meningitis, but in which there is not left either deafness or blindness or idiocy.

What makes me hesitate in coming to any conclusion as to the real nature of such causes is the circumstance that one scarcely ever finds in the *post-mortem* room adhesions of the membranes at the base of the brain or other clear evidences of a former meningitis. It has, however, been shown

that in epidemic meningitis recovery is not infrequent and that the disease often leaves behind it deafness or blindness; in several cases the remains of inflammatory exudation have been discovered when the disease has run into chronic hydrocephalus, which has proved fatal a few months later. These observations might at first sight appear to settle the question, but it may be objected that there is a fundamental difference in the fact that simple meningitis does not give rise to any similar form of chronic hydrocephalus, and further, that at present nothing is really known as to the morbid anatomy of those cases of epidemic meningitis in which complete recovery takes place. Such cases are undoubtedly due to the same cause which gives rise to "cerebro-spinal fever" in general, and so are properly included under the synonymous term of "epidemic meningitis;" but it has not yet been ascertained that they are attended with an actual visible inflammatory change in the membranes, and as regards simple meningitis it is impossible for us to take an equally broad view, and to waive the production of direct pathological evidence.

After all, however, the really important point is the practical one, namely, that cases which appear to be well-marked examples of inflammation of the membranes of the brain do not always terminate fatally. Until increase of clinical experience or the introduction of new methods of investigation shall have rendered this statement incorrect, no explanation of the real nature of the cases in question, however complete and however convincing, can deprive it of its significance.

Thus in a case of meningitis one should never entirely lose heart; scarcely any is so hopeless as to justify one's abandoning it altogether—particularly if the patient is a child.

Treatment.—At an early stage one important object is to relieve the headache and to diminish the cerebral excitement. The patient should be placed in a cool, dark, well-ventilated room, and should be kept perfectly quiet. The hair should be cut short, or shaved. Cold should be applied to the head, and for this purpose a large bladder containing small pieces of ice and a little water is more serviceable than anything else; evaporating lotions are far less effective and require very frequent renewal.

In 1878 I saw a case which appeared clearly to demonstrate the usefulness of the local application of ice. A man, aged forty-three, a baker, was taken with a kind of fit on the evening of the 26th of June, and was seen the same night by Dr Churchward with a temperature of 104·6°, a pulse of 160, a very flushed, hot face, vomiting, and great pain in the head and neck, as well as in the back and in the limbs. He had been driving about all day in a hot sun; three days previously he had stood for some hours in water up to his knees in consequence of his cellars having been flooded by a heavy rainfall; for more than a month he had been unusually irritable in his temper. For a day or two he seemed to improve, but during the night of the 28th he became collapsed and almost pulseless, with a cold clammy sweat. After this he remained sleepless and was very delirious, with contracted pupils and constant twitching of the hands. On the 30th I saw him and found the optic discs normal; his symptoms were then of the most alarming character. We prescribed a mixture containing the iodide and the bromide of potassium, and a draught of chloral and morphia in the hope of inducing sleep. Next day (July 1st) he appeared to be rather worse than better; he had not slept for more than five minutes; he had been sick again; he was constantly talking and picking

the bedclothes. Dr Churchward therefore ordered a towel to be wrung out of iced water, and to be kept applied all over his head and neck with lumps of ice between the folds. The effect seemed to be magical; in a few hours he became quiet and fell asleep; the sickness and the clammy perspirations ceased. He liked the cold to his head, and during the night he slept for six hours. On the following day (July 2nd) he was rational, and told Dr Churchward that he remembered nothing since the 29th June, except that another doctor had examined his eyes. The ice was continued until July 4th. His recovery was uninterrupted; on the 7th he got upon the sofa; and by the 11th he was able to be drawn out in a carriage. This patient died in the spring of 1880 of acute pneumonia. Dr Churchward obtained for me permission to examine his brain. There was no conspicuous change, nothing, I think, that would have attracted my notice if I had not known of his former illness. But the arachnoid and pia mater at the base of the brain appeared to me to be thicker and crisper than natural, especially over the right Sylvian fissure; the olfactory lobes were more firmly bound down; and there was more connective tissue on the under surface of the pons. The velum interpositum also seemed to be increased in density, although the lateral ventricles were not dilated.

In epidemic meningitis, Ziemssen and other German physicians keep ice-bags to the head, back, and neck for weeks togther, and find that they afford marked relief to the patient's sufferings, soothing him and enabling him to sleep.

Most physicians abstain from administering anodynes in cases of meningitis, as being likely to mask the symptoms; but at an early period of the disease this objection seems unfounded, and Dr Bristowe says that he has frequently given opium with manifest relief. Huguenin recommends the subcutaneous injection of morphia in small doses, and the use of enemata containing fifteen to forty-five grains of chloral. Ziemssen says that in epidemic meningitis remedies of this class are indispensable.

To check vomiting the patient may have little pieces of ice to suck; or bismuth, hydrocyanic acid, and similar remedies may be prescribed.

There is perhaps no reason to suppose that leeches, or venæsection, or the most active purging could cut short a meningitis which is going on to the effusion of lymph and pus. But is it equally certain that such measures are useless in those cases which are less severe? In epidemic meningitis, at any rate, German authorities now recommend all those "antiphlogistic" measures which were in vogue in this country thirty years ago. They not only apply leeches behind the ears and cupping glasses to the spine; in the most acute cases, or when the patient is very restless, they employ venæsection; and they administer calomel, or rub in blue ointment, at the same time washing out the mouth with solution of chlorate of potass, to prevent the gums from being affected by the mercury.

In simple meningitis, the older English physicians believed that they had seen successful results from energetic treatment. Some of Abercrombie's cases will perhaps still bear quotation. "A girl, aged eleven" (Case 69) "had violent headache and vomiting, with great obstinacy of the bowels, and these symptoms were followed by dilated pupils and a degree of stupor bordering on perfect coma; pulse 130. She had been ill five or six days; purgatives, blistering, and mercury to salivation had been employed without benefit. One bleeding from the arm gave an immediate turn to this case; the headache was relieved; the pulse came down; the vomiting ceased; the

bowels were freely acted upon by the medicines which they had previously resisted; and in a few days she was quite well." "A gentleman, aged twenty-one" (Case 72) "was first affected with confusion of thought and very considerable loss of recollection. He then complained of headache, and after a day or two had double vision. At this time he was out of bed the greater part of the day, but was restless and confused, and at times incoherent. He was then confined to bed, and had constant headache, much incoherence and oppression, the double vision continuing. The pulse was at first frequent, but fell gradually and sank below the natural standard; and the symptoms went through a course exactly similar to that which has been described in many of the fatal cases. As the pulse fell in frequency, he became more and more oppressed, until he sank into a state of stupor, from which he could scarcely be roused to answer a question of the most simple kind. The case went on in this manner for eight or ten days, during which time he was treated by repeated general and topical bleeding, cold applications, blistering, &c. The bowels were very obstinate, and large doses of the most active purgatives were given with little effect. The case was considered desperate, when he began to take croton-oil in full doses, repeated every two or three hours. In a few hours he was purged very actively nine or ten times; the same evening he was relieved from every alarming symptom, and in a few days he was free from complaint." "A girl, aged seven" (Case 75) "had severe headache, impatience of light, fever and slight delirium, followed by stupor, squinting, and great obstinacy of the bowels. The tongue was at first foul, but became clean after a day or two. She was considered as being in a hopeless state of hydrocephalus. At the end of a week, strong purging being produced, she recovered rapidly, and in a few days was free from complaint." It is, no doubt, possible that the favourable issue of these cases was spontaneous, and would have occurred independently of all active treatment. But, on the other hand, it may be that under the negative practice of the present day they would have terminated fatally.

Of course it is essential that our treatment should do no harm. When there is any doubt of the diagnosis between enteric fever and tubercular meningitis, it would be very wrong to purge. Other cases, in which uncertainty of diagnosis ought to influence our practice, are those of probable meningitis secondary to disease of the ear. This form of the disease is almost certainly hopeless, but we have already seen that pyæmia may simulate it very closely.

Again, in adult patients it is of the utmost importance that we should not overlook the possibility that syphilis may be the cause of symptoms resembling those of meningitis. We have seen that it may give rise to inflammation of the membranes of the brain, but it does not follow that syphilitic meningitis would be especially amenable to treatment. The point is rather that one may be called in to see a man who is insensible, and that the history of the case may seem to point to meningitis, but that the lesion may really be one of the more direct results of syphilis. It would then be cruel to ask of a wife, or of a mother, questions which might leave the most painful impressions, and which, if answered negatively, could never decide the matter. But the most careful search should be made for evidence of syphilis, such as the presence of nodes or gummata, and the recurrence of miscarriages or sterility on the part of the wife. If there is any reason to suppose that this cause may be in operation, full doses of

the bichloride of mercury or of the iodide of potassium should be given. If this treatment is unsuccessful it can do no harm, and its success is sometimes very great. Whether it is advisable to prescribe iodide of potassium or mercurials (except in purgative doses), in cases which are believed to be idiopathic meningitis, is very doubtful.

As to the stage when coma sets in, Sir Thomas Watson says that in his experience the patient has sometimes recovered consciousness after a cap of blistering plaster has been put upon his head. He does not tell us whether the improvement was permanent. Huguenin speaks in high terms of the value of energetic cold affusion for the same purpose. But it seems possible that to excite a merely temporary return of consciousness might be injurious instead of beneficial. The application of mustard plasters to the calves of the legs must surely be altogether futile. Our endeavour should rather be to sustain life from hour to hour, in the hope that the tide may turn. Sir Thomas Watson remarks that "patients apparently moribund are occasionally saved by the judicious administration of stimulants and restoratives, of ammonia, Hoffmann's anodyne, beef-tea, wine, and (it may be) of well-timed opiates."

In those cases in which recovery takes place it is perhaps advisable to give iodide of potassium during convalescence, and a good supply of nourishing food is then essential.

Adhesive Thrombosis of the Cerebral Sinuses.—Another affection of the membranes of the brain—thrombosis of the sinuses—may conveniently be described in this place, although pathologically it is remote from meningitis. We have already spoken of cases in which pyæmia results from a putrid inflammation of the great intracranial venous channels, itself secondary to disease of the temporal bone, facial carbuncle, or injury to the skull, and attended with the formation of a thrombus that rapidly soften downs into a puriform liquid. But the affection now to be discussed is primary, gives rise to no general infection of the blood, and consists in a simple plugging of one or more of the sinuses with a firm clot, which becomes closely adherent, has sometimes a laminated structure, and tends to undergo organisation into permanent tissue. Von Dusch and Gerhardt are the writers to whom we are most indebted for our knowledge of it. It is, I believe, of very infrequent occurrence; at least, I find only one example of it in our pathological records at Guy's Hospital.

Its most usual seat is the longitudinal sinus; sometimes it extends into the tributary veins, so that the hemispheres appear to be covered with coiling worm-like bodies; sometimes it is prolonged into one of the lateral sinuses. In some instances hæmorrhage in the pia seems to have occurred as a consequence of this affection, in others the substance of the brain is said to have been found ecchymosed or softened; but it appears doubtful whether those cases have been correctly interpreted.

There are certain conditions under which this form of thrombosis is especially apt to occur, and which, therefore, may suggest a suspicion of its presence. It has been observed in ill-nourished infants, six months or a year old, who have suffered severely from diarrhœa for some weeks before their death. Such cases generally resemble those of the "spurious hydrocephalus," described at p. 687, but with the addition of some more definite cerebral symptoms, such as nystagmus, squinting, ptosis, facial paralysis, and especially rigidity of the neck, back, or limbs. The fontanelle is generally

sunken, but in one of Gerhardt's cases it filled out and became tense towards the last.

In adults, adhesive thrombosis of the sinuses seems sometimes to occur spontaneously; sometimes it is consecutive to enteric fever, or parturition, especially when much blood has been lost. Accordingly this affection has been described as "Thrombosis from Marasmus," to distinguish it from infective or pyæmic thrombosis.

This rare disease is liable to be mistaken for several others which are comparatively common; namely, for the less acute form of meningitis, for tumours and other lesions limited to the upper parts of the hemispheres, or for the diffused affections of the brain which were described in the last chapter.

Indeed, the clinical recognition of plastic or adhesive thrombosis of a cerebral sinus is rarely possible. Some writers have described engorgement of veins running from the anterior fontanelle of an infant to the neighbourhood of the temples and ears, and the occurrence of epistaxis, as signs of plugging of the longitudinal sinus; while they have supposed that œdema over the mastoid process pointed to obstruction in the corresponding lateral sinus.

A curious case is reported by Dr Hyslop from the West Riding Asylum, of a woman suffering from acute mania, who lost all her symptoms for a period coincident with the appearance of hæmatoma aurium and œdema of the mastoid region and of the orbits. Temporary plugging of the sinuses was diagnosed ('Brain,' April, 1886, p. 90).

The duration of the thrombosis is variable; it may last several weeks, or death may occur a few days after its commencement, so far as this can be determined from the clinical history of the case.

Its treatment would probably consist in keeping the patient in a state of perfect quietude, and perhaps in administering ammonia.

HÆMATOMA OF THE DURA MATER.*—There still remains to be described an affection of the cerebral membranes which, having for many years been a puzzle to morbid anatomists, has recently become a subject of clinical interest also. It consists in the presence of one or more membranous layers on the inner surface of the dura mater, within the so-called arachnoid cavity. When recent they are soft and vascular, but in course of time they may become tough, white, and fibrous, so as to resemble in appearance the dura mater itself. Virchow says that in one instance he counted no less than six or seven of them lying one upon the other. They commonly extend over the greater part of a hemisphere, and at their margins they adhere together so as to form a closed sac, or a series of sacs, which are generally attached much more firmly to the inner face of the dura mater than to the arachnoid surface of the pia mater. In consequence they were formerly supposed to be formed by a separation of the imaginary parietal layer of the arachnoid from the dura mater, but this notion has long ago been refuted. Indeed, the cyst is sometimes perfectly unattached, so that it falls out as soon as the fibrous covering of the brain is cut through. In a large proportion of cases (50 per cent. according to Huguenin) the affection is bilateral, a separate hæmatoma concealing and flattening the convolutions of each hemisphere. A considerable quantity of blood, either recent or tawny-brown with age, is commonly found between the layers, which themselves are often deeply stained with hæmatoidin. But sometimes that which fills

* *Synonym.*—Pachymeningitis interna hæmorrhagica.

the adventitious cavity, or cavities, is a thin serous fluid which may contain a quantity of cholesterine; it is believed that such cases are the only examples of what was formerly described as *Hydrocephalus externus.*

In 1845, Mr Prescott Hewett, in a paper read before the Royal Medical and Chirurgical Society, maintained the opinion, which had before been promulgated by Houssard and Baillarger in France, that the starting-point of this disease is an effusion of blood. But a few years afterwards Virchow gave the powerful support of his authority to a very different doctrine (previously upheld by Bayle), namely, that the earliest morbid change is an inflammation of the dura mater; this, he supposed, becomes hyperæmic, and exudes upon its under surface a delicate material, richly supplied with wide, thin-walled vessels, which rupture and yield the blood that is so commonly found extravasated. Virchow's view has been adopted by most of those who have since written on the subject; and his name, "pachymeningitis hæmorrhagica," has met with very general acceptance.

Huguenin has, however, reverted to the theory that the disease begins as a hæmorrhage. If his observations are correct, one can hardly escape from the conclusions which he draws from them; and in any case the best designation for it seems to be the old one of "hæmatoma of the dura mater," which leaves the question of its origin open. In a large majority of instances the affection is merely a complication of cerebral atrophy, whether senile, or due to chronic alcoholism, or associated with general paralysis of the insane. Now, Huguenin states that in a number of cases of general paralysis he has been able to trace what he believes to have been the earliest stage of a hæmatoma, in the presence of a soft layer of blood-clot, spread out over the convolutions, having its greatest thickness (2 mm.) opposite the parietal eminence, and gradually thinning off towards its margins. This substance comes away from the dura mater in small shreds, which have no vascular connection whatever with that membrane. Moreover, the microscope shows that the clots contain at this period nothing but a network of coagulated fibrin, blood-discs, and leucocytes. Afterwards, indeed, the clot undergoes organisation, vessels are developed in it, and become continuous with those belonging to the dura mater, which now looks more or less injected. Anyone versed in the ordinary pathological doctrines of the day will have little difficulty in forming a conception of the nature of the subsequent changes, which lead to the formation of massive membranous layers, or to the accumulation of collections of blood, or of serous fluid in their midst.

The most usual seat of pachymeningitis hæmorrhagica or hæmatoma of the dura mater is over the upper surface of the hemispheres, corresponding to the dura lining the parietal bones. In more than half the recorded cases it has been bilateral.

As to the original source of the hæmorrhage, Huguenin thinks that it comes from the veins which open into the longitudinal sinus; he has noticed that these vessels are often varicose and thinned, that their coats are affected with fatty degeneration, and that they are sometimes filled with thrombi; once he actually discovered a laceration in the wall of one of the veins.

Hæmatoma of the dura mater occurs more often in males than in females. It is met with chiefly in those who are advanced in life, but exceptional cases of it may be seen at all ages; in 1864 Dr Wilks exhibited to the Pathological Society a specimen taken from a young man. It is

scarcely ever observed in the *post-mortem* room of a general hospital; Dr Wilks obtained his preparation from the dissecting room, the subject, a "half-witted" young man, having died in a workhouse.

There are enumerated among the causes of hæmatoma chronic affections of the lungs, heart, or kidneys, "pernicious" anæmia, scurvy, and hæmophilia, typhus, smallpox, and acute rheumatism. When these complaints end fatally, no doubt hæmorrhage into the membranes may sometimes be found; but it by no means follows that in cases which recover the same thing may occur so extensively as to lead to the formation of membranous layers.

Injuries to the head seem occasionally to give rise to the affection. In 1855 Dr Quain showed to the Pathological Society a specimen taken from a farmer, aged fifty-eight. He had for three years suffered from various cerebral symptoms, which dated from a fall from his cart, when he was temporarily rendered insensible and received a large scalp wound on the right side. Excessive anæmia, particularly Addison's anæmia gravis and chronic alcoholism, appear to be the best established predisposing causes. Whooping-cough is an exciting cause which can be readily understood.

The clinical course of hæmatoma of the dura mater varies widely in different cases. Sometimes the only recognisable symptoms are those of a fatal apoplectic seizure consequent on the sudden outpouring of a large quantity of fresh blood in or between membranes formed out of a coagulum of old date, which itself had in no way disturbed the patient. Very often the affection is found on *post-mortem* examinations of those who have died of general paralysis of the insane, unattended with any unusual symptoms such as to suggest the presence of a hæmatoma. It would be a great mistake to suppose that the epileptiform and other attacks which are of frequent occurrence in general paralysis are usually, if ever, traceable to that condition. Indeed, as Huguenin remarks, the wasted brains of these patients allow of the accumulation of a large quantity of blood, or of products derived from it, on their surface, without pressure. Even headache seems to be very generally absent in such cases.

However, it would seem that in some exceptional instances the affection gives rise to symptoms that may enable it to be diagnosed. Huguenin lays stress on the occurrence of two or more apoplectiform attacks separated by an interval during which the patient remains pretty well. He relates in detail the case of an intemperate man, aged thirty-one, who about two years before his death began to complain of severe headache, and who one day fell unconscious, with a slow, full pulse, contracted pupils, transitory convulsive movements of the right side, and partial paralysis of the right side of the face. After twenty-four hours he gradually recovered his senses and got up, but he continued to suffer from pain in the head, and slowly lost his memory and intelligence, and finally he had another seizure which proved fatal in four days. The brain was found atrophied, with dilated ventricles, and with a hæmatoma on each side consisting of a complete membranous sac divided into compartments. But I cannot endorse Huguenin's opinion that such a history is in any way characteristic of this affection.

Attempts have also been made to diagnose hæmatoma of the dura mater in cases which have ended in recovery. That it is possible for the affection to subside after giving rise to well-marked symptoms appears to be established by the following case recorded by Bouillon Lagrange and quoted by Huguenin in 'Ziemssen's Handbuch' (p. 402).

An old man, aged seventy-five, who was suffering from drowsiness, and who was already failing in intelligence and memory and bodily activity, had a fall from his horse. He was not injured outwardly, but he now became more deeply unconscious and had right hemiplegia. At the end of two months he was perfectly comatose and almost totally paralysed; he was unable to speak, and passed his motions under him. After this, however, he gradually began to improve, he regained his senses and his memory, he recovered the use of his limbs, and when two more months had elapsed he was considered to be cured. He remained quite well for six months, and then he was murdered. On a *post-mortem* examination, beside a recent fissure of the skull, the back part of the right hemisphere was found to be flattened by a cyst, which adhered to the visceral arachnoid and contained three or four spoonfuls of a bloody liquid.

Griesinger has even ventured to infer the presence of this affection in a man, aged fifty-seven, who recovered and was in good health at the time when his case was published. He had been a spirit-drinker, and for about five months had suffered from severe headache, extreme drowsiness, and confusion of ideas. His gait then became unsteady, his pupils were contracted, his pulse was rather irregular, and there seemed to be slight paralysis of the left facial nerve. He would sleep all day long, and passed his urine in his bed, but at the end of a month he began to improve, and in about ten days from that time he was perfectly well. Griesinger confesses not only that he expected a fatal issue, but that he originally diagnosed a rapidly-growing cerebral tumour. So little is positively known of recoveries from any organic disease of the brain, that the above observations of Griesinger and Bouillon Lagrange are well worthy of being remembered.

The best treatment for a case supposed to be one of hæmatoma would probably consist in the application of cold to the head, in the administration of purgatives, and perhaps in the abstraction of blood by leeches or by venæsection.

NEUROSES

OR

FUNCTIONAL DISORDERS OF THE NERVOUS SYSTEM

THE SPASMODIC NEUROSES

Arrangement of the Neuroses and particularly of Spasmodic Neuroses.
Facial or Histrionic Spasm—Spasmodic Wryneck.
Reflex and Functional or Movement Spasms: Saltatorial and Salaam Spasm—Scriveners' Palsy, Hammer Palsy, and other handicraft spasms.
PARALYSIS AGITANS—*Symptoms—Pathology—Ætiology—Treatment.*
THOMSEN'S DISEASE—*Its characters and symptoms.*
TETANY—*Description—Relation to Trismus neonatorum, Laryngismus, and Carpopedal contractions, and of all to Rickets—Treatment.*
CHOREA — *Nomenclature — Symptoms and course — Chorea gravis — Morbid anatomy—Statistics—Pathology and Ætiology—Relation to Rheumatism and Cardiac disease—Embolic theory—Treatment.*
TETANUS—*Symptoms—Varieties—Ætiology—Pathology—Diagnosis—Events—Acute and chronic course—Mortality and Prognosis—Treatment.*
HYDROPHOBIA—*Incubation—Premonitory symptoms—Characters when developed—Ætiology—Pathology—Diagnosis—Treatment—Prophylaxis—Pasteur's method of protective and curative inoculation.*

WE have now completed the survey of those diseases which can be more or less certainly associated with organic lesions of the nerves, the spinal cord, or the brain. There remain the Functional disorders of Motion, Sensation, and Intelligence, some of which have perhaps their special morbid anatomy yet to be discovered, while others are in all likelihood dependent, not on changes of structure which can ever be distinguished by the scalpel or the microscope, but on nutritive or molecular disorders which pervert action without altering mechanism.

We found it desirable to have regard to clinical characters even in our arrangement of organic diseases. But in our ignorance of the causes and nature of almost all functional diseases, we have no choice but provisionally to classify them by their symptoms and course. It will be convenient to treat first of Chorea and other spasmodic diseases, next of Epilepsy and other paroxysmal or convulsive attacks, and then of that singular malady Hysteria, which simulates organic disease of the brain and cord, and may present the appearance of hemiplegia or paraplegia, of convulsions, or spasms, or mania. A chapter on Insanity will come last.

The first group of diseases to be considered may be termed the Spasmodic Neuroses, for in all of them the chief symptoms are spasms or cramps in the voluntary muscles. Some of these are very closely connected together, being often found at once, or in succession, in the same patient; some are scarcely more intimately related to one another than they are to other neuroses. A convenient order will be to begin with those which are limited to a single muscle, or to a few muscles, and afterwards to pass to those which involve an entire limb, or the whole body. The following enumeration of them, with brief definitions, will perhaps be found useful.

1. *Histrionic spasm.*—A jerking movement, usually of the face, sometimes limited to a single muscle, repeated at considerable intervals without alteration for a great length of time; sometimes suddenly replaced by a similar movement of an entirely different part.

2. *Spasmodic wryneck.*—Paroxysms of clonic spasms in one side of the neck, rapidly succeeding one another, and leading to great distortion of the head; sometimes extending to the side of the face or to the corresponding arm, or passing into tonic contraction of certain cervical muscles; occasionally subsiding for a time, and recurring after the lapse of years.

3. *Writers' cramp* and analogous affections of musicians and other persons who perform skilled movements with their hands. It consists mainly in an incapacity to perform some particular action, in consequence of cramps being excited by the attempt. Closely related to these cases are spasmodic neuroses which follow less complicated movements, as Hammer Palsy, and those which depend on habitual movements not specialized by a handicraft, such as eating, speaking, and walking. Some kinds of Stammering and Saltatorial Spasm are examples.

4. *Paralysis agitans.*—An oscillatory movement, generally beginning in one of the upper limbs; for a time paroxysmal, but afterwards continuous so long as the patient is awake; at first ceasing when the affected muscles are called into voluntary action, but afterwards increased by all attempts to move; attended, in advanced cases, with some rigidity of the trunk, arms, and legs, and with a tendency to hurry in walking or to fall forwards.

5. *Thomsen's disease.*—A rare and remarkable congenital affection occurring in families, and characterised by tonic spasm in certain voluntary muscles, especially when first set in motion, accompanied with true hypertrophy of their tissue.

6. *Tetany.*—A tonic spasm, occurring in paroxysms lasting some minutes, or even longer; generally limited to the distal parts of the limbs; but in some instances extending to the face and trunk.

7. *Chorea or St Vitus's Dance.*—A disorderly succession of more or less coordinated clonic movements, occurring altogether involuntarily, and frustrating the ordinary motions of the limbs and body; often limited to the arm and leg of one side, or more severe in them than in the opposite limbs; occasionally replaced by incomplete paralysis, often attended with an altered mental state, and occasionally even with mania.

8. *Tetanus.*—Tonic spasms, occurring in paroxysms; beginning in the jaw or neck, and extending to the whole body; accompanied by extreme pain; in the great majority of cases traumatic in origin, and fatal in result.

9. *Hydrophobia.*—Spasms chiefly affecting the pharynx and larynx; excited by attempts to swallow liquids; the result of infection by the bite of a hydrophobic animal; ending in death.

FACIAL SPASM.*—This is a disorder for which we have in English no very good designation. The French term it *tic convulsif*, while the Germans employ the name of *mimischer Gesichtskrampf* for that form of it which affects the muscles of expression, or those supplied by the facial nerve. Dr Sieveking, in translating Romberg, introduced the term *histrionic spasm* as an equivalent for the German name, but it has not been generally accepted, and it is liable to be misunderstood, and taken as meaning that actors are especially subject to the complaint.

Its essential feature is the occurrence, at longer or shorter intervals, of a sudden and rapid involuntary contraction in a single muscle or group of muscles. One eye may be instantaneously closed and opened, or the forehead may be wrinkled on one side, and as quickly made smooth again; or the angle of the mouth may be twitched to one side, giving to the face the aspect of a meaningless grin. Or, again, the head may be made to nod forwards, or the neck may be distorted to one side, or the shoulder shrugged, or the diaphragm may contract and cause the utterance of a sharp cry. Trousseau mentions that after the lapse of twenty years he recognised a former schoolfellow, who happened to be walking behind him, by a sort of barking noise that he made.

The person himself is often unaware of the complaint, which is, indeed, scarcely more than a morbid habit or trick. Trousseau says that he was consulted by a lady whose three daughters had muscles in different parts of the body affected with spasmodic tic, for which she found great fault with them, without knowing that she herself had it in her face.

Another point which this writer mentions is that the movements sometimes shift from one part and attack another. Thus a patient came to him for violent spasms of the head and one shoulder; methodical gymnastic exercises were prescribed; and after some time the affection disappeared from the affected shoulder and presently showed itself in the other. A similar instance is related by Sir Thomas Watson, of a gentleman who when young used to give an involuntary shake of his head; a blister, applied to his throat for some affection of the air-passages, made this movement painful, and the movement ceased; but, to use his expression, it broke out in his nose, so that he was ever afterwards in the habit of wrinkling it. I lately saw a boy, aged twelve, who four years before had acquired a trick of sniffing and coughing as though he would be choked; this was followed after two years by a habit of "making faces;" and a month before he was brought to me he began to toss his head over to the right side at intervals of a few minutes; whereupon the sniffing and coughing were given up. Sometimes, however, an affection of this kind lasts for a whole lifetime unchanged.

It does not appear that these histrionic or pantomimic spasms are ever influenced by treatment, but probably they could in most cases be corrected when they first appear in children, in the same way as any other bad habit. The application of the continuous current to the portio dura is, however, recommended, and in a case mentioned to the editor by Dr Gamgee, of St Leonards, the result was good.

There is an occasional tonic form of unilateral facial spasm noticed by Marshall Hall, and in some cases this affects not the whole of the portio dura but those branches only which supply the orbicularis oculi. This variety was named Blepharospasmus by von Graefe.

* *Synonyms.*—Spasmodic tic—Histrionic spasm—Tic non-douloureux faciale—Clonic spasm in the area of the portio dura.

Spasmodic Wryneck.*—A form of spasmodic tic just mentioned consists in twitching of the patient's head to one side, this contraction being sudden and passing off instantaneously. But the cervical muscles are liable to another kind of spasm, which is called "wryneck" or "torticollis." This is characterised by a rapid succession of jerking movements, which draw the head with great force towards one shoulder, and give rise to extreme deformity. When one sterno-mastoid is chiefly involved, the corresponding ear is pulled down towards the clavicle, while the chin is pushed upwards, and the whole face is thrown to the opposite side. When it is the trapezius, the head is drawn backwards, without rotation of the chin; the shoulder being at the same time raised. The fact that the larger half of the head lies across the median line on the unaffected side has often led to mistakes as to which side is really the seat of the spasm. But this is a matter of direct observation, and not merely of inference; one or more muscles in a state of powerful contraction can generally be felt, or even seen, through the skin; and writers have asserted that they tend to undergo hypertrophy, while the opposite ones feel soft and are apt to become wasted. Sometimes, however, no such difference can be made out; one is then obliged to suppose that some of the deep-seated muscles of the neck are affected.

Ætiology.—Spasmodic wryneck occurs chiefly in adults. Dr Reynolds says that all but one of the patients whom he has seen have been more than thirty years of age, and the majority more than forty. However, one well-marked case has recently been observed at Guy's Hospital in a girl, aged eleven, who had had it ever since she was a baby twelve months old. Men and women appear to be affected in about equal numbers.

This disease is often directly traceable to cold. Thus Dr Golding Bird ('Guy's Hosp. Rep.' vol. vi) had a patient who was attacked shortly after having been thrown into a state of partial stupor by driving across an open country in a gig one severe winter's night. And Dr Bright relates the case of a woman who was sitting exposed to a draught of cold air, when she suddenly felt what she thought was "a nerve giving way" on the left side of her neck, whereupon her head was drawn to the right. We had two instances in each of which the complaint appeared clearly to be the result of a fall upon the head. Very often, however, no exciting cause can be discovered. The patient, if a woman, may have been pregnant when the complaint began; or it may have followed a severe mental shock, continued worry, or (according to Erb) an attack of enteric fever; but none of these conditions precede it often enough to be definitely associated with its ætiology. Dr Reynolds notes, as a significant fact, that he has not been able to trace it to overwork. But Dr John Harley has reported, in vol. lvii of the 'Med.-Chir. Trans.,' two cases in which this appeared to be the cause of wryneck, one being that of an engraver whose head was habitually kept raised and slightly rotated to the right, so that he might have an oblique view of the plate on which he was engaged. Moreover Dr Harley mentions the case of a governess who spent much of her time in ruling the pupils' copy-books, and who used to rotate her head rhythmically and with emphasis from left to right; she, in her declining years, lost her control over this movement; and her face was twisted every second to the right shoulder, unless her attention was strongly engaged in some other matter.

* *Synonyms.*—Torticollis—Tic rotatoire—Nickkrampf—Clonic spasm in the area of the nervus accessorius.

Another cause of wryneck is irritation from disease of the teeth. Two such instances are on record, in both of which the spasm seems to have been tonic. One is a case of Mr Hancocks' ('Lancet,' 1859, i, p. 80), of a girl who for more than six months had had her head drawn down nearly to the left shoulder; extraction of a stump and of a decayed tooth in the left side of the lower jaw completely cured her in a few days. The other case, which occurred in 1813, is related by Mr Mitchell in the fourth volume of the 'Med.-Chir. Transactions.' It is so far peculiar that the spasms began in the tongue and side of the face, and only affected the neck after the lapse of some days, but precisely similar modifications, in the extent to which different muscles are involved, occur in the ordinary clonic form of wryneck. The patient was a woman, aged fifty; during the attacks her neck was drawn round to the left shoulder, her arm was rigidly extended, her eyelids were widely opened, and the two eyes directed to the left, her mouth was opened and distorted to the left side, showing the clenched teeth; her tongue felt as hard as a board and was curved to the left side; her forehead was wrinkled, and all the muscles of her face were thrown into a state of rigid distortion. After three months the tonic spasm was succeeded by a rapid tremulous motion of the affected parts, and the attack passed off, to be repeated at intervals which rapidly grew shorter, so that a fortnight from the commencement of the disease the spasms became almost continuous. Extraction of some carious teeth and fangs in the left upper jaw was quickly followed by a cure. I am not aware that the ordinary clonic form of wryneck has ever been traced to local irritation of any kind.

Course.—In cases not directly traceable to cold, the development of the complaint is generally very gradual. Dr Reynolds describes the patient as at first feeling uneasy in his neck, and as thinking that something is wrong with his cravats or with the pillow, until after some months he discovers, or is told by a friend, that his head is not straight. During the early progress of the case, an ordinary observer might suppose that the man's shirt-collar was uncomfortable, and that he was trying to ease it by moving his neck to one side; or that he was looking over his shoulder, and endeavouring to get his head further round. Pain is at first absent, but after a time he complains of a dull, aching sensation, which extends to the shoulder or down the arm. At first, by an exercise of the will, he can overcome the spasm, so as to look straight before him, or towards the affected side; even at an advanced stage he can sometimes by a very powerful effort restore the equilibrium of the two sides for a moment, but at the cost of much distress, and with the risk of aggravating the severity of the spasms afterwards. As the disease progresses, he generally gets into the habit of bringing his head into the proper position with his hands. Thus a patient used to walk about with his arm raised, and ready to seize hold of his nose, which he employed as a kind of lever to control the movements. The spasms are always arrested during sleep, and they often cease when the patient is in the recumbent posture, and when he supports his head with his hands. They are at once brought on by any excitement, or by talking, or by attempting to walk. In the more severe cases the head is jerked about with extreme violence, and this goes on for hours or even for days without any interval. Sleep is almost impossible, and the patient's state is one of the utmost misery.

Spasmodic wryneck may remain stationary for years; neither advancing nor receding. But sometimes it extends from the parts first affected to the side of the face and the eye, or to the shoulder and arm. In two cases at

Guy's Hospital the movements began in the upper limb and attacked the neck secondarily. This, and the fact that the deep cervical muscles are sometimes the ones mainly concerned, prove that the disease is not specially associated with the sterno-mastoid muscle nor with the distribution of the spinal accessory nerve, as would appear from the description given by Erb and others. In some cases it passes off within a few months under some plan of treatment, and a long while afterwards returns with its characters unaltered. Thus in a case of Dr Golding Bird's, already referred to, there was an interval of about seven years between the first and second attack; and I have notes of another case in which the patient got well, returned to his work as a gasfitter for thirteen years, and at the end of that time was again seized with the disease, but on the opposite side. This neurosis does not appear like spasmodic tic, to desert one part of the body in order to attack another immediately afterwards.

In the advanced stages of the disease it sometimes but not always happens that the muscles which are the seat of the spasms fall into a state of tonic contraction, so that for hours together the head remains obliquely drawn down to the shoulder but perfectly motionless. Even then, however, any kind of emotional excitement, or the slightest attempt at voluntary movement, generally leads to the development of clonic spasms.

Writers have described *tonic* wryneck as a separate form (*caput obstipum spasticum*), and it is the fact that in some cases there is no history of the occurrence of the ordinary jerking movements at any period. Thus a girl, aged seventeen, who was in Guy's Hospital under Dr Habershon, had her head drawn down so that the chin rested on the second left costal cartilage, and the right side of the lower jaw was so close to the sternum that it was difficult to introduce a finger between them. Very slight twitching movements were all that had been noticed, but the affection was said to have developed itself while the patient lay paralysed in bed, unable to move a limb, or even to feed herself, and this perhaps accounts for the absence of clonic spasms. It is, however, to be noted that the girl was the subject of well-marked hysteria, for it is possible that the wryneck was really an expression of that neurosis, and analogous to hysterical contractions of the limbs.

Results.—One curious effect of wryneck when it occurs in childhood is the permanent deformity in the bones of the face to which it gives rise. In the case just alluded to the left side of the face was considerably larger than the right. When the girl's head was erect, the right eye lay at a higher level than the left one, the median line of the face, instead of being upright, formed an obvious curve, with its concavity towards the left, and the left side of the upper jaw projected beyond that of the lower jaw, while on the right side the relation between them was reversed. The vertebræ of the neck were also distorted, there being a projection on the left side, apparently caused by the transverse process of the atlas. Precisely similar deformities were noted in the little girl of eleven, who had been in the hospital a few years before. They are of importance, because they might be regarded as indications of a primary disease of the bones, such as does in fact often cause a prominence of the cervical spine on one side, and moreover is constantly attended with impairment of the movements of the head.

Treatment.—This is very unsatisfactory; on the one hand many cases are altogether intractable and are not benefited by any curative measures; on the other hand those in which relief is afforded, or which get perfectly

well, yield to the most diverse remedies, which must differ altogether in their operation; moreover, each in turn fails more often than it succeeds. Dr Reynolds speaks of galvanism as being very useful in cases which are not already of too long standing. He finds that a continuous current of moderate intensity, passed through the muscles which are the seat of spasm, causes them to relax, at least for a time. But he adds that even when by this means the head has been maintained in equilibrium for many minutes, day after day, he has often observed that, as soon as the poles are withdrawn, the spasms instantly return. And sometimes it has been obvious that the ultimate effect of the treatment has been to increase the movements. In a patient who some years ago came under Dr Habershon's care at Guy's a considerable amount of relief was afforded by the plan of fastening the sponges upon different parts of the neck, and leaving them for some hours at a time. Faradisation of the muscles on the opposite side of the neck is sometimes useful, by increasing their power of resistance. On the affected side there is an exalted sensibility to interrupted currents; the patient may be altogether unable to bear the application of one which is so weak as to cause no pain whatever on the healthy side.

Rest is of course essential; and it would seem desirable that some mechanical support should be provided which may keep the head in its proper position. Dr Reynolds, however, says that he has not yet seen any apparatus which a patient with confirmed wryneck could habitually wear, but that the appliance suggested by Dr Hearne, of Southampton, is useful in recent cases, and may at least control the movements for an hour or two; as, for instance, when a clergyman wants to get through a particular service.

Another measure of which he speaks highly is the hypodermic injection of morphia. Beginning with a tenth of a grain, he recommends that one should, if possible, increase the dose until two or even three grains are administered twice daily.

In one case recorded by Dr Radcliffe ('System of Med.,' ii, p. 133) arsenic was injected subcutaneously with very striking results. From five to fourteen minims of Fowler's solution were thrown into the connective tissue over the affected muscles at intervals of three or four days or longer; they caused much local irritation and inflammation; but this was afterwards diminished by diluting the solution with water. After the fourteenth injection, at the end of about nine weeks, the patient left the hospital almost well. This method of treatment has, however, been tried at Guy's Hospital by Dr Wilks and also by Dr Moxon without success.

Dr John Harley has recently ('Med.-Chir. Trans.,' lvii) related two cases in which the administration of large doses of succus conii proved very useful. Beginning with an ounce of the liquid, he rapidly pushed the dose in each case until one patient took three and a half ounces once daily, and the other four ounces twice daily. The effect aimed at was the production of a general relaxation of the muscles, so that at the end, the second hour after swallowing the hemlock juice, the patient should be unable to rise from the sitting posture or to walk unassisted, and that there should be ptosis, impairment of power to masticate and swallow food, and slowness of speech. In the first case the spasm became greatly diminished; but the drug had ultimately to be discontinued on account of the mental depression to which it gave rise. In the second case the affection was almost cured, and the patient resumed his occupation, working half-time, with only an occasional

tendency to twist of the head, if he became overtired. Dr Moxon has followed Dr Harley's plan with a decided measure of success.

In a case recently in Philip Ward the constant galvanic current failed to do good, but the patient gradually improved under a mixture of bromide of potassium and arsenic, and went out apparently cured.

Another plan of treatment, by which Busch succeeded in curing permanently three cases out of four, is the application of the actual cautery in lines, five or six inches long, to the neck on both sides of the spine, and maintaining suppuration for some weeks after the separation of the eschars.

Some years ago ('Med.-Chir. Rev.,' 1866) Mr Campbell de Morgan excised an inch of the trunk of the spinal accessory nerve, above the origin of the branches to the sterno-mastoid muscle, in one very severe case, with permanent relief to the spasm, but with equally permanent paralysis of the affected muscles. Another operation which has been performed several times with good results is the subcutaneous section of the sterno-mastoid muscle.

In those cases in which the spasm is tonic, it is always advisable to straighten the head under chloroform, and to endeavour, by mechanical appliances, to prevent the return of the contraction.

Clonic spasms may affect individual muscles of the face or limbs, and have been named spasmus nictitans, &c. Singultus or hiccough is a clonic spasmodic affection of the diaphragm.

MOVEMENT-SPASMS.*—Within the last few years several observers have devoted attention to cases in which spasmodic contractions of various kinds are brought about by voluntary movement, the exciting cause in each instance being generally limited to some one particular action, so that the patient remains quiet when sitting or lying down, and is able to use all his muscles for other purposes perfectly well. The best paper on the subject is, perhaps, one by Dr Weir Mitchell in the 'American Journal' for 1876. His name for this group of affections is that of "functional spasms;" but this might be taken as meaning "spasms which are functional and not organic," in which sense it would include all the diseases described in this chapter. "Function-spasms," "Movement-spasms," or "Fatigue-spasms," are terms which, if less euphonious, are also less misleading.

Many of Dr Mitchell's cases are exceedingly curious. Two are examples of what he terms "lock-spasm." A watchmaker, who often had to pick up and adjust very small screws, would find ten or twelve times a day that his thumb and forefinger suddenly became locked upon one of them, so that he had to release it with a loop of twine, not always without wounding the finger; even then the cramp would last for some considerable time longer. At a later period, when he was turning over the pages of a book, the finger and thumb would sometimes close with violence, so as to tear the leaf. Another man, a sawyer, was liable once, or even twice, a day to have the arm arrested, and fixed in a state of flexion, at the moment when his saw was drawn back to prepare for the downward movement. By no effort that Dr Mitchell could exert was he able to overcome the spasm; on one occasion he made the patient bend over, so as to bring the forearm into a horizontal position, and he then found that for five minutes the biceps

* *Synonym.*—Functional spasms (Weir Mitchell). This group includes most Reflex spasmodic neuroses, Saltatorial spasms and the Beschäftigungsneurosen of German writers, viz. writers' palsy, fiddlers', tailors', cigar makers' cramp, lock-spasm, milkmaids' cramp, &c.

supported a weight of eighty pounds suspended from the wrist. Such cases are clearly analogous to the cramps of telegraph workers and milkmaids.

In other cases, the attempt to walk was accompanied by a tonic spasm of certain muscles, so that a kind of stringhalt was produced. Yet another patient, a journeyman tailor, had an extraordinary seizure whenever he jumped up suddenly from the prone or the sitting posture. The right leg was then bent at the knee, the left was thrown over it in violent flexion, the body and the head were twisted to the right, the right arm was extended and raised, the left was thrown outwards and backwards in extreme pronation. Then there was a general writhing of the whole frame, the facial muscles twitching here and there; and the attack passed off with a groan of relief. This man could make every possible movement, slowly or abruptly as he pleased, so long as he remained seated or lying down; he was even able to get up; and, after cautiously standing still for a moment, could walk away as well as anyone else. Such cases resemble the saltatorial spasm to be presently described, and also the hereditary cases described by Thomsen (p. 734).

In other cases, again, spasms accompanied the act of laughing or talking, or, above all, chewing. One patient, as soon as he began to masticate his food, had his mouth jerked open, so that he had to keep shutting it with his hand. When he wanted to swallow, his face assumed an aspect of terror. "He would suddenly muster courage, and swallow the contents of his mouth at a gulp. Then instantly the jaw flew open, the head was drawn back and down upon the left shoulder, the face was convulsed, and sweat ran from the forehead." In another case of the same kind the gastrocnemii were involved in the spasm, so that the patient was jerked into a squatting position. An instance came under the author's observation in which the chewing of food brought on a kind of wryneck, the head being twisted over to one shoulder; and another, in which the effort of speaking was attended with an extraordinary series of grimaces. These are clearly analogous to the spasms brought on by handicrafts (p. 723).

But perhaps of all this group of spasmodic neuroses called forth by voluntary or reflex movements, the strangest are certain cases in which any attempt to stand excites violent convulsions. The patient is thrown off his feet, his legs pass into a state of clonic spasm, and he begins to execute a succession of the most rapid and irregular jumping and skipping movements, even though he may be supported on each side by another person. So great are his efforts that his face flushes, his pulse is quickened, and he breaks out into a profuse sweat. Yet when he is lying or sitting he remains quiet, and can move his legs perfectly well in all directions. For this affection a special name was invented in 1859 by Bamberger, who recorded in the Vienna 'Med. Wochenschrift,' two cases of what he called "Saltatorial reflex spasm, a remarkable form of Spinal Irritation." Dr Gowers described two cases of the affection ('Lancet,' July and August, 1877), with an analysis of the five previously recorded. In their details these cases differ to some extent from one another, and this affords an additional reason for including them in the group of "reflex movement-spasms."

Sometimes it has been possible for the patient to excite the movements by pressing with his feet against the foot-board of the bed, even while he is lying down; in one instance, tickling the skin of the soles would elicit them; in another, pressure upon a tender part of the

spine had the same effect. They have often been much augmented by emotional excitement; and this has sometimes led to doubts as to their genuineness, it being found that they are much less violent when no one is looking on; but the same thing may be said of many other affections belonging to the same class. Erb suggests that saltatorial spasms will ultimately be found to be exaggerated tendon-reflex; but Dr Gowers found that tension on the *tendo Achillis* produced no ankle-clonus. He is therefore no doubt right in thinking that the movements are due to the combined and simultaneous action of peripheral and voluntary stimuli upon irritable centres in the cord; but as the same stimuli are in action in all healthy persons whenever they stand upright, this throws little light upon the question. In Bamberger s first case, that of a youth of nineteen, the affection came on during convalescence from acute pneumonia; recovery took place in about a month under the internal administration of morphia. Other cases have been much more protracted; in one instance the spasm had not subsided when the patient died of fever at the end of five years. There have been two cases in women, both of whom were affected with hysteria; one had several attacks of the spasm, each lasting some months; her recovery was once quite sudden, exactly as so often happens in hysterical contractures.

There is sometimes pain in the contracting muscles, sometimes none; there is no loss of power or of sensation.

The reflex arc does not begin in the skin of the feet, but in deeper structures, probably in the tendon. It would therefore be analogous to knee-jerk. In future cases the condition of this phenomenon and of ankle-clonus will no doubt be investigated. In some cases, however, the afferent channels appear to be the cutaneous branches of the plantar nerves.

The muscles of the trunk and head are sometimes affected, as well as those of the legs, but the arms escape.

This curious affection has been observed in both sexes, and at all periods of life, from childhood to old age. It appears to be sometimes associated with spastic paraplegia as a sequel of some form of myelitis. But most cases seem to be "functional," and occur with hysterical symptoms. What was called chorea major, or dancing madness, would probably in a sporadic form approach some of the cases of saltatorial spasm in young women. It has also relations to tetany and to post-hemiplegic chorea.

The affection, once begun, may continue for months and years, or may return after disappearing. It does not appear to lead to further symptoms or to threaten life. No special treatment is known.

Many other rhythmical spasms may be observed, chiefly in patients not suffering from organic disease. One variety is what has been called "Salaam palsy" (*Eclampsia nutans*), a habit of continually bowing the head as if making a profound reverence. It has been frequently observed in children. Similar spasms may affect the arms, as in a boy once in Guy's Hospital, under Sir William Gull, who was continually moving one arm up and down. Others lift up one leg every third or fifth step in walking and strike it with the hand. Stammering and stuttering is due to laryngeal or labio-lingual spasm, or to both, and exhibits the same union of irregular contractions, with loss of power, and explosive completion of a coordinated function, which we have had occasion to notice in the whole group of which we are

here treating. Some of these spasms are clearly reflex, and are so described by Erb. Some resemble histrionic spasm, some saltatorial cramp, some hysterical movements, chorea major, and "ugly tricks" in children. Many are curable by discipline, or a shock to the imagination, or by growing out of them.

In some of his cases of function-spasm Dr Mitchell found benefit from the injection of solution of atropine into the substance of the affected muscles.

*Spasmodic affections caused by habitual handicrafts.**—There is a class of cases which evidently belong, pathologically, to the same group as the last, but which differ from the rest in the circumstance that the involuntary motions that attend them are only just sufficient to disturb the due execution of some highly specialised action, involving numerous and complicated muscular movements, and that over-exercise of this action appears to produce the spasms. They are examples of voluntary "movement-spasms," or what Dr Poore calls "Fatigue diseases."

A musician, for example, becomes unable to play the piano or the violin, whichever happens to be his instrument; a telegraph clerk cannot work Morse's machine; a tailor or shoemaker no longer has the power to sew; a milkmaid ceases to be able to press the milk from the cow's teats; a smith to bring down his hammer upon the anvil. In one case a man, whose whole occupation was to clean knives and boots and shoes for a large house, became completely incapacitated for this work, although he could still do everything else. Each of these cases, however, is exceptional; far more numerous are those in which the complaint consists in an inability to write, and this deserves special attention.

Writers' Cramp.†—The exact nature of the impairment in the power of writing varies widely in different cases of writers' cramp, so that hardly any two are alike. Sometimes the patient's complaint is that after he has been writing for a few minutes his hand and forearm feel wearied, sometimes that the fingers become suddenly extended so that the pen drops from his grasp, sometimes that the index finger is straightened and drawn off the pen-handle, sometimes that the fingers are pressed too tightly upon it so as to impede its movements. In many cases there is no visible cramp or spasm; all that can be seen by a looker-on is that the motions of the hand are arrested in the act of writing. But in some instances there is obvious shaking of the pen.

The handwriting is often altered in character; in some instances the change consists in a reduction of the size of the letters, the power to make free strokes failing, and the writing becoming "cramped;" other patients become unable to accomplish a "running hand," and are obliged to confine themselves to a slow "round text." The author had one patient who could do anything but shorthand perfectly well, but unfortunately he was a law-reporter. In the immense majority of cases, however, the peculiarity consists in an uncertain tremulous formation of the strokes which make up the letters. Indeed, when the affection is severe the handwriting becomes

* There is no name which would conveniently include all this group of spasmodic affections; that of "coordinated-business-neuroses," proposed by Benedikt, is somewhat unwieldy. "Handicraft spasms," perhaps, might serve.

† *Synonyms.*—Scriveners' Palsy—Penman's spasm—Mogigraphia (μόγις, with difficulty; γράφω, write)—Graphospasmus.

altogether unintelligible, consisting of a meaningless succession of shaky lines and curves.

Persons affected with writers' cramp adopt all sorts of odd devices to diminish the inconvenience which the complaint causes them. Some will write only with a quill, and, so far as it goes, this is undoubtedly a good plan, for the muscular effort required is very much less than with a steel pen. Others employ a large thick penholder, or fix a nib in a broad piece of cork. Dr Poore ('Practitioner,' xi) mentions the case of a man who used a rounded mass of wood of the shape of a boy's top, which he held tightly within the hollow of the palm of his hand. Others grasp a penholder with the closed fist, or fasten it to one finger by means of a ring.

Sometimes, however, the fault seems to be not so much in the hand as in the wrist and forearm. A patient of Dr Poore's said that the first thing he noticed was a difficulty in bringing down the hand upon the paper, and for three days he forced himself to write by holding down the right wrist with the left hand, but "at the end of this time that resource failed him." Indeed, one of the most important characteristics of "writers' cramp" is that it is a progressive disease. For a time a person affected with it may succeed in accomplishing the art of writing by bringing different sets of muscles into exercise. Thus, instead of employing the weak muscles of the thumb and fingers, he may use those of the forearm; or he may place the arm in some extraordinary attitude as soon as he takes up the pen, resting, perhaps, his wrist upon the table, and raising his elbow at an angle; or pressing the limb close to the side of his body; or, again, straightening the elbow, and holding the instrument at arm's length. But if he persists in writing much all these resources soon fail him. The muscles which he has called in to his assistance "give out" in their turn. Even if he should have taken the pains to learn to use the pen with the other hand, that too is after a time attacked. At advanced periods of the disease, the muscles are often affected with spasmodic contractions of considerable violence independently of all efforts at writing. Dr Poore's patient, to whom I have referred, complained that his hand was always poking about; it would sometimes bounce suddenly out of the side-pocket of his coat while he was walking in the street, and he once broke a jug in consequence of an unexpected spasm while he was pouring out some water. I had a patient who told me that his shoulder invariably gave a jerk when he put his hand up to his head. Lastly, in some cases, the spasm extends to the muscles of the neck and body. Dr Reynolds mentions an instance in which the effort to write caused the head to be drawn downwards to the right shoulder and the trunk to be contorted so as to be concave on the right side. The same writer also states that he has seen torticollis, occasional strabismus, stammering, and palpitation of the heart, associated with writers' cramp.

There is not generally any impairment of sensation. Sometimes the patient complains of "numbness," or "tightness," or "coldness," or of some peculiar sensation which he cannot define in one particular finger, or running from some part of the hand up towards the arm. In some rare cases actual anæsthesia is said to be present. Very commonly the act of writing, if continued for any length of time, causes the hand and wrist to ache, or even the shoulder or the spine. These abnormal feelings distress the patient to an extent which seems altogether disproportionate to that of the actual pain which he has to endure. Indeed, a peculiarity is often observable in the psychical state of those who are affected with writers' cramp in

its more advanced stages; they are highly irritable and excitable, and very desponding about their complaint. Dr Poore speaks of one man as having been apparently on the verge of suicide, and of another as having actually taken up a knife with his left hand and stabbed himself on the wrist. Such patients are generally nervous, and they are less than ever able to write when others are looking on, or when the subject-matter is of special importance. Dr Poore was told by one person that he got on better when copying than when composing what he wrote, and another patient had to watch the point of his pen, for if he looked away from it his handwriting at once became unsteady. Those who are affected with this disease are also said to sleep badly, and to be generally ill-nourished, with soft, flabby muscles. Dr Poore attaches some importance to a thin, brittle state of the nails which he has often noticed.

Ætiology.—It is generally assumed that the cause of writers' cramp, and of the affections allied to it, is overuse of the muscles, or working too hard and too long at that particular kind of labour, which becomes accompanied with the spasm. And it is undoubtedly true that most of those who are attacked are clerks, or accountants, or in some way professionally engaged in writing. Moreover, as Dr Poore points out, the commencement of the disease often dates from the time at which some heavy task has been accomplished, as when an architect has worked against time to complete the details of a plan, or a lady to finish within two or three days an etching for a bazaar. Still, it is also the fact that some individuals strain their powers to the utmost, and get through extraordinary amounts of writing, without ever suffering from it; and others are attacked who have at no time over-exerted themselves in any way, or have actually done less writing than is safely accomplished by the majority of persons. I have seen two such cases; one in a boy, aged fifteen, whose education seemed to have been rather neglected than otherwise; and one in a lady of fashion, who had at most to write a few notes to her friends. Dr Poore speaks of an instance as having come under his observation in which three generations in a direct line were affected. One of his patients referred the complaint to the severe shock caused by an alarm of fire.

Pathology.—The seat of these neuroses is doubtful, and there are two theories with regard to it. One, which is supported by Reynolds and by Erb, and seems to me the more probable, is that it lies in the central ganglia which effect the association and coordination of muscular movements for the more complex actions. The nutrition of these structures is supposed to be impaired as the result of over-exertion, and the consequence is a perversion of their functions, which expresses itself in irregular spasmodic movements. A strong point in favour of this view is the fact that when one arm has been affected the other is often afterwards attacked; and the relations which writers' cramp bears to torticollis and other spasmodic neuroses afford a further argument.

The other theory, which was first proposed by Zuradelli (1857) and is ably advocated by Dr Poore, is that the starting-point of the morbid change is exhaustion of some particular muscle or set of muscles—generally one of the small intrinsic muscles of the thumb or fingers—which are kept constantly in action throughout the whole time during which a pen is held in the hand. One or more of these, it is suggested, begins after a time to respond sluggishly to the stimulus of the will. The patient then unconsciously calls into play other muscles, generally those of the forearm. In

their turn, these two become worn out; and thus the process of substitution is carried on always with the same result. Dr Poore has in a large number of cases of writers' cramp tested the electric reactions of the muscles which are specially engaged in the act of holding a pen, and has found that often one or more of them showed a marked impairment of irritability as compared with those of the opposite limb.* It might be thought that a strong argument against the theory in question is afforded by the well-known and remarkable fact that a patient who is severely affected with the disease is often perfectly well able to perform all other actions. But Dr. Poore argues that the utility of the affected muscles for other purposes is only apparent; the hand and forearm, he says, are so bountifully furnished that for most movements there are more muscles than are required. If a man finds some of them to be weak, he unconsciously substitutes others in their place to do the work required. This writer maintains that certain among the more delicate actions of the hand are generally interfered with beside those which give to the case its principal character. Thus one of his patients could no longer hold a knitting needle; another was unable to take up her dress between her thumb and her forefinger; and a third could not toss over a letter into a letter-box. So, Erb states that the sufferers from writers' cramp may be unable to button their clothes, to work embroidery, or to play upon the piano. That the motor centres in the cortex are the seat of the disorder seems to be the most probable view. One of Dr Poore's patients had great difficulty in feeding and in dressing himself.

Benedikt has proposed to distinguish different forms of the disease, namely, a spasmodic, a tremulous, and a paralytic form.

Diagnosis.—The diagnosis of writers' cramp, or of the allied forms of handicraft and reflex spasmodic spasm, is tolerably easy when they have reached an advanced stage. But one must always recollect that impairment of the power of writing may result from a number of other affections of the nerves and muscles of the upper limbs. Progressive muscular atrophy, neuritis of the ulnar, the median, or the radial nerve, compression of either of these nerves by a tumour of whatever kind, may each give rise to this symptom. Dr Poore mentions a case of subclavio-axillary aneurysm in which the first thing noticed by the patient was that he could write only with great effort, and then illegibly.

As a rule, a person affected with writers' cramp goes on for a long time without medical advice, struggling against what he deems a foolish incapacity to carry on his duties. But when it does happen that one is consulted at a very early period, there may be great difficulty in determining whether the disease is really present or not. This is particularly the case if the patient is a nervous professional man, whose mind has for some time been dwelling upon the subject; or if he is a bank clerk, who has heard all about it from his fellow-officers, and who has perhaps been reading a number of medical books. Such persons come to one complaining that the act of writing causes a number of unpleasant sensations; and one may be in great doubt whether to laugh at their complaints or to take them seriously. It seems to me that the best indications are afforded by the effects of rest. If the supposed symptoms show themselves only at the end of a hard day's

* Electrical irritability is generally deficient, as Dr Frank Smith found to be the case in hammer palsy; but Dr Dubois, of Berne, has lately stated that in early cases of telegraph-workers' cramp faradic and galvanic irritability of the affected muscles are exalted, although afterwards depressed or lost ('Brit. Med. Journ.,' 1887, vol. i, p. 1302).

work, and are entirely gone the next morning or after the interval of a Sunday, they are not likely to be of serious consequence. Thus Dr Poore speaks of an eminent pianist who after practising for a considerable time suffered from an inability to adjust the movements of his fingers, so that he could not always strike the right note, but who, a few hours later, could play as well as ever. In such a case it is evident there is nothing seriously wrong. But I believe that I have seen one instance of true writers' cramp at an early stage, in which the patient said that his hand felt perfectly well every morning when he first got up.

Prognosis and treatment.—In most cases a right diagnosis is of the utmost importance, for on it may depend the whole future of the patient. At this period the discontinuance of all writing—or of whatever other action may be the exciting cause of the complaint—may completely cure it in the course of a month or two. Even then, indeed, there is always a risk that the disease may return if the muscles should be again overworked. But, most commonly, the prognosis that must be given is very unfavourable. At an advanced stage of the disease there is scarcely any chance of recovery, unless the patient can altogether give up his occupation for six months or even for a whole year. And in many cases treatment fails entirely, the disease advancing steadily, in spite of all that can be done. The only remedy appears to be galvanism. Of this Erb speaks highly, recommending the employment of ascending currents along the cervical spine, as well as peripheral galvanisation of the nerves and muscles specially concerned. But I am not aware that any results have been obtained by these methods which can be compared with those which Dr Poore got by a method of his own: this consists in making the patient perform rhythmical movements with the affected muscles, while a continuous current of moderate intensity is passed through them. His first case was that of a man, aged thirty-two, who had suffered from writers' cramp for more than nine years, and in whom it presented its severest form. Dr Poore exercised separately the deltoid, the biceps, the triceps, and the other muscles, as well as those of the hands and fingers. When the treatment was commenced the patient could not finish writing the word George; at the end of five months he was able to write a whole epistle, forming his letters well and firmly. He had been unable to feed himself; he could now do all that he wanted, and had embarked in a small business. Another of Dr Poore's cases is little less remarkable. A man, aged forty, had for four years suffered from the disease, and had for six months been unable to write, the attempt to do so giving him great pain. After the first application of the current this pain disappeared, within a week he could sign his name with tolerable ease, and at the end of ten months his handwriting looked as good as it had ever been, although he was not at that time able to hold his pen quite properly and tightly between the thumb and fingers. Unfortunately, however, Dr Poore records no similar successes in his later papers on the subject. Wolff's combination of galvanic treatment with massage is commended by Charcot, by Ross, and by de Watteville.

Bromide of potassium is said to be occasionally beneficial to patients affected with writers' cramp, by removing the depression and irritability from which they suffer. Strychnia is often injurious. Stimulating liniments and douches appear to be almost if not quite useless. I have already mentioned that using a quill instead of a steel pen is frequently found to be of great service. Erb further says that many patients are relieved by

applying a narrow bandage or strip of plaster round the wrists. This cannot, however, be expected to do any good where the only muscles affected are the little muscles of the thumb and fingers.

*Hammer palsy.**—This curious variety of handicraft spasm was described by the late Dr Frank Smith, of Sheffield ('Lancet,' March 27th, 1869, and 'Brit. Med. Journ.,' Oct. 31st, 1874). He observed eight cases among the pen, blade, and file forgers, who use a hammer of three to eight pounds weight sometimes for twelve or thirteen hours, delivering as many as 28,800 carefully adjusted strokes every day.

The patient finds his hand has lost its cunning; he is awkward and hits awry. Spasms of the flexors and pronators of the forearm appear, then gradual weakness of the whole limb, and finally muscular atrophy.

We have lately had a typical case of hammer spasm in Guy's Hospital (Philip Ward, 31, 1887) in a healthy, well-nourished man, of twenty-seven. He worked at nail-making with a hammer of two pounds weight, and worked by the piece. His arm on his attempting to move it, or on the hand being grasped, is at once thrown into clonic spasms, without pain, which affect the pronators and flexors of the forearm, and also the flexors and adductors of the arms. Sensation is perfect. There are no signs of affection of the cranial or other nerves, no wasting of muscles, and no cephalic symptoms.†

In Dr Frank Smith's cases the right leg was frequently weak; there was neuralgia or numbness, and painful spasm in the affected arm; there was sometimes ptosis and frequently thickness of speech, while in one instance (No. 7) there was aphasia. Hence the justification of the name he proposed: Hephæstic *hemiplegia*.

Of one case he writes: Neuralgia and an indescribable centripetal feeling frequently affected the right arm and forearm. The tactile sensibility of the forearm was almost lost: electro-sensibility and electro-contractility were almost extinct. Thermal sensibility remained. The temperature in the right axilla was 1° Fahrenheit below that of the left. There were no mental symptoms; sight, smell, hearing, taste, and speech were unaffected. The optic disc of the right side was paler than that of the left.

The facts of spasms being followed by paralysis, of extension of motor symptoms to the leg and face, and of the affection of speech, all appear to point to the motor region of the hemispheres as the seat of this remarkable disease. No autopsy has yet been made, and it is quite possible that the local lesion is, at least in its earlier stages, either vascular or otherwise unrecognisable after death. A "discharging," becoming afterwards a "destroying" lesion, would probably best explain writers' cramp and the allied spasmodic diseases. At least there seems less reason for placing the seat of the disease, whether "functional" or "organic," in the muscles, peripheral nerves, anterior cornua, or motor tract of the cord.

Some of Dr Smith's cases improved under sedatives and rest. In the case above mentioned in Guy's Hospital, Dr Poore's plan for scriveners' cramp was tried without benefit,—also Calabar bean, bromides, and succus conii raised to a two-ounce dose and continued three times a day for nearly a fortnight. Rest in bed seemed to be most useful, but the patient thought chloral hydrate at night not only gave him refreshing sleep but made the spasms less severe during the day.

* *Synonyms.*—Hammer spasm—Hephæstic Hemiplegia.

† This case, we afterwards found, was the same which formed the subject of a lecture by Dr Poore in the 'Lancet' for August 21st, 1886.

PARALYSIS AGITANS.—Another disease whose precise seat in the nervous centres is uncertain is that known as "paralysis agitans," or "shaking palsy."* It consists in a more or less violent oscillatory movement, affecting especially the limbs, but sometimes the neck and the tongue also. It almost always begins in one of the upper limbs; the movement may at first be very slight, in fact, scarcely more than a tremor. Charcot says it is sometimes for a time limited to one thumb. In the beginning the patient commonly attaches but little importance to it, but gradually the space occupied by the oscillations becomes greater and greater and the whole limb is involved in them, so that he is unable to go on with his work and has to seek medical advice. Sometimes, however, the agitation of the muscles is severe from the very first. Charcot speaks of this as the result of a sudden shock or terror, but I have notes of two cases in each of which the patient went to bed as usual and woke in the morning with the disease fully developed, the right arm being in one instance the part affected, in the other both the left arm and the left leg. In two other cases that have recently occurred at Guy's one side of the neck has been the earliest seat of the movements. After a variable interval—generally after some months—the oscillations appear somewhere else, most frequently in the leg of the same side as the affected arm, but sometimes in the other arm. Presently the remaining limbs suffer in their turn. The head also begins to shake, and the tongue becomes very tremulous when it is protruded, or even when it is lying within the mouth, but nystagmus, or oscillation of the eyeballs, seems never to occur, nor do the jaws ever take part in the movements.

During the early part of its course, paralysis agitans is almost always paroxysmal. Each attack generally lasts some hours, and it is followed by a feeling of great fatigue, which gradually passes off in the interval of rest before the next one begins. At a later period the movements become continuous.

The oscillations bear to voluntary motions of the affected parts a relation on which great stress has been laid by successive writers, but which is hardly so simple as has been represented. Mr Parkinson, who in 1817 first described "the shaking palsy," begins his monograph by defining it as follows:—"Involuntary tremulous motion, with lessened muscular power *in parts not in action*, and even when supported; with a propensity to bend the trunk forwards, and to pass from a walking to a running pace; the senses and intellect being uninjured." He goes on to distinguish the disease from the mere tremor of old age, as well as from other complaints, among which is that caused by the vapour of mercury; and it is the fact that during the early period of the disease the hand may become perfectly steady when it takes up a tool or a pen, and that the patient is able to control the agitated limb for some minutes by an effort of his will. Moreover, the oscillations often go on without intermission when the elbow rests upon the knee. But, on the other hand, it appears that at this stage the affection is usually dormant so long as the patient lies quietly on his back in bed, and it is certain that no movements occur during sleep except in the most advanced period of the disease, when (according to Parkinson) the patient's rest may be entirely broken by them. This writer also mentions another curious fact, namely, that one of his patients had an intercurrent attack of right hemiplegia, in which the face was drawn to the left side, and this lasted a fortnight;

* *Synonyms.*—Shaking palsy—Paralysis cum tremore—'Parkinson's disease'—Scelotyrbe festinans—Chorea procursiva.

during that time neither the arm nor the leg on the paralysed side was in the least affected with the tremulous agitation, but it returned as the limbs regained their power. In the later stage of shaking palsy, indeed, the attempt to perform any voluntary movement brings on the oscillations with greatly increased violence. They are also much augmented by emotion or excitement. Even the presence of a looker-on often affects such patients to a remarkable degree; a man who can write quite well when alone may be unable to form a letter while he is being watched by a clinical clerk. A patient of mine found much relief from keeping his arm in a sling, which greatly diminished the oscillations. In him, as in some others, I observed that grasping the agitated limb with my hand, so as to check its movements, led to a marked increase in them and to the development of similar movements in the opposite arm, which was less severely affected with the disease.

According to most writers there is in paralysis agitans, besides the oscillatory movements, a certain degree of impairment in the muscular power of the affected parts. Eulenberg, indeed, has found that even in severe cases of long standing the reaction to induced or galvanic currents is perfect, and both Trousseau and Charcot have taught that the muscles really retain their full force. The former writer relates a case in which the patient was able to exert much more power with the affected hand (which, however, was the right one) than with the other, which was healthy—considerably more power than Trousseau himself could exert. But this must not be taken as showing that the disease had actually augmented the force of the limb, since it was not known what the man's strength had previously been. Another point observed in that case was that the contractility of the muscles was very quickly exhausted; when told to open and shut his hand in quick succession the patient at first moved rapidly, but after scarcely fifteen seconds more slowly, and soon not at all. This point also is adopted by Charcot, and he further remarks that there is often a retardation of the influence of the will, there being, for example, an unduly long interval between a thought and its expression in words. Recently, however, M. Bourneville, the editor of 'Charcot's Lectures,' has made on six patients observations with the dynamometer which appear to show that their strength was really diminished to a considerable extent.

One thing which impedes voluntary movements in the more advanced stages of paralysis agitans is the occurrence of *rigidity* in the muscles of the affected parts. Charcot has pointed out that the patient commonly maintains an attitude which is characteristic of the disease. The head is bent so that the chin approaches the sternum, and he can with difficulty raise it or turn it to left or right. The body is almost always bowed forwards when he is standing. The elbows are generally drawn slightly away from the chest, the forearms are partially flexed, the hands, which rest together upon the waist, present a deformity somewhat like that which occurs in osteo-arthritis, the three inner fingers being inclined towards the ulnar side of the hand, and their joints being alternately extended and flexed, while the thumb and forefinger are stretched out and brought close together as in holding a pen. In the lower limbs the rigidity is sometimes very marked; they are semiflexed, the knees are brought together by a movement of adduction, the feet are curved inwards as in talipes equino-varus, the toes are arched like the three fingers. Lastly, according to Charcot, the features present a peculiar immobility and want of expression.

There is no actual impairment of articulation, but the speech is slow and jerking, as though a considerable effort of the will were needed for the pronunciation of each word. Charcot remarks that it reminds one of a bad rider talking when his horse is on the trot.

The way in which the head and body are bent forwards in the more advanced stages of paralysis agitans accounts for a symptom which Parkinson noticed, namely, that the patient tends to fall upon his face when he attempts to walk, and that his steps are consequently hurried, so that he runs instead of keeping to his ordinary pace. This had, indeed, been previously described by Sauvages, but as a separate complaint, under the name of *Scelotyrbe festinans.** The man has been said to be constantly trying to overtake his own centre of gravity. Parkinson mentions a case in which an attendant was obliged to walk backwards in front of such a patient with one hand on each of his shoulders to prevent him from falling. Moreover, when he first gets up from his seat, which he does very slowly, the patient makes a few hesitating and ineffectual steps before he seems to be able to start off, and that in walking he treads only upon his toes, being unable to bring his heels to the ground. But the most remarkable circumstance of all is that some persons affected with paralysis agitans walk backwards without intending it and when they mean to go forwards. Some years ago a striking instance of this occurred in my out-patient practice at Guy's Hospital. The patient, after a few ineffectual efforts to rise from his chair, would stand up, pause, give two or three abortive attempts at starting, and then succeed in making a few steps towards the door, when suddenly he found himself hurried against his will backwards into the umbrella-stand in the corner of the room. I was reminded of nothing so much as the way in which the engines of a steamboat can be instantly reversed. Charcot met with a case in which he could at any time induce such retrograde movements by unexpectedly giving a gentle pull at the patient's dress when she was standing up, and Dr Buzzard remarked exactly the same thing in an elderly woman, whose appearance and symptoms he graphically describes in his lectures. The movements always cease during sleep.

In the more marked cases of paralysis agitans the movements of the hands are not merely oscillatory, but resemble to some extent such as might be purposive. Charcot points out that the thumb may be carried over the fingers, as in rolling up a piece of paper, and that the fingers may be brought together, just as though a morsel of bread were being broken into crumbs.

Profuse sweating is an almost constant symptom when the oscillatory movements are at all severe. The patient may become so bathed in perspiration as to have to change his clothes many times a day. Charcot also mentions that he often suffers greatly from a feeling of heat, especially about the epigastrium and in the back, so that he insists on being kept lightly covered. But with the thermometer the temperature is always found to be normal.

The face has a remarkably impassive expression. The voice is often shrill, fresh, and piping, and Dr Buzzard believes that this change of an old man's voice to "childish treble" is an almost decisive symptom of shaking palsy.

In most cases, limbs affected with paralysis agitans have their cutaneous sensibility unimpaired. Charcot, however, says that a feeling of pins and

* Σκελοτύρβη (disorder of the legs, staggering) occurs in Strabo, Pliny, and Galen. It has been also applied to chorea.

needles in the hands and feet is sometimes complained of; and I find this mentioned in one case of which I have notes, the patient also saying that he was unable to feel the ground. Headache and vertigo are not uncommonly present; and at an advanced period of the disease there may be loss of memory, and even delirium.

Pathology.—As I have already observed, it is still uncertain whether paralysis agitans should be regarded as an affection of the brain or of the spinal cord. Parkinson supposed its seat to be in the cervical region of the cord, extending up to the medulla oblongata. But perhaps indications adverse to such a view may be found in the fact that after one upper limb the corresponding leg is generally next affected, rather than the opposite arm; and still more in the cessation of the oscillations during sleep, and throughout the continuance of a hemiplegic attack, so far as the limbs paralysed are concerned. Hitherto anatomical observations have thrown no light on the question. I have made one autopsy, that of a woman, aged forty, who died of phthisis after having suffered from paralysis agitans for eight years; for a year her speech had been impaired, and at the last it was unintelligible. The only unusual appearance that I could fancy I detected was that the substance of the pons towards the floor of the fourth ventricle looked unduly grey. Westphal and Wilks have also failed to find any lesion. Slight thickening of the cortical layer and reticulum were found by Cayley in the cord of a patient of Murchison's, aged seventy-one, who died of typhus after being twelve years the subject of paralysis agitans ('Path. Trans.,' 1871, p. 24). Charcot has been able to examine the nervous centres in six cases; in three they were perfectly healthy; in three they presented slight microscopical changes which were believed to be merely senile. In some cases recorded by other observers there has been an obvious atrophy of the brain, but only such as is commonly found in persons of the same age as that to which the individuals in question had attained. Eulenberg's paper in 'Ziemssen's Handbuch' is placed among "neuroses."

Paralysis agitans is by no means a common complaint. During the ten years from 1866 to 1875 only about fourteen cases were admitted into the wards of Guy's Hospital, and from 1876 to 1885 eighteen.

Age.—The disease is most often seen in persons who are advanced in years. Parkinson speaks of it as seldom occurring in persons below the age of fifty; but among the thirty-two cases at Guy's Hospital there were nineteen in whom it began at an earlier age, and in nine of them before forty years had been reached, four being thirty-six, one thirty-two, three thirty-eight, and the youngest but twenty-one years old. This last instance is not without precedent; for Charcot mentions a case of Duchenne's in which the patient was only twenty, and one of Fioupe's which occurred in a girl of fifteen or sixteen who had been terrified by a bombshell during the siege of Paris. In eleven of our thirty-two cases, the disease began between the fiftieth year and the fifty-ninth, and there were only two in which the patient was still older when first attacked, namely, at the age of sixty-one. It is therefore a mistake to suppose that shaking palsy is essentially a senile disease. But on the other hand, I am not sure that a strict line of separation can be drawn between it and the tremor which is so common in old people, and which (as Dr Maclachlan found among the inmates of Chelsea Hospital) has little or no tendency to shorten their lives. He mentions one case in a pensioner, aged 107, who had been affected with it ever since he was sixty. But it may be that the same affection, which in young

subjects is progressive, is in older persons comparatively stationary, or runs so slow a course that death overtakes them before it has had time to develop itself fully. Dr Handfield Jones has, indeed, expressed an exactly opposite view of the matter, namely, that paralysis agitans in young subjects is a less serious form of the disease and curable, whereas in older patients it is incurable; but there seems to be no adequate evidence in support of such an opinion.

Charcot says that paralysis agitans is equally common in women and in men; but this is a mistake. In all Parkinson's cases, and in twenty-five out of our thirty-two cases, the patients were of the male sex.

Origin.—The disease seems frequently to arise without any definite exciting cause; but sometimes it follows close upon some violent shock of terror or other emotion. Charcot says that in many of his female patients it developed itself during the political commotions which have agitated France. The prolonged action of cold and moisture has been mentioned as occasionally giving rise to paralysis agitans, but apparently on no satisfactory grounds. It has sometimes followed a local injury, as in a case of Charcot's, that of a lady who severely bruised her left thigh in falling from a carriage, and in whom shortly afterwards that leg began to shake, and at a later period all the other limbs.

Diagnosis.—The recognition of paralysis agitans is seldom difficult. A disease which was until recently confounded with it is the insular or multiple sclerosis, described in a previous chapter; the points of distinction between them are clear (p. 547). One must not forget that local organic disease of the brain—a tumour, for example—may give rise to paroxysmal attacks of spasm in one arm, or in one arm and leg, that in themselves are not unlike those which occur at the commencement of paralysis agitans; but the history of the case and the other symptoms will generally prevent one from making a mistake in this direction. Hysteria may simulate it. I have notes of a girl, aged eighteen, in whom the right arm began to shake three weeks after a fright. When she was admitted into the hospital it was in a state of continuous agitation, and if it was held the other limbs began to move in a similar way. However, she had had a screaming fit the very day after her complaint began, and she had globus and headache. She was treated with frictional electricity, sparks being taken from her body, and she quickly recovered.

Prognosis.—Paralysis agitans is a disease which runs a very slow course. Charcot speaks of it as sometimes lasting thirty years. Towards the end, the patient falls into a very sad condition. The movements, which are now incessant at least while he is awake, may be so violent as to shake the bed, and even the room in which he lies. He is unable to get up, or to dress or feed himself without assistance. His speech may become unintelligible. His mouth remains open, and the saliva runs from it. His fæces and urine are passed involuntarily. Bedsores may form, and he may die of sheer exhaustion, or he may be carried off at an earlier period by pneumonia or some other intercurrent disease. Charcot remarks that a few hours before death the movements sometimes cease entirely.

As regards the *treatment* of paralysis agitans at an early stage, when one limb only is affected, it might be worth while to try the effect of keeping it at absolute rest for a period of several weeks; if necessary, by the aid of a mechanical appliance.

Even at an advanced period of the disease recovery sometimes takes

place, but probably not in consequence of medical treatment. Dr Elliotson supposed that he cured one case with the subcarbonate of iron; Brown-Séquard that he cured another with chloride of barium. In two of our cases the last-named salt was given, in doses of gr. ¼ to gr. j. In one it seemed useless; in the other marked improvement took place, the patient (who was under Dr Moxon's care) being able within six weeks to walk twice the length of the ward merely holding a nurse's hand, whereas he had been so helpless as to be unable to get in or out of bed, and for five years he was dressed by others. A man under two grains of valeriante of zinc three times a day went out of the hospital "cured," but the noise of a passing waggon, as he was walking home to Bermondsey, brought back the jerking movements which had ceased entirely for several days. Dr Ramskill (Syd. Soc.'s translation of 'Trousseau,' vol. i, p. 499) had a well-marked case in which recovery occurred, after the failure of other treatment, under the administration of four minims of phosphorised oil of the Prussian pharmacopœia in a drachm of cod-liver oil three times a day.

A plan recommended by Eulenberg consists in the subcutaneous injection of liquor potassæ arsenitis diluted with two parts of water. The quantities which he employed would correspond with from 1½ to 2½ minims of Fowler's solution. But Charcot has made trial of this treatment, and found it useless. Dr Shaw, the medical registrar at Guy's Hospital, in a report made for this second edition, states that we have tried nitroglycerine and physostigma without effect.

Eulenberg says that he has employed galvanism without any benefit—the constant current passed through the head or along the sympathetic nerves. We have had at Guy's Hospital one or two cases (among more failures) in which the application of this form of electricity down the spine caused temporary improvement. The most striking was in a patient of Dr Habershon's. It was first found that galvanisation for ten minutes was attended with marked benefit for the time. The electrodes were therefore fixed upon the neck for three hours without intermission. After this treatment had been continued for some time the limbs became much steadier, but he was not cured. Dr Wilks teaches that no medicines do any good, but that galvanism sometimes relieves.

THOMSEN'S DISEASE.*—Although cases of this remarkable affection had been observed by Sir Charles Bell, the first full and particular account of it was given in 1876 ('Arch. für Psychiatrie u. Nervenkr.') by Dr Thomsen, a Danish physician, who was himself, with several other members of his family, the subject of the infirmity. In the same year Seeligmüller published a characteristic case. Several others have been observed in Germany, France, Denmark, and Italy, and Dr Buzzard has recently described two cases in our own country ('Brit. Med. Journ.,' May 14th, 1887). A full bibliography was given by Dr Paul Chapman, in the sixth volume of 'Brain' (April, 1883), and a continuation by Dr Hale White in the same periodical for April, 1886.

Symptoms.—The peculiar features of the affection are that when the patient endeavours to perform voluntary movement after rest, the muscles of the limbs when used are thrown into tonic contractions which prevent

* Called by Thomsen "Tonic contractions in voluntary muscles, as the result of hereditary psychical predisposition." Strümpell suggested "Myotonia congenita" as a convenient title.

motion and keep the parts in a state of stiffness like that of cramp—though without pain. As in ordinary cramp, the contractions, though violent, are ineffective and irregular, portions only of the fleshy substance being raised into bulging elevations. After a time the difficulty is overcome and the patient is able to rise from his chair and walk, run, or perform any action he desires with his limbs. But the difficulty returns again and again, so as seriously to interfere with his daily life. Seeligmüller's patient was a healthy young recruit who found himself slow and awkward in performing his military exercises. Not only the arms and legs but the muscles of the trunk are subject to this difficulty, and also those of the tongue and lips, so that there results a kind of stammering—a violent effort to speak, inability to do so, and at last an explosive utterance, which passes into ordinary easy articulation for the rest of the sentence.

The involuntary muscles, as those of respiration and micturition and the sphincters, are quite unaffected. There is sometimes a feeling of numbness or "pins and needles" in the limbs, and occasionally pains, but these appear to be often absent and never severe or long continued. There is no anæsthesia. The knee-jerk and plantar reflexes are usually reported to be present, and sometimes excessive; but in one of Dr Buzzard's cases the former was completely and the latter almost completely abolished. The galvanic and faradic irritability of the muscles is greater than normal, and they readily respond to mechanical stimuli. That of the nerves is unaffected, and there is no R.D. Fibrillary tremors have often been observed, and the contraction of a muscle after stimulation continues long enough to be photographed.

There is no atrophy of the affected muscles. On the contrary, they undergo true hypertrophy, especially, it would seem, the gastrocnemii, external vasti, glutæi, and trapezii, but always in a symmetrical manner. Yet the muscular strength of the patients is not at all proportionate to this apparently athletic development.

Some authors have described the mental faculties and temper as being affected, but there seems no reason to regard this as a character of the disease. The patient is often intelligent and anxious to be relieved of his infirmity, but he feels awkward and unready; "before doing anything he must get his muscles ready," and hence is incapable of prompt and rapid movement in response to the will. The general health is unimpaired.

Pathology.—No autopsy has yet been made in a case of Thomsen's disease, so that its seat and morbid anatomy are completely unknown. The primary change may be in the muscles or motor nerves, in the motor tracts of the cord or in the cerebral cortex, or it is quite possible that there is no lesion discoverable by the scalpel or the microscope. Fragments of muscles have been removed by the "harpoon" (cf. pp. 526, 528), and the fibres have been reported by Erb to be decidedly thicker than usual, more cylindrical and with thicker sarcolemma; the striation is unaffected and there is no fatty or interstitial overgrowth as in pseudo-hypertrophic paralysis. Dr A. McL. Hamilton, of New York, and some other writers, have argued against the symptoms of Thomsen's disease being more than accidental or secondary. Dr Thomsen himself, as seen by the title under which he described it, regarded the affection as rather mental or emotional than organic in origin.

Ætiology.—The reality of the disease as a recurrent combination of symptoms is, it appears to the editor, amply established by its hereditary and

congenital origin. Dr Thomsen saw signs of it in his own children while still in the cradle; his own mother was moderately affected, and two of her brothers typically so; of her thirteen children, six, besides Dr Thomsen himself, were subjects of the infirmity; and of his own five sons, four inherited it from their father. Benedikt, Leyden, Eulenberg, Pitres and Dalledet, and other authors, found the same hereditary and family distribution. It appears to be far more marked than in any other affection of the nervous system, except perhaps Friedreich's disease, but like that rare and obscure condition, its duration is more extended laterally than vertically, *i. e.* it affects many brothers and sisters in a family, but does not so readily pass to the next generation, and rarely to a third. Both sexes are liable to be affected.

Course and prognosis.—The symptoms do not appear to be aggravated after they are once fully developed, but there is no instance of their having disappeared, nor is any treatment even supposed to be efficacious. The condition is probably as innate and ineradicable as stammering or timbre of voice or tricks of speech and gesture, and, like them, if ever "grown out of" the result is not obtained by direct therapeutical means, but by the gradual effect of circumstances and of time.

TETANY.*—In the spasmodic affections hitherto described the convulsive movements have been of a clonic kind. But there is one member of the group in which tonic spasm is the essential symptom. This is the disease to which Lucien Corvisart in 1852 gave the name of *tetanie;* previously it had been described by Dance in 1831 as "a kind of intermittent tetanus;" and other observers had designated it "idiopathic" or "rheumatic contraction of the extremities." Trousseau's lecture on this curious affection first drew attention to it in this country; the earliest case that I know to have been recognised clinically is one recorded by Dr Moxon in a paper in the 'Guy's Hospital Reports' for 1870. The name is unfortunate, for it is pathologically and clinically very different from the terrible disease always known as tetanus.

As may be supposed from some of the other names that have been given to it, tetany is generally confined to the limbs, and affects chiefly the distal ends of them. Sometimes it is limited to the forearms and hands; much more rarely to the legs and feet. Commonly it involves all four extremities at the same time or alternately.

The patient first has a sensation of tingling in the parts which are to be affected, and then begins to find that their movements are no longer free. Soon the thumbs become forcibly adducted; the fingers of each hand are closely pressed together, and are half flexed at the metacarpo-phalangeal joints but usually extended beyond; the palms are hollowed, by the approximation of their inner and outer surfaces. Hence Trousseau's comparison to the conical form of the accoucheur's hand when introduced into the vagina to perform turning. The nails may be driven into the skin so violently as to produce marks, or even (it is said) to give rise to sloughs. The wrists are half flexed, and the forearms strongly pronated. In the lower limbs the toes are bent down and adducted; the soles of the feet are hollowed, and the backs arched; the heels are pulled up by spasm of the muscles of the calves. The extremities on both sides are affected.

* *Synonyms.*—Tetanilla—Tonic cramps of the fingers and toes in children—Rheumatic contractions of the extremities—Remittent tetanus.

The contracted muscles feel hard; their resistance may be overcome by the employment of some force, which generally gives pain, but sometimes relief, to the patient. Thus the fingers and thumb may be straightened; but as soon as they are released they again become flexed as before. Another way of arresting the spasms is by means of cold. Trousseau mentions that persons in whom the lower limbs are affected can often regain the free use of them, for the time, by merely standing with naked feet on a stone floor; and in hands and forearms the same result can be brought about by immersing them in cold water.

During the paroxysms the affected parts have their movements much impaired. If, as is sometimes the case, the patient is a woman nursing a child, she cannot hold it in her arms. There is partial anæsthesia, the power of determining the size and hardness of objects being lost. In walking, the sensation experienced is the same as if the feet were treading upon a thick carpet. Pain is sometimes altogether wanting, but it is usually present in more or less severity, and is compared to that which accompanies ordinary "cramps" of the legs. Another common symptom is that the backs of the hands and feet become slightly tumid, and their veins a little prominent.

In from five to fifteen minutes the spasm generally passes off, but sometimes it lasts without stopping for an hour or two, or even longer still. As it subsides sensations of formication are again experienced.

After a variable interval another attack commences, and this goes on for several days, and even for two or three months. Even when a long period has elapsed the disease may return. A child, whose case is recorded by Dr Moxon, was first attacked by tetany when five months old, and had it on five distinct occasions, at intervals of from five to twelve months before it reached the age of three years and a half. One of Trousseau's patients, a young man of twenty-one, had the disease every winter for four years, the contractions coming on every day during a period of two months.

A curious fact which Trousseau pointed out is, that in a patient affected with tetany one can at any time bring on the spasms by compressing the principal nerves or arteries in the upper part of one of the limbs.

The electrical relations of the motor nerves and muscles in this disease have been investigated by Erb ('Ziemssen's Handbuch,' xii, p. 345), and by Kussmaul, Benedikt, and Chvostek and Weiss, with the result that the excitability both to faradic and to galvanic currents has been found greatly increased except only where the facial nerve is distributed (see Dr Buzzard's observations: 'Clin. Lectures,' pp. 414, 417).

Cases presenting the characters just described are decidedly of rare occurrence; only two or three have presented themselves at Guy's Hospital since the one which came under the care of Dr Moxon in 1870. One in a boy three years old, was described by Dr Wilks in a paper in the same Reports for 1872, and in his lectures he recounts two others in youths of sixteen and eighteen. But it appears likely that a very common affection is really a minor variety of tetany, namely, "carpo-pedal contractions," or "turning in" of the thumbs and great toes, which one looks for almost as a matter of course in rachitic children, whose nervous centres are in a state of irritation. Dr Hughlings Jackson regards these as rudimentary forms of epileptiform convulsions; but surely they bear a much more obvious relation to tetany.

It is by no means necessarily the case that in tetany the spasms are

limited to the extremities. Trousseau describes instances in which the face and trunk were also affected. In these cases the face became distorted; the eyes acquired a squint; the sterno-cleido-mastoidei, and the pectorales, were rigid; the recti abdominis stood up like tense cords. The jaws were firmly clenched, and the speech even was impaired, in consequence of the tongue having become involved. Laryngeal spasm sometimes occurred, causing lividity and apparent danger of suffocation. Febrile disturbance was also present. Yet, even in such severe cases, the patient would often get up; and if an adult, she would attend on other patients, although suffering from pains in the loins, and feeling bruised and exhausted. Sooner or later recovery almost invariably took place. Trousseau, however, mentions one instance in which death occurred from phthisis during a relapse of tetany, and another, which was supposed to have ended fatally within a few hours of its commencement. Dr Moxon has suggested that this was really a case of tetanus; but the hands and feet were characteristically affected; and these are the very parts which in that disease constantly escape the spasms of the tetanus.

Notwithstanding occasional extension to other parts, tonic spasm of the fingers and toes is the distinctive feature of tetany, and this enables us to include under that head another affection of early life; namely, *Trismus neonatorum*. It occurs in infants within a week after birth, and sometimes even in the first twelve hours. From Dr West's description of its symptoms it appears that, besides trismus and opisthotonos, there are powerful clenching of the hands, flexion of the feet upon the ankles, and bending of the toes; and he goes on to say that "when the fit subsides the child still lies with its hands clenched, and its thumbs drawn into the palm, the legs being generally crossed, and the great toe separated widely from the others." The head is thrown back; and the opisthotonos continues, although in a diminished degree. Before long the little patient becomes unable to swallow, and perhaps comatose; and death quickly ensues.

It may be thought that the presence of opisthotonos disproves the view above expressed concerning the "trismus neonatorum;" for it is true that Trousseau nowhere mentions by name that symptom as occurring in tetany. But he does speak of one patient as having all his muscles rigid, and being as stiff as a poker; and tonic spasm of the back of the neck commonly enough occurs in children in association with "carpo-pedal contractions."

The relation of these two affections to tetany is confirmed by their *ætiology*. The trismus of newly-born children was at one time supposed to be a traumatic tetanus, excited by irritation which started from the umbilical cord. But that notion was refuted by the subsidence of the disease in the Dublin Lying-in Hospital, in consequence of the introduction of an effective system of ventilation. Previously, one in every six of the infants born there had died when less than a fortnight old; and nineteen deaths out of twenty were due to trismus. Afterwards the mortality was only one in fifty-eight and a half, and but a ninth part of it was from the disease in question. I believe that in the West Indian Islands it is still common, and also in St Kilda; probably wherever it occurs it is due to a vitiated state of the air in the lying-in chamber. In London it must be of very rare occurrence, for Dr West saw only one case of trismus neonatorum.

In a valuable paper in the 'Lancet' (May, 1887) Dr Cheadle relates a case of tetany in an adult, and remarks on the association of this disease with previous diarrhœa or lactation in women. He believes that rickets in infants is apt to produce various spasmodic affections, which sometimes take the

form of infantile convulsions, sometimes of carpo-pedal contractions, sometimes of laryngismus stridulus, and sometimes, though less commonly, of tetany. Dr Eustace Smith and Dr Goodhart take the same view; and the latter writer quotes Mr Hutchinson's observation of the frequent coincidence of zonular cataract with infantile fits and rickets. Rickets has been present in every case of tetany seen at Guy's Hospital.

Trousseau speaks of tetany as occurring most frequently in women between the ages of seventeen and thirty, particularly in those who are suckling their children. One is tempted to think that there may be something in the diet adopted during the puerperal period by French physicians which lowers the health, and so brings on a disposition to the disease. Trousseau, however, speaks of it as being the direct result of exposure to cold. The woman, whose case he records as having terminated fatally, had shortly before been delivered, and had suffered from obstinate diarrhœa. On several occasions she got out of bed in the night, and went to fetch water from a fountain in the yard of the hospital; indeed, she did this on the very night before she died. Trousseau described diarrhœa as a frequent predisposing cause of tetany; he had also seen it after enteric fever and after cholera. He had met with instances of it in women over forty years of age and even in adult males. Tetany sometimes occurs as an epidemic neurosis in girls' schools, and in convents. Such cases bring it into relation with hysteria, chorea major and the extraordinary attacks of "convulsionnaires."

The immediate *pathology* of tetany is quite unknown; and it has no ascertained morbid anatomy. It has no connection whatever with rheumatism, in any intelligible sense of the word; all that French and German writers meant by calling the contractures "rheumatic" was that they occasionally follow exposure to cold. Dr Hughlings Jackson suggests the hypothesis that cerebral influence is diminished and that (supposed) cerebellar influence over the motor nerves is increased.

Dr Moxon has remarked that the disease which has been described as spasmodic ergotism, and which was prevalent in certain parts of Germany in the earlier part of the present century, was similar in its symptoms to tetany. Mr Wright's account of it ('Edin. Med. Journ.,' 1839) almost proves that the two disorders are identical, and that the former was not due to a specific poison, but to general deterioration of the health from a defective supply of nutriment. He states that in the eighteenth century a similar disease occurred in Sweden. (See also on this point a paper by Bauer in the 'Berliner klin. Wochenschr.,' 1872.)

The *diagnosis* of tetany is not difficult, spinal meningitis, especially epidemic cerebro-spinal fever (p. 690) being the only affection with which it is likely to be confounded. There is no rise of temperature in tetany, and when it occurs epidemically it is obviously a neurosis.

The *prognosis* is probably always favourable in genuine cases, but death may occur from general convulsions or other complications to which rachitic children are liable.

The *treatment* which Dr Moxon adopted in his case of tetany consisted in the administration of bromide of potassium in five-grain doses; in four days the spasms ceased to return, and the child afterwards took cod-liver oil and steel wine. There can be no question that such remedies are to be preferred to bleeding and cupping as suggested by Trousseau. Indeed, that writer himself makes an exception for weakly and debilitated subjects, for whom he recommends quinine with small doses of opium or belladonna. In

severe cases he says that the inhalation of chloroform has sometimes given marked relief. He mentions a case which Aran cured by applying to the affected parts pieces of linen soaked in chloroform. Dr Wilks also finds cold applications the best.

Fresh air and exercise and moral discipline are probably the best treatment in many cases. In infants, chloral hydrate (with or without bromides), followed up by good milk, cod-liver oil, meat juice and other nutritious food, will in most cases cure by removing the cause as well as the symptom, in cases of tetany, or carpo-pedal contractions or trismus. Dr Cheadle believes that Calabar bean will sometimes succeed in overcoming the spasms when other measures fail.

CHOREA.*—Like some other diseases, this is now universally known by a name which was at first applied to quite a different complaint.

The term *chorea Sancti Viti* was originally applied to the dancing mania which prevailed in certain parts of Germany in the fourteenth and fifteenth centuries. It was also known as St John's dance. It is said that the designation is first met with in the account of an epidemic at Strasburg, in 1418, when those who were attacked were sent by the authorities to the chapel of St Vitus, at Zabern, to be calmed by religious processions and masses.† Such cases will be hereafter mentioned under hysteria, and are related to some forms of saltatory spasm. They are altogether unconnected with the disease now to be described, to which the name of chorea was first applied by Sydenham.‡ The use of an old appellation in a new sense could not but tend to confusion, and for a long time afterwards Sydenham's chorea was distinguished as "chorea minor," or "chorea Anglorum."

Symptoms.—These are chiefly two: an inability to keep at rest while awake, and an incapacity for performing voluntary movements with precision. If the patient means to sit or to stand still, she (for chorea is more common in girls than in boys) begins after a few seconds to fidget. She shuffles her feet over the floor, or throws one foot over the other, or twists it first outwards and then inwards. Or, perhaps, she lays her hand palm upwards upon her lap and then suddenly reverses it. Or she may shrug up one shoulder, or throw it forwards, or open and close her mouth or her eyelids without purpose. The muscles of the face are continually working. She probably is herself unaware of some of the movements, as, for instance, when a squint develops itself, or when her eyeballs are jerked from side to side. If one asks her to show her tongue she often seems at first unable to put it out, but afterwards she suddenly thrusts it forwards and then as suddenly withdraws it, her jaws snapping together in front of it. If she wishes to carry a cup to her mouth she cannot help throwing her arm in various directions, and accomplishes her object only

* *Synonyms.*—Chorea Sancti Viti minor—Danse de Saint Gui—Chorea minor.

† See curious historical details in Hecker's 'Epidemics of the Middle Ages.'

‡ "De Chorea Sancti Viti: Convulsionis species quædam est, pueros puellasve a decimo ætatis anno ad pubertatem invadit, &c. . . . . Antequam poculum ad os possit adducere, mille gesticulationes, circulatorum instar (like a mountebank), exhibebit. Non enim rectâ lineâ ori admovet, sed deductâ a spasmo manu, huc illuc aliquandiu versat, donec tandem forte fortunâ labris proprius apponens liquorem, repente in os injicit et avide haurit" (Sydenham: 'Processus integri in morbis curandis,' 1693). It is remarkable, that while Romberg calls Sydenham's original description of chorea masterly, and von Ziemssen says that it leaves nothing to be desired for clearness and precision, Dr Sturges thinks we must suppose either that the period and manner of its attack have changed since Sydenham's time, or else that this observer was in gross error in his description of what he saw.

after several unsuccessful attempts, and then she perhaps seizes the edge of the vessel with her teeth, and is obliged to gulp down all its contents at once for fear of spilling them. When she tries to walk she moves by fits and starts, and jerks her body and limbs first to one side and then to another. Attempts at voluntary movement bring on the choreic symptoms worse, whereas in slight cases a child may lie in bed or sit still without showing the disease.

The respiration is disorderly in rhythm. The articulation is apt to be hurried. The patient, if told to count, may give out several numbers one after another with explosive violence, and then pause to take a deep breath; or she may utter only one sound with each expiration, drawing in air hastily before she goes on to the next. Von Ziemssen says that he has seen with the laryngoscope unsteady and quivering movements of the muscles of the glottis, and that an imperfect degree of tension of the vocal cords is shown by the low pitch and monotonous character of the voice, and by the short space of time during which a note can be kept up in singing. Romberg relates a case in which there was annoying hiccough, and two other instances in which inspiration was attended with a whistling or snapping noise; in one of them the chest was observed to be suddenly drawn inwards by spasm of the intercostal muscles. According to Trousseau deglutition is sometimes impaired.

During sleep the choreic movements cease. Jaccoud also remarks that in slight cases they sometimes remain absent for a little while after the patient awakes. Marshall Hall, however, is said to have observed that if she dreams they may for the time return.

Rosenthal and Benedikt have tested the reaction of the muscles to faradic and galvanic currents, and have found their excitability greater than under normal conditions.

When chorea is slight or of moderate severity, the spasms are often confined to one side so far as the limbs are concerned. The disease is then sometimes called "hemichorea," but a special name is hardly needed. Dr Hughlings Jackson has pointed out that the muscles of the trunk and face are always affected bilaterally, a fact which I shall presently show to be of considerable theoretical interest. Different writers have made different statements with regard to the relative frequency with which the right and the left limbs are affected. According to Dr Jackson the former more commonly exhibit the movements; according to Jaccoud, the latter. Dr Anstie also remarks that one may often at the first glance recognise a child affected with slight chorea from her sitting with her right hand grasping her left wrist to keep it still. Among thirty-three cases out of 150 in which the affection was unilateral, there were eighteen on the left to fifteen on the right side* ('Guy's Hosp. Reports,' 1873, "Rheumatism and Allied Disorders").

The sensibility of the skin is generally said to be unaffected, but Trousseau declares that it is almost constantly disordered, the patient experiencing formication and tingling, especially on the affected side, or even anæsthesia. Numbness is not uncommonly mentioned in the reports of cases admitted into Guy's Hospital. Choreic patients often complain of headache. Sir Thomas Watson remarks that he has sometimes known the pain to be limited to the side of the head opposite to the limbs which prevented the

* Since then, among 164 additional cases I find the affection decidely predominant in the right arm or leg, or both in seven cases; in the left in thirteen (1887).

movements. The pupils are generally torpid and rather widely dilated, and in one case von Ziemssen observed that the pupil was much larger in the eye corresponding with the affected arm and leg. Rosenthal noted the fact that the pupils returned to their normal condition when the chorea subsided.

Children affected with chorea are generally pale, often decidedly anæmic. They may also be thin and delicate in appearance, but this is generally due to previous rheumatism and cardiac disease, at least in some of the more marked cases; and chorea may be seen in stout and healthy children. We must remember that the favourite age of the disease is not early childhood, but the period between eight and fourteen years of age, when both boys and girls, even when in sound health, are usually slender and often pale if their appearance is compared with the rosy cheeks and plump limbs of their earlier childhood.

The temperature is almost always normal.* The urine, if it deviates from the normal state, is commonly scanty and high coloured, in marked contrast with the pale fluid which is passed so abundantly by females who are hysterical. According to Dr Bence Jones, there is an excessive secretion of urea, and others have said that the amount of urates is increased. Analyses made in two cases by Dr Handfield Jones ('Clin. Trans.,' iv) yielded contradictory results.

The pulse is commonly quickened, especially when the movements are severe. Some writers assert that the heart's action may be irregular or intermittent, but this is in the author's experience quite exceptional. On auscultation over the cardiac region a blowing *systolic murmur* is often audible. It is not audible at the apex, and is rarely heard in the axilla and back. With regard to the significance of this sign widely different opinions have been held. Many observers have maintained that the bruit is functional; some that it is anæmic, others that it is due to choreic spasms of the musculi papillares interfering with the due closure of the mitral valve. But, as we shall presently see, when the disease happens to terminate fatally, that valve is almost invariably found to have vegetations upon it which are identical with those that are present in acute rheumatism. And since the murmur also is the same in the two diseases, it seems reasonable to attribute it to the endocarditis in the one as much as in the other. In many cases no bruit is audible. Its absence, however, is not to be taken as a proof that the valve is unaffected, for Kirkes and Wilks have both recorded instances in which vegetations were found after death, but in which the heart-sounds during life had been normal. Again, in some cases in which a murmur is present it passes off as the patient recovers from the chorea. This fact has been regarded as a proof that at least in these cases the sound is due to functional disturbance and not to valvular inflammation; but such a conclusion is not warranted, for a precisely similar disappearance of the murmur is often observed in cases of rheumatism.

Among 150 cases of chorea in Guy's Hospital during the years 1870–71–72, the state of the heart is not mentioned in eleven, and the

* The temperature cannot be taken in the mouth or the axilla. The rectum, or the stream of urine (p. 37, note), are the proper places for the thermometer. The fact that the constant muscular movements do *not* raise the temperature of the body is of great physiological importance. Dr Woodman and Dr Long Fox (Wunderlich, Syd. Soc. Trans., p. 426) have found it as low as 97° or 96°, although, on the other hand, Dr Finlayson's case is quoted (probably complicated), in which the average evening reading of the thermometer *in recto* was 103·2° (cf. *infra*, p. 744).

presence of any abnormality is expressly denied in eighty. A systolic bruit was heard in the remaining fifty-nine, and in forty-three of them it was distinctly loudest at the apex. In only nine is it described as basic; two of these were indistinct and two were combined with a diastolic murmur.

Among 180 fresh cases in the same hospital during the five years 1874–78, collected for the editor by his house physician Dr George Halstead, sixty-two were reported to have a murmur of some kind (again about a third of the cases); of these it was an apex systolic bruit in fifty-two, a basic bruit in seven, of which two were diagnosed as aortic and two as pulmonary, a præsystolic apex bruit in two, and a pericardial rub in one.

The meaning of these results will be best discussed when we come to consider the pathology of chorea and its relation to rheumatism.

Varieties.—In some cases of chorea the symptoms are but little marked at its commencement; and even throughout its whole course the patient may exhibit comparatively slight movements. A much more prominent feature may then be *loss of power* in one or more of the limbs. Thus, she may complain that her arm feels heavy, or she may drag her arm slightly in walking. Such cases are often brought to one as paralytic. In other instances she merely has a trick of bringing a particular set of muscles unexpectedly into action, so that she makes a grimace, or throws her hand or arm into an odd position. Or she may let a jug fall, and this may happen two or three times in succession in the course of a week or ten days. Or it may be noticed that she does not write her exercises so well as she used, or that she no longer plays steadily on the piano. All these things are apt to be set down to carelessness, and her parents and teachers are the more likely to blame her unjustly, because she commonly exhibits a change in her disposition, becoming inattentive and forgetful, apathetic, peevish, and ill-humoured, or extravagantly gay and excited in her manner.

Severe and dangerous cases on the other hand also occur, but happily as the exception, when the disease reaches a far higher pitch of severity than has hitherto been described. The patient becomes unable to stand; even when she is recumbent her limbs are tossed about in all directions, so that boards well padded have to be fixed on each side of her bed to prevent her throwing herself on to the floor. In spite of all the precautions that can be taken she is very apt to bruise herself and to rub off the skin from her elbows and knees, so as to cover them with crusts and sores. She ceases to sleep altogether, and the violent movements go on day and night without intermission. In such cases emaciation takes place with wonderful rapidity. Dr Tuckwell has related the case of a boy whose flesh was wasted to the utmost at the time of his death, but who six days previously had borne the appearance of vigorous health, with remarkably well-developed muscles; and conversely von Ziemssen mentions one in which a girl, eleven years old, gained during convalescence 5½ lbs. in ten days, her weight rising from 55½ to 61 lbs. in that time. One cause of the extreme wasting which accompanies severe chorea is doubtless the difficulty with which food is administered. The patient often seizes a spoon, or the spout of a feeding bottle, as if she would bite it in two, and so as actually to break her own teeth, or perhaps she wounds the fingers of her attendants. Indeed, such injuries are not always inflicted accidentally, for the mental state of a patient affected with severe chorea is often one of mania. She may be continually shouting, singing, and talking at random. Or she may unexpectedly perform some strange action; for instance, a girl who was in hospital some years ago,

suddenly got out of her own bed, and turned a somersault across that of another patient. There is no necessary relation between the intensity of the choreic movements and that of the pyschical disturbance. Dr Wilks says that the mind remained perfectly clear in one of his patients, who had the worst attack of chorea which he ever saw terminate favourably. On the other hand there occurred some years ago in Guy's Hospital a fatal case in which there were such marked mania and so complete a loss of consciousness, while at the same time the movements were so jactitating and like those seen in epilepsy, that the diagnosis remained doubtful until the autopsy was made, when recent vegetations were found upon the mitral valve. Dr Hills, of the Norfolk County Asylum, once stated that more than one patient suffering from chorea had been sent to that institution as insane. Loss of power of speech is common in all but slight cases of chorea and is universal in chorea gravis.

Course.—Chorea may be said to be generally a chronic disorder, but its duration is very variable. Almost all the statistics which have been published concur in stating a period of from two to three months as the average. Thus Wicke found it eighty-nine days in a series of 125 cases, and Sée sixty-nine days in one of 117 cases; while from much smaller data Gray, Tuckwell, and Hillier in this country each made it about ten weeks. Individual cases, however, range widely on each side of these limits; but far more widely *beyond* the average duration than *within* it. A necessary consequence is that the introduction of one or two very prolonged cases may greatly disturb the statistical result; even in this series of Drs Gray and Tuckwell ('Lancet,' 1871), in which the longest case did not extend beyond twenty weeks, there were only six cases above the average to ten below. But we shall see that patients sometimes apply for treatment in whom the disease has already lasted for many months or even for some years. A single instance of this kind would inevitably spoil, for the purposes of comparison, any series which did not embrace a very large number of cases. A point of considerable importance is that, as a rule, chorea is more likely to last for a great length of time when its symptoms are comparatively mild; patients who are obliged to have boards placed along the sides of their beds often get well before those who are able to be up and about the ward throughout their stay in the hospital.

Fatal event.—Children, who are the chief sufferers from chorea, very rarely die of it. At Guy's Hospital it appears that between the years 1848 and 1875 there have been in all twenty cases of the disease which terminated fatally. But five of these must be left out of consideration, since death was due to an accidental complication (such as dysentery or diphtheria) or to concurrent rheumatic pericarditis, cardiac dropsy, or the like. Now, of the remainder there are only four in which the patients were below the age of fourteen: two were seven years old, one twelve, and one thirteen. The remaining eleven patients were more than fourteen years old, nine of them between the ages of fifteen and eighteen, one forty years old, and one fifty years old. Few of these women seem to have been pregnant; but it is an ascertained fact that the mortality is very great in this condition, which frequently ends in abortion or premature delivery. It seldom happens, even in the most severe forms of chorea, that the patient dies within three or four weeks from the commencement of the disease. One of the most rapid cases on record is the one referred to above, in which there was at first a doubt whether the neurosis was not epilepsy rather than

chorea ; the whole duration of the patient's illness was said to be only two or three days. In 1853 a girl, aged sixteen, who was already in the hospital for syphilis, was attacked with maniacal chorea, and died in six days. About two months before another case, in a boy of the same age, had terminated fatally in nine days. The immediate cause of death seems to be almost always the intensity of the nervous symptoms themselves, but the movements often subside, and may even cease entirely during the last few hours, the patient lying comatose and passing urine and fæces involuntarily. The temperature of the body may rise during this period of the disease. I made an autopsy in one case in which it was 104·7° before the patient died, and Dr Frederick Taylor had in our clinical ward a case in which the thermometer registered a temperature of 108° immediately after life was extinct. It does not appear that endocarditis or pericarditis is directly concerned in bringing about the fatal issue, or, indeed, that its development affects in any way the progress of the disease. The presence of bronchitis has been noted in several of the reports of autopsies at Guy's; in one instance it perhaps accelerated the patient's death. It has been supposed to have been caused by exposure to cold, owing to the bedclothes having been thrown off in the violent movements. In 1873 Dr Habershon had a fatal case in a boy, aged twelve, who had been three weeks in the hospital when his breathing became obstructed by swelling of the tongue. Tracheotomy was performed, but without saving the patient's life. Probably the glossitis was the result of injuries inflicted by the teeth, for in another instance, in which the tongue had been severely bitten in two places, there was discovered after death a foul ulcer which exposed the sublingual gland and extended for the depth of an inch into the muscular and other tissues.

According to Dr Radcliffe and von Ziemssen meningitis occurs in some cases of chorea, and in others myelitis ; and they accordingly mention those affections as sometimes bringing chorea to a fatal termination ; but we may venture to doubt the diagnosis of any case in which unmistakable inflammatory changes were not discovered either in the membranes of the brain or in the spinal cord.

Four fatal cases occurred among the 150 tabulated by Mr Manser for the 'Guy's Reports' (Third Series, vol. xix, pp. 329–32). 1. F., thirteen, 5th March. Mitral and aortic vegetations, ulcer from biting tongue. 2. F., seven. Aortic vegetations, faucial diphtheria. 3. F., pregnant; mitral vegetations. 4. M., thirty-eight. Aortic and slight mitral endocarditis.

Among 180 cases, collected by Dr Halstead from the records of the same hospital, during the five years following those which furnished the above statistics, there were only five deaths. In a little girl of seven, who died after being six weeks in the ward, there were found the usual fibrinous vegetations on both mitral and aortic valves. In a girl of fifteen, after a month's treatment, there was, beside the same almost constant lesion, lobar pneumonia and a bedsore. In a girl of eight there were mitral and aortic nodules with broncho-pneumonia and adherent pericardium. In a girl of fifteen there was mitral endocarditis, adherent pericardium, and a "cardiac" lung. In a lad of seventeen, who had suffered again and again from chorea since he was nine years old, no lesion but mitral vegetations was found.

If to these nine fatal cases we add the seven additional cases numbered 5–10 in the paper above referred to (vol. xix, p. 333), we have a total of sixteen *post-mortem* examinations. In every one of these there were fibrinous nodules found on the mitral or aortic valves. In only two of these there

was a history of rheumatic fever, and in seven its occurrence was explicitly denied. Diphtheria was apparently the cause of death in three cases, but it would perhaps be bold to call it purely accidental. In three cases there was acute lobar pneumonia, and in a fourth lobular inflammation of the lungs. Only one of the patients was pregnant.*

Recovery.—This is generally gradual. Sometimes, however, the breaking out of an exanthem, or of some other febrile disease, is followed by the sudden subsidence of the movements. This is in accordance with a maxim of Hippocrates : "Spasmos febris accedens solvit." Dr Radcliffe states that he has met with seven cases in which it occurred; and some striking examples of a similar kind are recorded by Rilliet and Barthez. According to the observations of Sée, however, the neurosis is not likely to be cut short unless it had reached its acme of development before the commencement of the fever. In many instances the first effect of the latter is to aggravate the movements, which yet may in those very cases cease a short time afterwards when the crisis of the acute disease has passed

It sometimes happens that the subsidence of the spasms in severe cases of chorea is not followed by any evident amelioration in the patient's general condition. She may become perfectly quiet, and yet may remain for two or three weeks unable to stand, and possessing very little power in her arms. Or there may be a paralysis limited to a single limb, or to the two limbs on one side, the "choreic hemiplegia" of Dr Todd. In association with such symptoms, or independently of them, the intelligence may remain defective; or a condition of mania or melancholia may develop itself. Dr Barnes met with a case in a pregnant woman, who as she regained her strength became insane and had to be removed to Bethlem. Other patients, after the choreic movements have passed off, still remain absolutely silent for days together, making no attempt to reply to the anxious inquiries of relatives and friends. The observations of Dr Hughlings Jackson have associated such defects of speech with a preponderance of spasm in the right limbs, or limitation to them.

Alarming as these various symptoms are, they almost always pass off in their turn, and the patient ultimately regains a state of perfect health, at least so far as the muscular and nervous systems are concerned.†

The strong tendency of chorea to relapse again and again will be mentioned further on.

Pathology.—The questions of the nature and causes of chorea are so intimately connected together that it is impossible to discuss the one apart from the other. They involve a series of problems which are of great interest, and which have important bearings upon the ætiology of the neuroses in general.

In the first place, there are still differences of opinion as to whether the disease has its seat in the spinal cord or in the encephalon. When the

* Among 439 cases published by the Collective Investigation Committee only nine deaths occurred ('Brit. Med. Journal,' February 26th, 1887).

† Trousseau, however, speaks of children who have never again shown the same degree of intelligence as before; von Ziemssen says that slight defects in the coordination of the movements, a precipitancy in the performance of certain manual actions, or a tendency to facial grimaces, may persist for years or even be permanent; and Dr Radcliffe is inclined to think that the disease is apt to be followed by other neuroses, particularly epilepsy, at a later period of life. Dr Bristowe mentions a case in which there was a degree of rigid flexion of the hip- and knee-joints, with overlapping of the knees from preponderant action of the adductors of the thighs, and a tendency to talipes equino-varus,—conditions which seemed to him to indicate degenerative changes in the lateral columns of the cord.

functions of nervous centres in the cord were first discovered, and when it was found that frogs and other animals could perform coordinated movements after excision of the cerebrum, it was natural that attempts should be made to refer to disorder of those centres all affections of which the main symptoms are irregular motions of the body and limbs. Thus Romberg, writing in 1851, placed chorea without limitation among the "spinal spasms;" and so recently as 1873 Jaccoud defended a similar doctrine in an elaborate argument. In England, however, the opinion has for some years prevailed that the disease is seated in the sensori-motor ganglia, at the base of the brain, and especially in the corpora striata.

In favour of the spinal theory of chorea, certain vivisections have been adduced. It appears that dogs are liable to a similar complaint. Now, Chauveau, of Lyons, conceived the idea of cutting through the cord close to the skull in such animals, and he found that after this had been done the movements went on in exactly the same way as before, the spasmodic contractions of the diaphragm being in one instance sufficient to keep the dog alive for three hours. In two other experiments that muscle happened to be paralysed, so that artificial respiration had to be maintained; but the result was the same as in the first observation, as far as concerned the choreiform spasms. And one further point was made out, namely, that a second division of the cord, at the bottom of the dorsal region, put an end to the motions in the tail and in the other parts beyond the section. Chauveau's experiments have been repeated by Legros and Onimus; and they have ascertained, in addition, that by irritation of the posterior columns of the divided cord the spasms were increased, while they were arrested by complete section of the same structures.

It is, however, now ascertained that canine chorea is not identical with the disease of that name in man,* and, even if it were, the question arises, whether in the human subject the higher nervous centres do not assume functions which in brutes are performed by lower ones. English pathologists have been able to make out a strong case in favour of their own opinions. Some of the points were stated by Dr Russell Reynolds as far back as 1855. As he remarked, the spasms produced by persistent irritation of the cord are tonic rather than clonic; and it is further inconsistent with a spinal origin that the choreic movements should be in any degree capable of being controlled by the will, that they should be increased by emotions or by voluntary efforts, that they should cease during sleep, and that they should be diminished by direction of the patient's attention to other objects. In addition, Dr Broadbent ('Brit. Med. Journ.,' 1869) has insisted on the fact (which Romberg had previously observed) that tickling the palm of the hand or the sole of the foot of a child affected with chorea leads to no increased spasms; on the contrary, it is borne without difficulty, and the tendency to the excitation of reflex actions sometimes seems to be less than in health. Another point on which he lays still more stress is that the spasms are so often unilateral. An affection of one half of the spinal cord throughout its whole length, from the crus cerebri downwards, without implication of the other half, is, he declares, scarcely conceivable. And the improbability of such a localisation of the disease is indefinitely increased by the fact that the muscles of the face and trunk are bilaterally affected, these very muscles being liable to be set in action on both sides by stimuli derived from a single corpus striatum, whereas there is no reason to suppose

* See Mr Victor Horsley's Lectures ('Lancet,' 1886, vol. i, p. 54).

that disturbance of one half of the cord could affect them in a similar manner. Lastly, the fact that the mental faculties are so frequently impaired in severe cases of chorea would naturally incline one to localise the disease in the closest possible proximity to the hemispheres, since it must be due to an extension of the morbid process to the highest cephalic centres.

Dr Dickinson made ('Med.-Chir. Trans.,' 1876) a series of anatomical investigations in fatal cases of chorea, from which it would appear that certain morbid changes, consisting of congestion of the vessels, of peri-arterial degenerations, and of minute spots of scleroses, are discoverable in the upper regions of the spinal cord, as well as in the lower parts of the brain. These observations of Dr Dickinson's have not yet been confirmed by other pathologists; but even if the constant occurrence of the appearances which he has described should be fully established, it would still be a question whether they are not merely secondary effects of the disease. Whatever view one may hold as to the starting-point of the choreic spasms, one cannot doubt that all the nervous structures which lie between it and the muscles must have their functional power strained to the utmost during the continuance of such violent movements; and therefore we ought to expect that they should all exhibit any degenerative changes which may after a time develop themselves. It is important to note that Dr Dickinson's cases had all reached an advanced stage. Moreover, it is clear that the persistence of the morbid changes which he describes is compatible with the subsidence of the spasmodic movements and the restoration of health. For in one patient who had twice before had chorea (the last time having been a year previously), the duration of the fatal attack was only thirteen days. And yet changes of old date—peri-arterial degeneration and scattered spots of sclerosis—were found, besides recent congestion of the spinal cord and of the sensori-motor ganglia.

On the whole, then, the corpora striata seem to be a more probable seat of chorea than the cord—one of these ganglia being affected when the disease is unilateral, both of them when all the four limbs take part in the movements.

Another possibility, however, is that the seat of the "discharging lesion" in chorea is the motor region of the cerebral cortex, and many of the physiological as well as clinical objections to the other proposed localities do not apply to this—partly perhaps because its functions are still less perfectly investigated.

The next question is as to the nature of the anatomical change. Dr Broadbent maintains that the spasmodic movements which characterise the disease are not significant of any one lesion, but indicate that the functions of a particular part of the brain are deranged. To adopt his language, chorea is "a symptom not a disease. It has been called an insanity of the muscles; it would be better designated a delirium of the sensori-motor ganglia, since it bears the same relation to those parts that the delirium which may occur in a variety of maladies bears to the cerebral hemispheres."

Now, it is possibly true that choreiform spasms may accompany different morbid states of the nervous centres. But it is no less true, and far more significant, that the disease described in the preceding pages as chorea—instead of being an accidental complication of other maladies—occurs chiefly in a particular class of patients, and under circumstances peculiar to itself.

Dr Sturges in his interesting 'Lectures on Chorea' (1877) points out with much acumen how choreic movements may be paralleled by the nervous twitchings of mental embarrassment or the restless and purposeless movements of a fidgety child. Yet admirable as is the critical and negative part of his remarks, it remains true (1) that if a functional disease, the symptoms of chorea must yet depend on functional disturbance (*i. e.* disordered nutrition from irregular supply of blood, or from some poison, or from a molecular change of other origin) which affects one part of the nervous muscular apparatus and not another. Syncope results from anæmia of the cerebrum, not of the liver; tetanus from strychnia affecting the cord, not the bulb; and epilepsy from some unknown minute derangement in the cells of the cortex, not in those of the retina. Our business is to fix the "seat" of a disordered function as much as of an impaired structure. (2) It remains true that while no line can be drawn between health and disease, or between the greatest premonitions of an impending malady and the slightest early symptoms of it when developed, we must nevertheless recognise certain limits where variations, which are slight or irregular or individual or recoverable, become serious enough to threaten comfort or life, or where they lose the accidental character of individual variation and assume the constant and common characters of "recurrent concomitant symptoms" (Symptomencomplex). We then know that a disease is before us, according to the only reasonable definition of the word; a condition which has to be understood and treated by a skilled adviser. (3) Like other diseases, chorea has its own natural history, its beginning, middle, and end, its proclivities, and its antipathies. In fact according to principles laid down in the first chapter of this book, there is scarcely a member of the whole nosology which better deserves to be called "a disease" than St Vitus's dance.

Sex.—Chorea is much more common in *females* than in males; and (like hysteria) it shows its predilection for the former sex in the case of children before the age of puberty; but, unlike hysteria, it is much more apt to occur in children than in adults. Tabular statements in regard to these points have been published by the editor in the nineteenth volume of the 'Guy's Hospital Reports,' Third Series. He found that among a hundred and fifty patients 42 were males and 106 females. These numbers were extracted by Mr Manser in 1873. Mr Halstead found the corresponding figures for 1874–78 were 43 male to 129 female patients. The late Dr Hughes, among 100 cases found 27 boys and 73 girls (*ibid.*, Second Series, vol. iv, p. 372), and among 209 cases 42 male and 167 female patients (*ibid.*, Third Series, vol. i, p. 245). So that the proportion in a large London hospital among in-patients and out would be less than one fourth male to three fourths female (154 to 475). Of 531 cases treated in the Hôpital des Enfans Malades at Paris during twenty-two years,* 138 occurred in boys and 393 in girls, *i. e.* rather more than one to three, in consequence of what we shall presently see to be the smaller excess of female patients among those under puberty. Among 422 children under twelve treated for chorea at Great Ormond Street, Dr Hillier found 122 boys to 300 girls, again rather less than one to three. Among 141 children at the Evelina Hospital, Dr Goodhart had 43 boys and 98 girls. A large proportion of boys was also found by Rufz at the Children's Hospital at Paris from 1824 to 1833 (overlapping the first three or four years of Sée's period at

* Sée 'De la Chorée et des Affections Nerveuses,' Paris, 1851.

the same institution), viz. 51 to 138; and in Wincke's monograph on chorea published at Leipzig in 1844, the proportion was 117 to 210, or more than half the patients were boys, whose ages ranged from four to eighteen. If we put together all the above statistics (which, with the partial exception above noticed in the Paris cases, are independent of each other) we find that the total numbers are: of 2030 patients, 583 male to 1447 female, or a proportion of about 2 to 5.*

Age.—Chorea is characteristically a disease of childhood, seldom or never seen in infancy, and rare after twenty, except in the case of puerperal or pregnant women. Sydenham's lower limit of "the tenth year" is, however, too high. From the fourth or fifth to the fifteenth year for boys and to the seventeenth for girls is the choreic period.

Of 216 patients with chorea, observed in Guy's Hospital, 187 were between six and seventeen years old. Of *first attacks*, among 322 patients recorded there, only two were two years old, and none under that age. The disorder first appeared in five patients between two and five years old, in 102 between five and ten, in 134 between ten and fifteen, and in 55 between fifteen and twenty. Only two male patients were over twenty when first affected with chorea, one not yet twenty-one, the other thirty-eight; while there were six young women between twenty and twenty-six.

Of Dr Hughes's hundred patients in Guy's Hospital, only one was under eight years old (a boy of five), thirty-two were between eight and ten, forty-five between ten and fifteen, twenty between fifteen and twenty, one (a young man), was twenty-five, and two young women were twenty-two and twenty-eight. In his second series of 198 cases, nine were between four and eight years old, 148 were between eight and sixteen, and above sixteen there were only four men aged 18, 20, 21, and 43.

In Paris, Sée found that out of 531 cases of chorea, 453 occurred between six and fifteen; in Philadelphia, Hammond found the proportion 67 out of 82, and in Germany, Rufz gives the corresponding numbers to be 180 out of 189.

Except in pregnant women chorea is extremely rare over twenty-five, but we had what was an unexceptionable case in a man of thirty-eight, and several authentic instances have been recorded of the occurrence of chorea at an advanced age. Dr Graves mentions the case of an apothecary in Dublin who was attacked when seventy years old; Romberg saw a case in an old woman of seventy-six having, however, began at six;† and Trousseau relates in detail an instance which came under the observation of Dr Henri Roger, in a lady, aged eighty-three, who recovered from the disease in five weeks. Charcot has described senile chorea as a distinct variety of the disease, but we may perhaps be allowed a doubt whether all these cases would stand criticism. On the other hand, examples are not altogether wanting of the presence of chorea in new-born infants. Thus Richter is quoted by von Ziemssen as having recorded two cases in each of which a female child was affected at birth, the mother having received a fright while advanced in pregnancy; the infant suffered while awake from clonic spasms, which were absent during sleep, and afterwards ceased almost entirely. A somewhat similar instance is related by Dr Long Fox as having occurred congenitally in an infant born six weeks before the proper time.

* Among 436 cases of the Collective Investigation Committee the proportion of the sexes was 115 to 322, or a little more than 1 to 3.

† Among the 439 cases of the Collective Investigation Committee there were only 10 above twenty-five years old. Five of these were old women between sixty-three and eighty-six.

Still the fact remains, that in the great majority of cases chorea is a disease of childhood between the ages of six and fifteen years, or between the period of the commencement of the second dentition and that of puberty. The preponderance of females among those who are attacked by it is doubtless due to the peculiar sensitiveness and mobility of their cerebral organisation. It accords with this supposition that the children most liable to suffer are those who are delicate and excitable. As to the question whether this disease is especially apt to occur in those who inherit a tendency to the neuroses in general there is some difference of opinion. Trousseau and Anstie answered this question affirmatively, Sée gave a contrary opinion.

Among persons more than seventeen years old the preponderance of females is far greater than it is in children. In Dr Hughes's hundred cases there were seventeen women above the age of sixteen to five men. In the editor's first series there were twenty-one women above fifteen to four men, and in the second series twenty-seven to four men.

Recurrence.—Another important point in the ætiology of chorea is its liability to recur again and again in patients who have once suffered from it. A large proportion of the patients admitted into any hospital have had the disease once or oftener before. We have recorded the case of one young man who had an attack every autumn from the age of fourteen to that of twenty-two, and of a girl who was seized each May, from her eighth year to her fourteenth. The duration of relapses is generally less than that of the first illness, but Trousseau mentions some exceptions to this rule.

Mental shocks.—The immediate exciting cause of chorea is often a severe nervous impression such as a fright. Many striking instances of this have been placed on record. Thus Romberg relates the case of a girl, aged ten, who was one morning violently alarmed by a dog which jumped at her and barked, and who was seized with chorea the same evening. Again, von Ziemssen speaks of a boy, aged ten, who was terrified by a shot falling unexpectedly close to him on a field, and in whom the disease reached a great pitch of severity within a few hours. Trousseau gave the case of a girl, aged sixteen, who had been caught hold of by a man as she was going downstairs one evening without a light, and who was so frightened that she had a nervous fit, and from that moment became affected with St Vitus's dance. Bright described the case of a boy who had already recovered from an attack of chorea, and who was sleeping with his father when the latter was seized with a fit of apoplexy; the boy was so alarmed that his disease returned. And, to give an instance that has recently occurred at Guy's Hospital, a child admitted under Dr Pavy with chorea, was frightened by seeing her brother in flames, ran out of the house screaming, jumped over three walls, took refuge in a neighbour's house, and was brought home and put to bed. Her mother at the time noticed a peculiar twitching movement about her face and irregular motions of her limbs; next morning she was unable to stand, and the disease quickly developed itself. There is no doubt whatever that much exaggeration has prevailed in regard to the association of chorea with mental impressions, and that parents and relations often jump too hastily at conclusions in regard to it, particularly when the fright occurred some time before the disease begins to appear. But the cases just quoted seem to be beyond cavil or dispute, and I think that they afford good grounds for supposing that the same cause has really been in operation in other instances where a longer interval has elapsed. Again, Dr Hughes ('Guy's Hosp. Rep.,' 1856) related a fatal

case in which the symptoms were slight when the patient was admitted, but became suddenly aggravated in consequence of a fright caused by another woman in the same ward.

In other instances chorea is apparently the result of *imitation*. It has long been taught at Guy's Hospital, both by Dr Addison and by the physicians who have succeeded him, that not more than one or two patients affected with this disease should be placed in the same ward with other children, lest they should copy it. I cannot, indeed, learn that any of the sisters or nurses remember an instance in which this has occurred, and I believe that at the Children's Hospital such precautions are believed to be altogether unnecessary. But some examples of the occurrence of chorea in an almost epidemic form have been placed on record, of which the most striking appears to be that related by Bricheteau. At the Necker Hospital one afternoon a young girl was admitted, suffering severely from the disease; in the evening a patient, already in the ward, who had previously had chorea and was suffering from hysteria, began to exhibit movements and in twelve hours had to be tied down; next day two other cases occurred, and within the four following days five more, making eight in all; the disease might probably have spread still further had not the patients been from that time isolated.

Pregnancy.—Another condition which plays an important part in the causation of chorea in girls after the age of puberty and in young women is *pregnancy*. Dr Barnes has collected ('Obstet. Trans.,' vol. x) a series of fifty-eight cases of this kind, and Wurzel has added eight others, making a total of sixty-six. The period of gestation at which the spasmodic movements are most apt to begin is from the first to the third month, but sometimes it is much later; and two instances have been recorded in which they followed parturition. First pregnancies are much more often accompanied by chorea than subsequent ones. This of itself suggests that one element in the production of the disease may be the emotional excitement which necessarily arises in a woman who finds herself for the first time pregnant, and the suspicion is confirmed by Dr Wilks's observation that a large proportion of those who are attacked are unmarried girls, to whom their condition is one of shame and distress. Thus a connecting link is established between the chorea of pregnancy and that which occurs under other circumstances, and a still stronger one is the fact that in a considerable number of cases the patient has already had the disease on one or more occasions at an earlier period of life. Among the sixty-six cases already referred to there were fourteen in which previous attacks had occurred.*

So far the causal relations of chorea differ little, if at all, from those of the neuroses in general; and it may be added that, according to Trousseau, chlorosis and anæmia generally are among the conditions which dispose to its development.

Rheumatism.—We now pass to an entirely different set of agencies which play a most important part in the ætiology of this, but of no other nervous disease; I refer to rheumatic fever and its unknown causes. There is, indeed, still much difference of opinion with regard to the nature of the connection in question. We have it on the authority of Dr Bright that as far back as 1802, rheumatism was stated to be one of the causes of chorea, in the Syllabus of Lectures on Medicine delivered at Guy's Hospital. That great physician was himself convinced of the relation, and thought that an

* See also Dr Lever's paper ('Guy's Hosp. Rep.,' Second Series, vols. v, p. 3, and vi, p. 233).

intervening link between the two disorders was to be found in the presence of inflammation of the pericardium, from which he imagined that irritation was transmitted to the nervous centres, just as in other cases it might be communicated from the intestines or the uterus. And in support of such a view there certainly is a case recorded by Dr Wilks, in which pericarditis caused by renal disease was accompanied by choreic movements. That case, however, is at present unique; and inflammation of the pericardium is after all present in very few cases of chorea. On the other hand, it is certain that the association between the neurosis and rheumatism is far wider and closer than would appear from Bright's theory. Beside acute rheumatism, chorea is the chief and almost the only cause of a simple inflammation of the valves of the heart, and this lesion probably occurs in almost every case of chorea. At least, out of eighteen fatal cases which have occurred in Guy's Hospital between 1848 and 1876, and in which autopsies were made, in only one was endocarditis altogether absent; and that the frequency of its occurrence was not dependent merely upon the severity of the chorea in those particular instances is evident from the fact that in five of them the patient's death was accidental and due to some complication or intercurrent disease. The circumstance that this neurosis and acute rheumatism are almost alone in giving rise to such an affection of the cardiac valves would of itself suggest a close relation between the two; and this conclusion is confirmed by the frequency with which they occur in the same individual; a child who has had St Vitus's dance falls ill with rheumatic fever a few years afterwards, or *vice versâ*; or, again, slight choreic movements appear in the course of a rheumatic attack, or rheumatic synovitis in the course of chorea.

Beside Bright and his pupils, the younger Babington and Hughes, Dr Copland and Sir George Burrows were early advocates of the belief that there is a real connection between chorea and acute rheumatism, that the latter predisposes to the former. Sée introduced the same theory into France, and it is generally acknowledged in Germany and America. The only plausible arguments against it are well stated by Dr Sturges ('Chorea and Whooping-cough,' p. 16). The fact, if it be one, has in its favour that it is quite unexplained and supports no theory of either disease.

Statistics are as follows:—Among the patients of Guy's Hospital, (*a*) in the late Dr Hughes's first series of 104 cases ('Guy's Hosp. Reports,' 1846), special inquiries were made in fifty-eight cases, there was a cardiac murmur in nine, and a history of rheumatism without bruit in eight more; (*b*) in his second series of 209 cases, compiled by Mr Burton Brown (ibid., 1855*), special inquiries were made in 104 cases, and in eighty-nine of these there was either bruit or rheumatic history; (*c*) in the series of 150 compiled by Mr Manser, and published by the Editor in 1874 (ibid., Third Series, vol. xix), forty-two had suffered from rheumatism, and in three of these rheumatic fever supervened while they were under treatment for chorea; there was a bruit in fifty-nine, none in eighty; (*d*) in the fourth series, of 163 cases compiled by Mr Halstead (unpublished), fifty-three had had rheumatic fever or distinct pains in the limbs, believed to be rheumatic, and in thirty-five of them the rheumatism preceded the chorea by less than six months; there was a bruit in sixty-one, none in 111.

Of Prof. Sée's 128 cases, sixty-one had probably had rheumatism.

* The first volume of the Third Series was published in 1855, not 1856, as stated by von Ziemssen, Bd. xii B, S. 443.

Of Dr Sturges' 100 cases, only twenty had had rheumatism.

In 104 consecutive cases of chorea at the Westminster Hospital, Dr Donkin found twenty-seven cases of previous rheumatism.

Of Dr Goodhart's 130 cases (collected from several sources), eighty-nine were on positive evidence believed to have had rheumatism.

Of Dr Angel Money's 214 cases (also collected), thirty-three had had rheumatic fever, twenty-three had had rheumatism, and nine were doubtful.

In 172 cases of chorea at the London Hospital Dr Stephen Mackenzie found a history of distinct rheumatism in forty-seven ('Trans. Int. Congr.,' 1881, vol. iv, p. 97).*

Two children in the same family often have chorea, but we rarely find that the parents of a choreic child have themselves suffered from the malady when young.

Embolic theory.—Attempts have been made to explain the connection between chorea, rheumatism, and cardiac disease upon another theory, which, although it commands the support of some distinguished observers, does not appear to be well founded. Its author was the late Dr Kirkes. Starting from the frequent association of endocarditis with chorea, he suggested that the cause of the spasmodic movements might be the introduction of inflammatory products and fibrinous particles from the diseased valves into the blood, and the consequent disturbance of the parts supplied. Dr Hughlings Jackson has expressed a similar view in a more definite form, maintaining that the cause of chorea is embolism of minute arteries in one or both of the corpora striata.

A few observations have since been made by different pathologists which have been thought to corroborate Dr Jackson's theory. In a very severe case, attended with maniacal symptoms, Dr Tuckwell ('Med.-Chir. Rev.,' 1867) found at the under and outer aspect of the right hemisphere a large red patch of softening, affecting to some extent the white as well as the grey matter; and a branch of artery which ran straight into it contained a small, white, tough fibrinous concretion, tightly wedged into an angle of bifurcation, and connected on all sides with long, black coagula, extending into the trunk of the vessel and its branches. On the outer aspect of the same hemisphere was a similar patch, but smaller, and limited to the superficial cineritious substance of the convolutions; in this nothing like an embolus was discovered, but the parts had been cut through in various directions before the dissection of the vessels was commenced. In another case of very acute chorea, which terminated fatally by hæmorrhage into the brain, Dr E. L. Fox ('Med. Times and Gaz.,' 1870) believed that he detected microscopic emboli in the vessels of the corpus striatum.

Dr Angel Money has endeavoured to produce chorea in monkeys and cats by injection of starch granules or insoluble salts into the cerebral arteries, and the results will be found in the 'Medico-Chirurgical Transactions' for 1885; see also 'British Medical Journal,' July 17th, 1886.

But is it possible to determine whether clots in minute cerebral arteries are of *ante-mortem* formation, still more whether they entered the vessels from below, instead of being formed *in situ?* Moreover, the minute, firmly adherent vegetations which are found on the valves in chorea, do not seem at all likely to be detached and carried away by the blood-stream. If they were so liable to be washed off, what could prevent some of them from

* The Collective Investigation gave 116 instances of precedent rheumatism (excluding mere "rheumatic" pains) in 439 cases of chorea.

being carried into the spleen and kidneys, and producing infarctions there; but such appearances have never been discovered in any of our fatal cases of chorea. Again, as Dr Bristowe remarks, it is difficult to understand the limitation of chorea to the limbs of one side on the embolic theory, since a shower of minute emboli must be supposed to enter the arterioles of one corpus striatum only. We are familiar, however, with endocarditis in which vegetations are carried from the diseased valves, and cause embolism in the brain and in other parts; and in these cases chorea never develops itself.

But the strongest argument of all—and one that seems to me to settle the question—is the relation of chorea to *mental shocks*. Some writers have supposed that the cases in which the disease follows a fright are distinct from those in which it is associated with endocarditis; but this is certainly not the fact. On the contrary, as already stated, vegetations are found on the cardiac valves in all fatal cases, almost without exception; for instance, a child died under Dr Wilks's care who was attacked by chorea after being terrified by the gunpowder explosion at Erith, and *post mortem* the mitral valve was found inflamed. Now, it is obvious that the disease cannot possibly have two different exciting causes in a single case. It cannot be at one and the same time the result of a mental shock and of embolism of several minute cerebral arteries. In such cases we cannot but admit that the endocarditis is an effect of the chorea.

Still it is not to be denied that any view of chorea presents difficulties. It is strange that a disease so closely related to acute rheumatism should be excited by a nervous shock; it is still more strange that, when so produced, it should possess a power like rheumatism itself of setting up inflammation of the cardiac valves. No more striking instance could be found of the complicated working of different predisposing and exciting causes, which we so often observe in the ætiology of the neuroses.

Treatment.—This is a very difficult question; for chorea offers peculiar obstacles to the satisfactory investigation of the action of remedies. Many of the severer cases tend naturally to a more than usually rapid recovery; and in other instances (as Dr Wilks has proved) the being admitted into the ward of a hospital and kept in bed, is of itself sufficient to bring the complaint quickly to a termination. Under either alternative, the medicine which may have been prescribed is apt to get undue credit. Lastly, in the great majority of cases, *chorea subsides of itself after eight to twelve weeks*, under whatever treatment. Now, the method usually adopted is to give one drug for three or four weeks; if that fails, to change it for another, which they continue for about an equal period of time; and, if there is still no result, to begin the administration of a third. The result necessarily is that even if all these medicines are really equally inert they gain very different degrees of credit. The uselessness of the one which is first given is sure to be apparent; but the second runs a chance of apparent success; while the last one is almost certain to acquire the reputation of having cured a case in which its competitors had altogether failed. Yet that very drug, if placed first on the list for a succeeding case, would perhaps show itself as impotent as the others. Probably, a large part of the prevailing uncertainty as to the influence of remedies upon chorea is explicable in this way.

It would seem that there are only two methods of avoiding these difficulties. One would be to treat a considerable number of cases with some one medicine throughout the whole course of the disease, and then to

compare them with the series of cases related by Drs Gray and Tuckwell, which were allowed to terminate without interference. These observers administered arsenic to fifteen choreic patients; and they state that the average duration of these cases was almost absolutely the same as if no medicine had been given. The very volume of the 'Lancet' (1871) in which their investigations are recorded contains reports of twelve other cases by Mr Butlin, treated by Drs West and Dickinson with sulphate of zinc. But it is impossible to compare their results with those of the Oxford physicians. Two of the cases had lasted for so great length of time before they came under observation (thirteen months and four years respectively) that their introduction would swamp the whole series. Yet after all these two only present in an extreme form difficulties which belong in a less degree to all the other cases, every one of which had lasted for several days, and many of them for some weeks, before the administration of the medicine was commenced. It is not easy to see how one could avoid this source of fallacy, except by confining one's observations to the children in one particular school, where a uniform treatment could be employed from the very beginning of the disease. So that it is almost impossible to obtain statistical proof of the value of medicines in the treatment of chorea.

But there is very strong evidence, of another kind, in favour of at least one remedy. Very protracted cases, which had resisted all other methods of treatment, have sometimes been found to yield in a very short space of time to *arsenic*. Some striking examples of this are recorded by Romberg. One is that of a girl, aged eleven, who had for eight years suffered from intense chorea, affecting especially the right half of her body. All drugs had been found useless until she began to take Fowler's solution; in about two months there was a marked improvement, and at the end of two months more she had entirely recovered. Another patient, a girl aged ten, had had the disease two years; arsenic was prescribed, and within three weeks the symptoms presented a marked abatement; and ten or eleven weeks afterwards she was discharged cured. A third instance is that of a girl, eight years old, who for six months had been the victim of chorea to such an extent that she could not walk, nor stand, nor speak articulately. The remedies which had been tried had failed; Fowler's solution, in doses of four drops three times a day, established a cure in eight weeks. So far as appears, the only objection that can be offered to these cases is that it is not distinctly stated whether or not the patients, while the medicine was being given, remained otherwise under the same conditions as before.

Most observers are of opinion that others of the so-called nervine tonics are also useful. Thus, the *sulphate of zinc* has for many years been largely used at Guy's Hospital; one-grain doses of it used to be given at first, which were gradually increased until the patient took a scruple or more. It seems more advisable to begin with a larger dose, for instance, with five grains. This seldom causes nausea or sickness more than once or twice. Another drug which once had a great reputation is the *carbonate of iron*.

Salicyl-compounds have been lately used, and, in some cases, with apparent success ('Brit. Med. Journ.,' 1887, vol. i, p. 436).

Static electricity, used in the old-fashioned way, was formerly much used at Guy's Hospital by Addison, Golding Bird, Gull, Hughes, Babington, and Lever ('Guy's Hosp. Reports,' series I, vols. ii, vi, and vii; series II, vol. viii), and galvanic and faradic electricity have likewise been tried, praised, and abandoned.

The milder cases of chorea, again, there is reason to believe may to some extent be checked by gymnastic exercises, by military drill, or by the use of a skipping-rope. French physicians have laid stress upon this method of treatment, and it has had undoubted successes.

On the other hand, one sometimes has to deal with cases which are so severe that the administration of nervine tonics, requiring time for their operation, is obviously inapplicable, because the patient is in imminent danger of dying within two or three days. There is the greatest difficulty in determining the value of remedies in cases of this kind. Probably death is sometimes inevitable. But even then the inhalation of *chloroform* gives great relief to the patient's sufferings; and if there be a chance of recovery it may do something towards economising his strength. It also saves him from the sores which would form over the bony prominences if the movements were uncontrolled. In cases which are a little less severe, *chloral* appears to be the best medicine. Several writers have related cases in which it seemed to be effectual; not the least striking is one of Dr Gairdner's, of a girl who took a drachm of it by mistake, and was poisoned, but who on her recovery was found to be cured. Again, the muscular depressants—conium and the Calabar bean—have sometimes been employed. At one time the author prescribed the succus conii in considerable doses for several choreic patients, and some of these recovered more quickly than was expected.

A point of great importance in very severe cases is that the patient should be kept well supplied with food; nutrient enemata ought to be administered frequently; and probably it is right to give full doses of alcohol.

TETANUS.*—We now come to a dangerous and happily rare spasmodic disease, the history of which dates back to classical times. So completely has the word become identified with *tonic spasm* that physiologists speak of continuous contraction of a muscle as "tetanus," whatever may be its cause. Physicians, on the other hand, now recognise as entirely distinct several neuroses which are all attended with tonic spasms:—Hysterical Contractions of the Limbs, certain reflex disorders (especially a form of Trismus and tonic Wryneck), and the complaint above described under the allied name of Tetany, together with Trismus neonatorum. Evidently, therefore, something more than a particular kind of spasm is required to characterise tetanus. Further definition is found in the seat of the spasms, in their more or less constant order of development, in the traumatic origin of the disease, and in its nearly constant rapid course and fatal issue.

Symptoms.—In the great majority of cases tetanus begins as a stiffness or rigidity of certain muscles of the face or neck. Very commonly those first affected are the masseters and the other muscles of mastication; the consequent inability to separate the teeth freely is known as *trismus*, and has given to tetanus the English name of "lockjaw." Sometimes, however, the earliest symptom is a "stiff neck," which the patient for a time regards as quite unimportant. Or, it may be a peculiar grinning expression of the face, as in a little girl who some years ago died in Guy's Hospital; her mother found fault with her for making faces; soon afterwards she was nearly choked in attempting to swallow some food. The disease is often first discovered when the patient wakes up in the morning. As a rule, it is preceded by a wound, or by an injury of some sort, which may or may not

* *Synonyms.*—Τέτανος (a stretching, strain, tension, from τείνω), used by Hippocrates. Tetanus—Rigor nervorum (Celsus)—Lockjaw—Fr. Tétanos—Germ. Starrkrampf.

have been sufficiently severe to require surgical treatment. Sometimes, before the tetanus sets in, darting pains are complained of in the injured part, which may shoot up the limb; there are four instances of this among seventy-two cases which were collected from our books for the 'Guy's Hospital Reports' in 1857 by the late Mr Poland. Sometimes, again, it is said to be ushered in by rigors, but in general there are no premonitory symptoms whatever.

Very rarely it happens that the trismus and other early symptoms, after lasting a few days, subside; the disease may then be said to abort. Far more frequently the tonic spasm increases and spreads to the muscles of the trunk and limbs. The patient's aspect is then very remarkable. The face may be described as having an unnaturally aged appearance, the forehead being wrinkled and the features contracted and drawn. The angles of the mouth are wide apart, and the lips are stretched over the closed teeth, so as to produce a fixed smile, which is known as the *risus sardonicus*,* and is at once seen not to be indicative of any pleasurable feeling. The naso-labial furrows are exaggerated. The eyelids are half closed; their muscles are seldom affected by the cramp. The jaws are sometimes clenched so firmly that not even a paper-knife can be wedged in between the teeth. The trunk is rigid, and it is almost always curved, so that the back forms a deep hollow. Thus the occiput is buried in the pillow, and the throat is stretched upwards. If the patient were to attempt to lie straight his body would be found arched and supported upon the head and the heels. This condition is called *opisthotonos*. At the same time the chest is of course thrown forwards, and it is more or less fixed in a state of expiration, while the abdomen is flat or sunken. The tension of the affected muscles is obvious to the touch and sight; this is particularly the case with the recti abdominis, which are often described as feeling "as hard as boards," and which in persons who are not too fat stand out in knotty masses through the integuments. Sometimes their fibres give way, and blood is extravasated so as to form a palpable swelling. The limbs are commonly extended, and there may be a marked stiffness of the shoulders and hips, and less often of the elbows and knees. Beyond a little undue resistance to passive flexion of the wrists, there is seldom any impairment of the movements of the hands or fingers. In one case it was recorded that the soles of the feet were arched and the extensor tendons rigid.

From an early period of the disease there is pain resembling that which is experienced in a limb affected with cramp, and one of the first symptoms is often an acute pain at the lower part of the sternum piercing through to the back; this is supposed to be due to spasm of the diaphragm. There may also be a distressing sense of oppression from embarrassment of the breathing, and the same cause may render the voice feeble, or reduce it to a whisper. The patient is frequently unable to micturate in consequence of the rigidity of his abdominal muscles.

According to Rose, of Zürich, many cases of tetanus end fatally without being attended by any symptoms beyond those which have been already described, but in a much larger number the spasms undergo aggravation from time to time at longer or shorter intervals; during these paroxysms, which last from a few seconds to three or four minutes, the aspect of the

* *I. e.*, as usually interpreted, the Sardinian laugh: from the classical tradition of a herb growing in Sardinia (Σαρδώ) which produced involuntary facial spasms. Cicero has *ridere γέλωτα σαρδάνιον*, and this form makes the above derivation doubtful.

patient becomes frightful. The contraction of the features and the opisthotonos are greatly augmented. The tongue is often caught between the teeth and severely bitten. The face and even the hands become livid from interference with the respiration. These paroxysms sometimes seem to arise spontaneously; sometimes they are obviously reflex, being brought on by a touch from another person, a draught of cold air, a sudden noise, or some voluntary effort, such as the attempt to turn round, to speak, or to swallow. Bauer remarks that they cannot always be excited by the application of a stimulus for the purpose, particularly if the patient expects it. They are generally attended with a great increase in the pain, which may amount to the most extreme anguish. Sir Gilbert Blane, however, met with a case in which, although it terminated fatally, there was merely a sort of tingling sensation of rather a pleasurable kind.

Sleep is generally absent from an early period of tetanus, but Sir Thomas Watson relates how when a patient fell asleep the tonic spasm ceased for the time; even the abdominal muscles became perfectly soft and yielding but instantly resumed their contracted state as soon as he awoke. The mind is perfectly clear and unclouded; only when the end is near at hand is there sometimes a little delirium.

The pulse is at first natural, but towards the last it becomes very rapid, perhaps 160 or 200 in the minute.

It was long a disputed question whether tetanus is attended with fever. Observations made during the last few years have shown that the temperature may be normal throughout the whole course of even the most acute and severe cases. On the other hand, in those which are comparatively slow in their progress the thermometer occasionally indicates 102° or 103° without any discoverable cause. Before death hyperpyrexia sometimes rapidly develops itself, temperatures of 110° or 112° being registered. The suggestion has been made that the heat evolved is due to the muscular contractions themselves, especially since in tonic spasms no "external work" is performed. But in reality only a very small part of the rise could be accounted for in this way, and its great height must be ascribed to disturbance of a central regulating machinery, exactly as when the same thing occurs in a case of fractured spine, or of cerebral hæmorrhage, or of the *status epilepticus*. A further point is that tetanus is among the few diseases in which the temperature has been observed to rise one or two degrees after death.*

In severe cases the skin is generally bathed in sweat; and an eruption of sudamina is not infrequently present. Dr Wilks has recorded in the 'Guy's Hospital Reports' for 1872 an instance in which the perspiration from the forehead gave to white linen clothes a reddish stain, which looked as if it were due to the presence of blood, but this was found by Dr Stevenson to be absent.

Varieties. — The symptoms of tetanus sometimes deviate from the ordinary type. From the most ancient times, several distinct forms of it have been described. Writers have said that the body may be arched forwards instead of backwards, the head and the knees meeting in front of the chest; and have called this, after Aretæus, *emprosthotonos*. In another

* This heat has then been supposed to be derived from the solidification of the muscles in *rigor mortis*. This explanation, however, does not seem to be quite satisfactory, for, although the *post-mortem* rigidity is said to begin early, and also to last longer than usual, there appears to be an interval of relaxation from the vital spasm before it sets in.

variety, for which the name *pleurothotonus* has been invented, the curve is on one side. But it has long been known that in comparison with opisthotonos they are exceedingly infrequent; and it is absurd to make of them separate kinds of tetanus. Indeed, Rose has lately maintained that they are really never seen in this disease, although he admits that they may occur in hysteria. The French military surgeon, Larrey, has been the great authority for emprosthotonos; but Rose shows by detailed criticism that none of his cases belonged to any but a very mild form of tetanus, so that it certainly seems possible for mistakes to have been made as to the significance of a position of the body which is, after all, generally adopted by patients suffering under irritation of the nervous centres; and the more so since Larrey entertained the notion, which is certainly without foundation, that the distribution of the spasms varied according as the wound which caused the tetanus was in front or behind. In 1870, however, a woman, aged forty, died in Guy's Hospital of this disease, in whom it is said that "the anterior muscles were mainly affected, so that there was a condition of emprosthotonos."

There is one aberrant variety of tetanus, in which the earliest symptom is a spasmodic affection of the muscles of the part originally injured, and in which the paroxysms, when they set in, affect those muscles far more than any others. Such a case was recorded by the late Mr Key in the third volume of the 'Guy's Hospital Reports,' and we have since had a well-marked instance of the same kind.

An occasional complication of tetanus, to which Rose has drawn attention, is facial paralysis. In 1871 a woman died under Mr Poland in Guy's Hospital, in whom this symptom was present on the left side, and whose ocular muscles were also affected in a strange way, the left eye being immoveable, turned upwards and outwards, while the right one was as rigidly set straight forwards. Neither meningitis nor any lesion of the brain itself was discovered at the autopsy. The original accident was a fracture of the orbital plate of the frontal bone, caused by the point of an umbrella. This corresponds with a statement of Rose's, that the starting-point of the tetanus in such cases is always within the distribution of the facial nerve; he supposes that the trunk of the nerve becomes swollen, and is compressed within the bony canal through which it has to pass. If this view is correct, the symptom in question affords a proof of the occurrence of an "ascending neuritis" in tetanus, and is thus of great theoretical interest. Indeed, in some few cases, nerve-trunks are said to have been found reddened and swollen in parts of their course, from the seat of injury up to the end; but as such changes are often not to be discovered, it is doubtful whether much importance is to be attached to them.

Ætiology.—In reference to the *causes* of the disease I do not propose to enter upon various questions, which are fully discussed in surgical works, as to how far the liability to it is influenced by the seat or the extent of an injury, by the healthy or unhealthy state of the lacerated structures, or by the introduction of foreign bodies. But it is important for the physician to be alive to the fact that tetanus often follows very slight cuts and trifling abrasions, which are apt to be altogether forgotten by the patient and overlooked. Rose says that nearly half of his cases were sent into the hospital as cases of rheumatism or of some internal disease. Instances have been recorded in which it has apparently been set up by the extraction of a tooth, by venæsection, by the application of a blister or a cupping-glass or a

seton, by the sting of a bee, and by a cut from a whip. It has now and then been observed after simple fractures of the limbs, or after blows or falls upon the back of the neck, without any breach of surface. In 1860, a girl, aged five, died in Guy's Hospital, who on the day before she was attacked by the disease had had a fall in which she was said to have slightly strained her back and grazed her elbow. Three other patients showed, one a slight scar over the knee, another a small sore on the elbow, and the third a little scab with pus beneath it at the elbow, in addition to a cicatrix half an inch long on the forearm, due to a cut received about six weeks previously. In 1873 a boy was admitted under the author's care in whom stiffness of the neck and jaws had come on four or five days after the healing up of a small "gathering" on one big toe, due to irritation from a small nail in his boot. The patient recovered. Again, tetanus has sometimes occurred soon after parturition or abortion. In one case which was observed at Guy's in 1870 no cause for it could be discovered except a prolapsed and excoriated condition of the cervix uteri. Whether it is ever due to an internal lesion or to the presence of worms in the intestines appears to be exceedingly doubtful.

It is remarkable that in only two of our cases, in which the disease has appeared to be referable to a trifling injury, has there been reason to suppose that cold played any part in its ætiology. One was in a patient of the author's who, besides having a festering sore on the elbow, had got wet through in a shower of rain three days before the tetanus set in; this perhaps affected him the more because his work was behind the ovens at a biscuit manufactory. The other was in a man under Mr Bryant, who had had a slight scratch on the little finger, which soon healed up; he also was exposed to wet two days before being attacked with stiffness between his shoulders. It seems to be an established fact that in some cases in which the affection is clearly traceable to a severe wound it is, nevertheless, also due, in part, to changes of temperature. Army surgeons have often noted that after a battle the wounded are especially apt to be attacked by it when they lie in tents on a damp surface, or when cold nights contrast with hot days. And it would seem that the liability to exposure to draughts in tropical climates is the best reason that can be assigned for the frequency with which, as is well known, the disease follows all sorts of injuries among the coloured populations of the East and West Indies.

So, also, there appears to be no doubt that cold sometimes gives rise to tetanus directly in persons who have received no injury whatever. On July 17th, 1863, a man sat in a draught, and afterwards felt a stiffness in his limbs; this continued during the 18th and the 19th; on the 20th more marked tetanic symptoms set in, aud he came to Guy's Hospital and was admitted; at 7 a.m., on the 21st, he had a severe paroxysm affecting his chest, in which he died. Another patient, in 1869, had got his feet wet the day before the disease began. Sir Thomas Watson cites a case of Dr Gregory's, that of a man who, "having fallen asleep in moist grass, awoke with a stiff neck, which afterwards went on into regular tetanus."

But it now and then happens that not even this cause can with any plausibility be assigned to it, and that its occurrence remains altogether inexplicable in the present state of our knowledge. Three such cases have ended fatally in our wards between 1863 and 1875.

Whenever the disease arises independently of an injury—whether or no it is traceable to cold or wet—it is called "idiopathic," to distinguish it from ordinary "traumatic" tetanus.

On the whole, tetanus is more common in men than in women; in young persons than in those who have passed the middle period of life; in the robust and the healthy than in those who are weakly or diseased.

Pathology.—Pathologists were generally agreed as to the absence of obvious changes in the nervous centres in cases of tetanus. But in 1865 Mr Lockhart Clarke recorded in the 'Med.-Chir. Transactions' (p. 255, pl. iv) some cases in which he believed that he had discovered areas of semi-fluid disintegration, or white softening, in the grey matter, but also in the white columns of the cord. Similar observations were afterwards made by Dr Dickinson, who, considered that the morbid material which he found in the same structures, and which closely surrounded the blood-vessels, was an exudation. Still more recently Dr Coats, of Glasgow, has demonstrated like appearances in the bulb and the pons. All these pathologists also lay stress on the congested state of the blood-vessels, but that had long ago been noticed, and had been shown to be of no real significance, being attributable either to the mode of death or to cadaveric hypostasis. The question whether any greater importance is to be attached to the supposed lesions above referred to is one which has still to be further discussed, and in relation to several other diseases. But whatever conclusion may be arrived at, there are obvious difficulties in bringing such morbid changes to bear upon the pathology of tetanus. It is doubtful whether we can at present form any clearer conception of the action of tetanus than of any other member of the group of spasmodic neuroses to which it seems to belong.

In his article on Tetanus in 'Holmes's System of Surgery,' the late Mr Poland argued against a primary central lesion (or an ascending peripheral neuritis as it would now be called,—*a neuritis migrans*, as Lockhart Clarke supposed) and in favour of the view that tetanus is really a "specific" disease, a form of blood-poisoning. This hypothesis has lately been supported by Professor Coats.

Diagnosis.—This is seldom difficult. It is only at the very commencement of *trismus* from dental irritation in infants that one could mistake it for true "lockjaw." Rose says that even at the earliest period of tetanus he has always been able to discover a certain degree of stiffness of the back of the neck, the patient being unable to bring the chin freely down to touch the chest. Another practical suggestion of his is that by introducing one's finger into the patient's buccal cavity on each side one can feel the hard edge of a rigid masseter much more distinctly than from outside the cheek. Inability to open the jaws, from any chronic affection of the temporo-maxillary joints, is in this way easily distinguished from all forms of trismus.

Tetanus is sometimes simulated by *hysteria*. Sir Thomas Watson mentions an instance of this kind in a girl, who "would all at once be drawn into a position such that the top of her head and her feet alone supported her, while her body was bent backwards like a bow; then, after a time, with equal suddenness, the opposite position was assumed, her forehead and her knees being brought together." His statement, I believe, suggests the criterion which is applicable to all cases of this kind; namely, the irregular and inconsistent nature of the symptoms. Another affection which has perhaps to be considered is *spinal meningitis*. A case which is given in 'Reynolds' System' as a typical example of the latter disease, would show that they may resemble one another very closely, if we could accept the interpretation of it without question; but I am disposed to agree with Dr Moxon that, after all, it may have really been an instance of tetanus.

We shall find that hydrophobia is sometimes complicated with spasms like those of tetanus.

The most important point of all is that one should not set down to this disease the effects of the poisonous *strychnia* and brucia, or of the substances from which those alkaloids are derived. A boy, aged twelve, was brought into Guy's under my care at 9 a.m. one morning, suffering from opisthotonos, and from spasms of the respiratory muscles, so severe, that he almost ceased to breathe and had a sense of immediately impending death. He was employed in a druggist's shop, and he confessed that between 7.40 a.m. and 8.30 he picked up some black stuff (afterwards ascertained to be extract of nux vomica) and put it into his mouth for liquorice, until he found it bitter, when he spat out as much of it as he could. Ice applied to his spine gave him relief. From some urine passed at 1.20 p.m. Dr Stevenson succeeded in obtaining the colour reactions of strychnia and brucia. By that time his symptoms had passed off, and he was discharged from the hospital a few days later. The characteristic features of such cases are the suddenness with which opisthotonos and the most violent general spasms set in, the absence of persistent rigidity of the affected muscles during the intervals (so that the mouth can be freely opened), the fact that the hands are involved, and the rapid death or recovery which ensues. Even if the poison should be administered in small doses repeated at frequent intervals, there is no reason to suppose that the progressive development of ordinary tetanus would be simulated.

Fatal event.—As a rule, tetanus ends fatally between the third and the seventh days. The occurrence of death within twenty-four hours from the commencement of the disease is very infrequent. Among Mr Poland's seventy-two cases there were only two instances of it; one patient, a young man who was attacked six days after admission with a compound fracture of the leg, died in nineteen hours; the other, a girl who had been burnt, is said to have lived only four or five hours. The most quickly fatal case on record is one of a negro servant who lacerated his thumb in breaking a china dish, and who was almost instantly seized with convulsions and died in a quarter of an hour; but Sir Thomas Watson, although he cites it, expresses a doubt as to whether this was a genuine example of tetanus. In most instances death occurs during a paroxysm which surpasses all previous ones in severity. Its cause is generally supposed to be spasm of the diaphragm, or other respiratory muscles, or perhaps of those which close the glottis. Mr Poland mentions that in one of his cases the heart's action continued for a short time after the breathing had ceased. In 1875 I had a patient under my care who on several occasions turned perfectly livid, so as to excite the strongest apprehensions that he was at the point of death, while a clenched state of his hands was the only outward indication of an increase of spasm. At last, after four days, one of these seizures did in fact prove fatal. Bauer, however, expresses the opinion that the danger from failure of the respiration in tetanus has been generally over-estimated; carbonic-acid poisoning, he says, relaxes the muscles before the mischief is done, and there is no doubt that death sometimes begins from the heart. Rose relates an instance (which, however, was attended with hyperpyrexia) in which he had his finger on the pulse when it suddenly stopped for ever. Some observers have supposed that in cases of this kind the ventricles are seized with spasm. Stress has been laid on the fact that on *post-mortem* examination the heart is often found closely contracted, but it has been

forgotten that this is in all probability merely the result of rigor mortis. Rose speaks strongly in favour of the view that the real cause of death is cadiac paralysis; he thinks that the muscular contractions oppose a resistance to the circulation through the arteries which the central organ is unable to overcome.

Lastly, there are cases in which life is prolonged for three or four weeks, but which yet ultimately prove fatal. The cause of death is then generally exhaustion. Sometimes the spasms have altogether subsided, so that the patient is thought to be in a fair way towards recovery, although he is excessively weak and emaciated and prostrate, with sunken features and a scarcely perceptible pulse. Among the cases collected by Mr Poland there are two which proved fatal during a paroxysm as late as the twenty-first or the twenty-second day, and two others in which death was referred to exhaustion on the sixteenth and the thirty-second days respectively.

Convalescence.—When recovery from tetanus takes place, a certain degree of stiffness of the muscles often remains for a considerable time, and very slowly passes off. Mr Poland mentions a case in which there was still some stiffness of the jaws after a year had elapsed. In 1871 Mr Golding-Bird had a boy under his care in Guy's Hospital in whom the spasms lasted for fifty-one days, and even after they had subsided his limbs still remained rigidly flexed, he became exceedingly emaciated, and bedsores developed; so that, although he was floated on a bath on water, he ultimately died on the one hundred and seventh day.

Course.—Although almost all writers recognise an acute and a chronic form of tetanus, they generally admit that no line of demarcation can be drawn. It is true that when successive cases are arranged in groups according to the length of time which happened to elapse between the injury and the onset of trismus or of stiffness of neck, a rapidly fatal termination is found to occur more frequently in those in which the trismus is early than in those in which it is late. A similar difference in intensity, although not so marked, seems to be observable in cases in which the primary lesion is extensive and severe, as compared with those in which it is slight. It is therefore not surprising that instances of "idiopathic" tetanus should, as a rule, be less urgent in their symptoms than those which are of "traumatic" origin, and since the subsidence of the disease, in those cases which recover, is never otherwise than gradual, one can understand how it has come to pass that the idea of chronic tetanus has become associated not only with a hopeful prognosis, but also with the absence of a severe injury or of any injury at all; or, again, with the occurrence of a long interval since the date of an injury, rendering the connection between them doubtful. But although this is tolerably correct, so far as groups of cases are concerned, it is very apt to mislead when applied to an individual patient. In some instances of idiopathic tetanus death ensues as rapidly as in most of those which follow quickly upon a severe compound fracture or a burn. So impressed is Rose with the vagueness of the current distinction between the acute and the chronic form of the disease, that he limits his "acute tetanus" to those cases in which there is nothing but a continuous tonic spasm. The supervention of paroxysms is described by him as belonging to a so-called "third stage" which the cases in question do not reach, although he himself points out that the most rapidly fatal cases of all are seen in those in which paroxysms are present from the very first. It is clear that the division into acute and chronic tetanus had better be given up altogether.

Prognosis.—The only ground on which a forecast of the disease can with any safety be based is that afforded by the rate at which the symptoms of

the disease are developing in the particular case under observation. Unfortunately it must be added that rapid progress is much more surely indicative of a fatal ending, than is a slow course of a good prospect of recovery, or even of certainty that death is very near at hand. Spasm of the respiratory muscles sometimes destroys life suddenly and almost without warning. The average mortality of the disease among the seventy-two cases collected by Mr Poland in 1857 was 86 per cent.

Some years ago Dr Wilks and the author saw with Dr Anderson, of New Cross, a gentleman whose symptoms seemed to deviate from the ordinary features of tetanus, so that we none of us felt able to speak positively as to the probable issue of the case, nor even as to its diagnosis. It seems to me now that it might fairly have been regarded as one of those instances in which the paroxysms constitute the most marked element of the disease, but in which, although they recur but seldom, they destroy life early. The patient's main complaint was of a spasmodic pain in the right hypochondrium, which he attributed to wind. When it came on, he used to get up and walk about, declaring that he could not lie down; he would stand leaning against the bedpost, and would call to his wife to rub his back. About a fortnight previously he had run a garden fork through his great toe; four days before we saw him he began to complain of stiffness of his jaws, and from that time he was unable to open his mouth fully. These facts of course led to a strong suspicion that the abdominal pain was due to tetanic spasm. He died in three days from our first visit, and about twenty-four hours before he was sitting up in bed and making his will.

Treatment.—The difficulty of prognosis in this disease necessarily brings with it a corresponding difficulty in estimating the results of treatment. There are, indeed, certain measures the propriety of which is obvious. The patient should be placed in a quiet, dark room, and should be withdrawn from all noise and bustle, and from the visits of too many sympathising friends. He should speak as little as possible. The food which is given to him should be nutritious but in a fluid form. He should probably have wine or brandy in full quantities. If he is unable to swallow, enemata of beef-tea and brandy may be administered at regular intervals, unless even this brings on paroxysms of spasm, as is too often the case. Rose makes a suggestion, which appears to me very valuable, that anæsthesia should be induced regularly once or twice a day by chloroform, for the purpose of enabling food to be injected into the stomach through a tube. Purgatives should not be given, except at the commencement, nor unless there is clear evidence of the necessity for them; nor should they be repeated.

The question whether an injured limb should be amputated on account of the supervention of tetanus, and whether the excision of nerve-trunks is or is not advisable, must be left for surgeons to decide. But there can be no doubt that any small festering cut or sore that may be present should be poulticed and soothed; and it must be remembered that in some cases a splinter of wood or some other foreign body has been unexpectedly found beneath a cicatrix, and sometimes even embedded in a nerve-trunk.

As regards the treatment of tetanus by drugs, the first thing to be said is that at present we know of no medicine sufficiently potent to arrest the disease when its symptoms advance uniformly and quickly. Rose, indeed, mentions an instance in which trismus set in and was followed in a few hours by a violent paroxysm of opisthotonos, but in which no further development of tetanus occurred after the administration of one sixth of a

grain of acetate of morphia, and the liberation of a quantity of pent-up pus and putrid matters by the knife. But, so far as I know, this case is unique. When the disease runs a rapid course, one can often do nothing better than keep the patient continuously under chloroform, so as at least to secure euthanasia.

On the other hand, when the progress of tetanus is slow, it often seems that the death of the patient is little more than an accident, from the supervention of a paroxysm which happens to exceed a certain limit of severity. In such cases it is reasonable to suppose that narcotic medicines, or those which depress the activity of the muscles, may directly save life. As might be expected, *opium* has been largely employed. Persons suffering from tetanus are found to be highly tolerant of it. Sir Thomas Watson mentions the case of a lady who took more than four ounces of laudanum a day during twenty days, and who recovered. And he refers to another case in which an ounce of solid opium was swallowed in divided doses every day for three weeks. Of late years the hydrate of *chloral* has sometimes been used in cases of the same kind with apparent success. In 1870, for instance, Mr Birkett had under his care at Guy's Hospital a man, aged twenty, who on June 24th had received a kick on an ulcer. Next day he felt rigidity of the muscles of the face and was unable to swallow solids. He was admitted on the 28th. At first thirty grains of chloral were given every four hours, but on the 30th, thirteen doses having been taken, a grain of opium was ordered to be taken every three hours instead. However, the spasms became more severe; and on July 4th a drachm of chloral was prescribed at one dose, and half a drachm on the following evening. After this fifteen grains were given every other hour until the 11th, when the quantity was increased to twenty-five grains every other hour. Two days later it was reduced again, and the disease now gradually subsided in the course of the next three or four weeks. A full report of this case will be found in a paper in the 'Guy's Hospital Reports' for 1878 by Dr Frederick Taylor, who remarks that in some other instances chloral has been found to give rise to great drowsiness, without much affecting the spasms.

Among the depressants of muscular action *physostigma* and *curare* are those which seem most likely to be useful. The first of these medicines has been employed at Guy's Hospital in one case which terminated favourably. The patient was a man, aged twenty-one, who, having got drunk on November 30th, 1874, began to suffer from stiffness in the back on December 1st. He was admitted, under Dr Wilks, with fully-developed tetanus, on December 5th, and spasms recurred every three or four minutes. The extract of Calabar bean was given at first in small doses, but afterwards one grain of it every two hours; and on the 10th, after a very severe seizure, seven doses of a grain each were administered at intervals of fifteen minutes in succession. He began to improve about December 17th, but the stiffness of the joints did not finally disappear until after the middle of January, 1875. Against this case, however, must be set another one under the author's care in the summer of 1875, in which death occurred on the sixteenth day, in spite of two and a half grain doses of extract of physostigma, repeated at one time at intervals of only an hour. Subcutaneous injections of curare were administered to a man who was under the care of Mr Durham in 1876; but it seems doubtful whether the doses which were employed were sufficiently large to be really efficacious, although the report says that they were followed by sleep and relief of pain.

Another medicine which has now and then been used in cases that have

ended in recovery is quinine. An instance of its administration with apparent success was recorded by Dr Bright in 1836 in the first volume of the 'Guy's Hospital Reports.'

It must of course be admitted that the occasional subsidence of tetanus under different remedies is not, in itself, a proof that they are really of value. But, on the other hand, it is equally true that the occurrence of a rapidly fatal termination in a far larger number of cases, in spite of the same remedies, does not show that they are useless. We need not therefore be greatly discouraged by the collection of cases published by Dr Taylor in the 'Guy's Hospital Reports' for 1878, where there are recorded nine cases in which death occurred under chloral (six by the fourth day, and the other three on the eighth, tenth, and twelfth days), and seven cases which ended fatally under Calabar bean (six by the fourth day, the seventh on the eighth day). What is really wanted is a sufficiently extensive experience to enable one to say whether the number of deaths from this terrible disease can be diminished by the administration of any drug whatever.

HYDROPHOBIA.*—From the time of Aristotle it has been known that dogs are liable to a fatal disease which they transmit by their bite; and this disease, when occurring in man, was called "Hydrophobia," from the dread of water which is one of its chief symptoms.† In the lower animals, however, that very symptom is absent. Although the symptoms of this terrible disease are those of paroxysmal nervous convulsions, its natural place is probably in association with glanders and other specific diseases.

Incubation.—Judging from what we know of the inoculation of poisons in general, we should have expected that the introduction of the virus of rabies through a bite would have produced its effect after a definite interval, and without much delay. The contrary, however, is the case. After the healing of the wound, which takes place naturally and quickly, there occurs a "period of incubation," which is often prolonged beyond that of all other infective diseases, and which is of the most uncertain duration, both in the lower animals and in man. Different writers state its average length somewhat differently. Bollinger, in 'Ziemssen's Handbuch,' says that in 60 per cent. of all cases in the human subject it is between eighteen and sixty days, in 6 per cent. between three and eighteen days, and in 34 per cent. longer than sixty days. How protracted it may be we cannot yet positively tell. Instances have been recorded in which three years, five years, even twelve years, were supposed to have elapsed. In 1854 there occurred at Guy's Hospital, in the practice of Dr Hughes, a case in which it was said that five or seven years had passed since the bite. It is possible, however, that in these cases a subsequent infection took place without the patient's knowing it. That this is possible must be admitted; for, on the one hand, the origin of hydrophobia cannot always be traced at all, although it is most unlikely that it ever arises *de novo;* and, on the other hand, Mr Youatt succeeded in tracing certain cases to sources that might very easily have been overlooked; one, in a man, to his having tried to untie with his teeth a knot in a cord by which a rabid dog had been confined; and another, in a woman, to her having used her teeth to press down the seam in mending a tear in her dress caused by the bite of an animal suffering under the disease.

* *Synonyms.*—Rabies canina—Lyssa (λύσσα, raging madness).—Fr. La rage—Germ. Hundswuth, Wasserscheu.

† "Ὑδροφοβίαν Græci appellant: miserrimum genus morbi."—Celsus, lib. v, cap. xxvii, § 2.

The following are the results of inquiries as to the period between the bite and the appearance of symptoms from cases in Guy's Hospital. Six were published by Dr Bright (1820–30). Seven more were reported by Mr Cooper Forster in the 'Guy's Hospital Reports' for 1866 (Third Series, vol. xii, p. 1). The incubation in these thirteen cases varied between four weeks and eleven months, except one in which it was supposed to be from five or seven years. In ten cases which have occurred since the publication of that report and the present year (1867–86) collected for this edition by Dr E. W. Goodall, the periods of incubation were nineteen weeks, thirty-five days, thirty-eight days, forty days, nine weeks, nearly twelve weeks, thirteen weeks, and about twelve weeks respectively, and, more doubtfully, four weeks, while in the tenth case (a man aged thirty-six, who died in 1878) no certain evidence could be obtained, either from the patient or his friends, that he had even been bitten by a dog. No doubt the period of incubation varies more than in other specific diseases, but in the most authentic and well-observed cases it is rarely more than twelve months.

In 132 cases of hydrophobia selected by the Registrar-General (1886) on account of the circumstances being accurately known, the shortest incubation was eleven days, in a child bitten by a rabid cat. In 23 cases it was under a month, in 64 between one and two months, in 21 between two and three, in 124 it was under five months, in 127 under ten months, and in 130 under two years. In one case it was between three and four years, and in one other above four years.

Prodroma.—In some cases the earliest indication of the onset of hydrophobia is afforded by an uneasy sensation in the seat of the bite, which becomes painful, or tingles, or itches, or feels cold; sometimes the cicatrix itself may be seen to be reddened, livid, or swollen; perhaps it may open afresh, or a papular eruption may appear around it. The pain or other morbid sensation extends upwards along the nerves; from the hand, for example, into the arm and up to the shoulder, as in a coachman whose case is recorded by Sir Thomas Watson. In a gentleman who came under the care of Mr Cooper Forster in 1866, the pain was of extraordinary severity; it came on in paroxysms, obliging him to stop suddenly in the street, and to cry out; it was not referred to the course of any particular nerve. There was in that instance no redness or tenderness of the cicatrix. The arm, however, felt much colder than the other one. He had been bitten eleven months previously; in the intervening period of time he had three or four times complained of pain up the arm and twitchings in the hand.

It seems very doubtful whether there is any tendency to lymphatic inflammation beyond what might arise after any other injury of an equal degree of severity. Watson cites two instances of such an occurrence; but in one of them, at any rate, the red lines which extended up the patient's arm were probably due rather to the caustic which had been applied.

Other early symptoms are a peculiar restlessness, irritability, and depression of spirits. The patient suffers from nausea and loss of appetite; he complains of headache, is sleepless, and has a distressing sense of apprehension. It is curious that he often says nothing about having been bitten, and will even vehemently deny it. And yet he may make other remarks which show that his mind is dwelling on the subject, and that he is trying to persuade himself that he need not be afraid. The most characteristic symptom is a repugnance to fluid, which may show itself in various ways. Sometimes there is a little difficulty in swallowing, from a feeling of tight-

ness about the throat. Sir Thomas Watson's patient, the coachman, refrained on account of a similar sensation from sponging himself as usual with cold water, though he remarked that he "could not think how he could be so silly." Very often the breathing is interrupted by frequent sighs, which may even stop the man in the middle of a sentence.

Symptoms when developed.—The above prodromal or early stage generally lasts from one to three days, but sometimes it is altogether absent. In either case, the full development of the disease is ushered in by the sudden occurrence of violent convulsive paroxysms, affecting the muscles of deglutition and those of respiration, and repeated at more or less frequent intervals. They are brought on by a variety of causes. The attempt to drink almost always instantly precipitates one; the sight of fluid in a basin often has a similar effect. Sir Thomas Watson mentions a case in which the patient was thrown into a violent state of agitation by hearing a dresser, who was sitting up with him, make water. The least draught of air, the waving of a mirror before the eyes, the opening or shutting of a door, the slightest touch upon the skin, the attempt to comb the hair, may each excite an attack. Yet there is an apparent caprice about them. Sometimes the patient succeeds in gulping down a little fluid by carrying it to his mouth with the eyes shut; or he may insist on the withdrawal of the bystanders as enabling him to swallow better. Sir Thomas Watson saw one man who so dreaded anyone's breath on his face that he would not converse with the apothecary of the hospital except in such a position that the chin of each of them rested on the other's shoulder. The seizures often consist of a series of shuddering or sobbing movements, more or less like those which occur when one steps into a cold bath. Sometimes, however, they are attended with tonic spasms of the cervical muscles, the sterno-mastoidei (for example) starting forwards strongly. The following description of the more violent paroxysms was drawn up by Dr Bushell, now Physician to the British Legation at Pekin, from a case which occurred while he was dresser to Mr Cooper Forster. "At the onset of a severe spasm the patient springs up in bed, and puts his hands furiously to his throat, as if to tear something away; the head is thrown violently back, the mouth is opened, and the eyeballs are protruded; then he makes several expiratory efforts, sometimes with a shrill screaming cry; the head is thrown violently from side to side; the hands are tossed wildly about, beating his chest, and striking anything that is near." Sometimes the jaws are sharply brought together, so that one can hardly tell whether the patient is not trying to bite those about him. The production of a barking noise, like that of a dog, appears to be fabulous. Bollinger denies that trismus ever occurs, or a general tonic spasm like that which characterises tetanus. Nor is there an authentic case in which complete opisthotonos was present, although Dr John Ogle described one in which there was said to be emprosthotonos ('Med.-Chir. Rev.,' 1868).

The duration of the paroxysms is variable. According to Bollinger, they occasionally last from thirty to forty-five minutes. When they are at all protracted, the patient's face and limbs become pale and livid and covered with sweat. The countenance assumes an aspect of terror, and the pupils are widely dilated. The pulse is rapid, the respiration also quickened, irregular, and more or less forced.

The temperature varies greatly. It is usually moderately raised, sometimes normal, and occasionally rises to 103° or even 105°(as in a case reported by Mr Southam, of Manchester, 'Brit. Med. Journ.,' 1881, vol. ii, p. 814).

The urine appears to be normal as a rule; sometimes febrile, and rarely albuminous. The occasional presence of glycosuria is perhaps due to inhalation of chloroform.

In women and young children the course of hydrophobia is often comparatively mild, although it is even more quickly fatal; this was so in two of Sir Thomas Watson's cases, one in a lady aged thirty-two, the other in a girl only five years old, and also in a little girl who died in Guy's Hospital in 1875. She was so slightly affected that Mr Stocker supposed the case to be one of hysteria. In other instances, however, children affected by hydrophobia exhibit the most frantic mania.

In some very rare instances the paroxysms are said to be altogether absent, so that the patient is able to drink, though with difficulty, throughout the disease. But, as a rule, there is an almost absolute inability to swallow, even during the intervals. The patient, who is much distressed by thirst, may resolutely carry the cup, or the glass, to his lips, but as soon as a little fluid has entered his mouth, it is forcibly ejected, and all the rest is spilt over his clothes. The tongue is said to be generally clean and moist; but the fauces and the palate may be seen to be injected, and their glands are swollen. A viscid saliva which collects in the mouth is a source of great annoyance; it is hawked up with noisy effort, and spat out upon the floor in all directions, or upon the faces and clothes of the nurses.

Another symptom, which is present in certain cases, is priapism, with frequent involuntary emissions. Trousseau speaks of the hyperæsthesia of the genital organs as very painful.

The mental state, even between the convulsive attacks, is almost always one of great agitation. As the disease advances, the patient often raves at those around him, accusing them of being the cause of his illness, and complaining of their gazing upon his misery. Even if what he says is not nonsense, he generally talks hastily and excitedly. Sometimes he is wildly maniacal, so that he has to be confined in a straight jacket, or to be held down by several powerful men.

Not infrequently, death occurs by suffocation in the middle of a paroxysm, and more or less suddenly. The whole duration of the disease is seldom a week, and sometimes it terminates within from twelve to forty-eight hours from its commencement.* Cases which last six days are exceptional; Sir Thomas Watson's coachman did not die until the middle of the seventh day. In that instance the end was very gradual; the pulse grew rapid and weak, the mental powers failed, he lay moaning and tossing from side to side, frothy saliva ran from his mouth, he lost the power of moving his left arm, fluid stools were passed involuntarily, the lower extremities grew cold and the coldness extended up to the chest. Some patients have towards the last become paraplegic. In the more protracted cases the spasms may cease for some hours before death, the patient be quite calm and able to talk or drink or wash his hands without discomfort, but nevertheless the coldness of his surface, and the absence of pulse at the wrist, show that there is no real improvement.

Contagion from rabies.—Hydrophobia is doubtless caused in all cases by the transference to the patient of the specific virus of rabies; but as to the conditions under which this may occur, many interesting questions arise.

* In eight cases death occurred within twenty-four hours, in eighty-seven between the first and the seventh day, in five on the eighth, and in one case on the ninth, tenth, and twelfth days respectively (Registrar-General's 'Report,' 1886).

Thus it has not yet been finally determined whether the disease is under any conditions communicable from one human being to another. No case is known of the kind, and medical men and nurses always escape, although the poisonous secretion is often ejected upon their clothes and hands and faces. In former times the dread of catching the disease was so great that the patient was often smothered between feather beds by his relations, or was allowed to bleed to death through an opened vein. Bollinger says that on the military frontier of Austria persons labouring under hydrophobia, or suspected of it, are even now liable to be shot by their neighbours, and that those who have been bitten by rabid dogs sometimes commit suicide.

There is no doubt that the disease is infectious and arises only from a previous case, nor that the virus resides in the saliva and salivary glands. Magendie long ago produced rabies in dogs by inoculating them with the saliva of hydrophobic patients. Pasteur has now proved that the spinal cord is also the seat of the virus; and that inoculations from it, especially if introduced under the dura mater of a dog, after trephining the skull, will reproduce the disease in dogs and rabbits.

The disease in animals.—Rabies may occur in many kinds of animals besides dogs. It is common in wolves, jackals, and foxes. Cats are sometimes affected by it, but far less frequently than dogs. A scratch from a cat is believed to have conveyed it to a child, no doubt from the claws having been impregnated with poisonous saliva. Among herbivora, horses, oxen, goats, sheep, pigs, rabbits, and guinea-pigs are capable of being infected experimentally by inoculation, or if they are bitten by dogs suffering from it. Mr Youatt recorded a case in which a groom took hydrophobia through a scratch which he received from the tooth of a rabid horse.

A most important question is what indications should lead one to decide, when a person is bitten by a dog, that the animal is rabid. Some writers have supposed that a healthy animal may convey the disease if it should happen to be fierce and angry, but such an idea is opposed to all our present knowledge. Mr Youatt met with cases in which a dog exhibited no symptom of rabies when it inflicted the fatal bite, though it was soon afterwards attacked. No fewer than eighteen or nineteen instances of this kind have been collected. The simplest way of accounting for them is to suppose that the disease is infective even during its period of incubation, which in dogs is believed to be generally of from three to five weeks' duration, but occasionally to be prolonged over as many months. It is evident, therefore, that when a dog is killed soon after having bitten a human being, one can never be quite sure that hydrophobia may not supervene. The proper course is to keep the animal securely confined for some weeks.

There are two varieties of the disease in dogs, one characterised by maniacal excitement, the other by paralysis of the jaw, so that it hangs down and allows a frothy saliva to run out of the mouth. In each form the bark is altered into what is described as a "hoarse inward sound, dissimilar from its usual tone, and generally terminating with a peculiar howl." Towards the last the hind legs and the loins become paralysed, so that the dog staggers about and falls. The popular distinction between ordinary rabies and "dumb rabies" with paralysis is not without foundation.

One of the earlier symptoms is an extraordinary perversion of the appetite, the animal eating hair, straw, sticks, bits of leather, earth, stones, and other substances, which remain undigested and may be discovered in the stomach

after death, mixed with a dirty-brown pulpy fluid. Their presence often affords a valuable indirect proof that a dog which has been killed under the suspicion of rabies was really affected with that disease. It has been said that a verdict of acquittal may be given whenever the digested remains of the animal's natural food are found in its stomach and chyme in its small intestine; but of course the disease might even then have been incubating.

Rabies is always fatal in dogs: usually in a week after the symptoms have appeared, occasionally after nine or ten days.

As already remarked, a dread of water is not a symptom of rabies in any animal except man. "Mad dogs," as they are commonly called, plunge their muzzles into water and lap it up eagerly (for they are very thirsty) although they may not be able to swallow.

In rabbits the symptoms of rabies (transmitted from dogs) are like those of dumb madness, in the absence of excitement and the development of paraplegia, which, as in dogs, takes the form of "acute ascending paralysis" (cf. p. 461). A man who was bitten by a rabid cat in 1886 died under Dr Bristowe in St Thomas's Hospital with symptoms of this kind.

Distribution and frequency.—At one time hydrophobia was supposed to occur chiefly in temperate climates, but this is not the case.* Like other specific diseases it is often absent from a place for several years together, until some accident introduces it. Thus it has recently been much more common in London than for many years before.

Between 1820 and 1830 six cases of hydrophobia occurred in Guy's Hospital. Two occurred in 1831, one in 1837, and then none for nearly twenty years. In 1856 there was a single case admitted, in 1865 two, and in 1866 the case of a private patient of Mr Forster's was alone recorded. Then again there was a pause for several years, the next case occurring in 1874. In 1875, another was admitted, three in 1877, and one in 1878. After five years' interval there was one case in 1883, two in 1885, and one in 1886.

The Registrar-General's returns for London show twelve deaths from hydrophobia in 1838, and four in 1839. Then only one, three, four, two, three, two, in the successive years to 1845; none in 1847, '49 and '52, and only one in 1846, '48, '50, '51 and '53. Seven were returned in 1854 and two in 1855 and '57. None in 1856 and none in 1858, '59, '60, '61 and '62. Two in 1863, and none again in 1864, but nine in 1865, and six in 1866. There were three in 1867 and '69, none in 1868 and '70; one in 1871, '72, and '73. Then there were nine in 1874, six in 1875, six in 1876, and sixteen in 1877; five in 1878, two in 1879, three in 1880, five in 1881, four in 1882, eight in 1883, nine in 1884, and in 1885 no less than twenty-six.

Such evidence of the disease occurring in epidemics is corroborative of the view that it is always due to contagion. Another circumstance pointing in the same direction is that rivers sometimes seem to limit its diffusion in a remarkable manner. Schrader has stated that in 1852 and 1853, when it was raging in Hamburg and in the neighbourhood on both sides of the Elbe, no cases occurred on the islands on that stream. According to the popular belief, the disease is more frequent in the hot season than during

* The following statement of the deaths from hydrophobia which occurred in the Punjab is taken from a report by Brigade-Surgeon Bellew:—Population of the province, 17,514,978. Deaths from hydrophobia: in 1879, 69 deaths; 1880, 107; 1881, 139; 1882, 128; 1883, 117; 1884, 158; 1885, 146.

winter and spring. Of 132 cases throughout England and Wales, fifty-one occurred in July, August, and September.

Of those persons who are bitten by rabid animals, some only are attacked by hydrophobia; several writers agree in fixing the proportion at about 50 per cent., but this is very doubtful. Some of those who escape perhaps owe their immunity to an idiosyncrasy which renders them insusceptible of the virus; at least this appears to be the case in the dog. Sir Thomas Watson mentions that a dog was caused to be bitten on thirty different occasions but without any result. Another circumstance affecting the transmission of the disease may be the presence of clothes or other coverings, by which the teeth are wiped clean of the virus before they penetrate the skin. The undoubted fact that rabid wolves are more dangerous than dogs may be due to their flying straight at the naked throat. Writers are agreed that a breach of the cutaneous surface is necessary to allow of the entrance of the poison; in Sir Thomas Watson's oft-quoted case, however, both the coachman and his fellow-servants declared that the terrier dog by which he was inoculated had drawn no blood, but merely indented the skin of the hand. Mucous membranes are supposed to be capable of being infected without any interruption of their continuity. A case is recorded in which a rabid dog licked the face of a sleeping man near his mouth and communicated the disease, although not the slightest scratch or abrasion could be found after a very strict search.

The large experience recently obtained at the Pasteur Institute in Paris amply confirmed Mr Forster's belief that bites on the face are the most fatal and probably they have the shortest incubation. Next come the hands, and lastly, the parts habitually covered.

A question of some consequence to pathologists is whether the virus retains its powers after the death of a rabid animal. Mr Youatt thought that this was not the case, but Bollinger cites the case of a student at the Veterinary College at Copenhagen, who opened the body of a dog that had died of the disease the night before; his finger was slightly fissured at the time, and about six weeks afterwards he died of hydrophobia.

Seat.—Until lately it has been generally admitted that hydrophobia, like the neuroses in general, has no morbid anatomy, at least so far as the nervous centres are concerned. But Benedikt as early as 1875 ('Virchow's Archiv' for that year, p. 537) and Dr Gowers ('Path. Trans.,' 1877), Dr Coats ('Med.-Chir. Trans.,' 1878), and Dr Ross ('Path. Trans.,' 1879) have found that the vessels in the bulb towards the floor of the fourth ventricle are surrounded by masses of leucocytes within their sheaths, and sometimes collections of these exuded cells (miliary abscesses) occur among the nervous elements. Similar changes, but less marked, were also found in the spinal cord. They do not, however, occur in all cases, for in some beautiful preparations made by Dr Frederick Taylor no deviations from the normal appearances are to be recognised. On the other hand preparations made from the patient who died in our wards in 1885, by Dr Hale White, showed unmistakable extravasation of leucocytes, and here and there of blood-discs, in the sheaths around the vessels in the bulb. In the spinal cord and in the brain the lesions were more scanty and doubtful.

Experimental evidence confirms the belief that the cord and bulb is the seat of the disease.

Other conditions, more or less constantly found after death, are congestion of the mucous membrane of the pharynx and epiglottis, of the stomach

with hæmorrhagic erosion, and of the lungs. They are more uniform in rabbits than in human beings.

The real *pathology* of the disease is one of the most difficult problems that we have to encounter in the science of medicine. What becomes of the virus during the prolonged and indefinite stage of incubation? What change ushers in the development of the disease itself, which is now to prove fatal within a very few days? Some writers suppose that a "recrudescence" takes place, the poison having hitherto been imprisoned in the wound, but being at this time absorbed into the blood. One cannot imagine that hydrophobia is due merely to a transference of morbid action along the nerves (a peripheral ascending neuritis migrans, such as perhaps occurs in tetanus), for fresh poison is generated, so that the saliva and even the blood become infective. Indeed, if it is the fact that a dog can communicate the disease during the period of incubation, the whole theory of recrudescence must be given up.

There can, however, be no reasonable doubt since Pasteur's experiments, to be presently described, have been made, that hydrophobia is a specific contagious disease, a "zymotic" "blood-poison." The microbe on which it probably depends has not yet been identified.

Diagnosis.—Hydrophobia is not generally difficult to recognise. There has been no doubt as to the nature of any of the cases, all of them fatal, that have been observed at Guy's within the last few years. One of the oddest of all vagaries of medical opinion is the notion that there is really no such disease, and that all the persons who are supposed to have died of it have really succumbed either to a traumatic tetanus or to fright. This idea seems to have been started early in the century by Bocquillon, and it was upheld by Sir Isaac Pennington, Regius Professor of Physic at Cambridge, and more recently by Prof. Maschka, of Prague. But as Sir Thomas Watson long ago remarked, young children and idiots, who could never have understood anything about the disease, have fallen victims to it, and many of the adults who have been attacked have been men of strong minds who have refused to believe that they were seriously ill. As for tetanus, the symptoms in most cases are altogether different.

It must, however, be admitted that medical literature contains a considerable number of well-recorded cases, concerning which it is perhaps impossible to say positively whether they were instances of hydrophobia or not. The difficulty is with regard to those cases in which recovery is supposed to have taken place. For all experience tends to show that whenever the clinical characters of hydrophobia are present in a typical form the patient almost invariably dies, or, in other words, nearly every case in which the fatal issue has been arrested seems to have presented some aberrant feature or other which casts a doubt upon its genuineness. And yet one cannot deny that, taken as a whole, the symptoms have often resembled those of hydrophobia more closely than those of any other known disease.*

* Here, for example, is a case recorded by Dr Nicholls, of Chelmsford, in the 'Lancet' for 1878. The patient, a carter, aged twenty-five, was bitten by a stray dog in a neighbourhood where there had recently been a case of hydrophobia, and where several dogs known to have been bitten were still at large. Some weeks afterwards, having in the meantime thought little about the matter, he became weary, his legs ached, and he had loss of appetite. Two days later he refused a glass of ale at a customer's house, saying he could not drink it. On the fourth day he drank a quantity of coffee, but said that he "gulped it down," and complained at intervals of his throat. He was also much annoyed by trifling noises, and particularly by a toy windmill outside his house. On the fifth day, having delivered a load of coals in the country, he called at a public-house, and after three

In some cases of epilepsy, hysteria, or mania, there is spasmodic difficulty in swallowing liquids which may be more or less like that which occurs in hydrophobia. But the doubt, if any, is soon dispelled by the progress of the disease.

Treatment.—The treatment of hydrophobia by drugs is hopeless. Morphia, chloral hydrate, or inhalation of chloroform, relieve the spasm for a time if employed boldly, but do not even postpone the fatal issue. Curare is probably equally useless, and the same must be said of cannabis indica, atropine, cocaine, salol, and methane, as well as of quinine, mercury, and the other drugs employed by Morgan and the older physicians (see Mr Dowdeswell's paper in the 'Proc. Roy. Soc.' for 1887). Darkened rooms, hot baths, electricity, and every other appliance that reason or credulity could suggest have been fully tried with equally negative results.

Promoting free bleeding from the wound, sucking and washing it, and applying nitrate of silver or other suitable caustic,* are rational methods of treatment, and, if adopted early, may undoubtedly prevent the virus from reaching the general circulation, and thus save the patient's life.

Since, however, the first edition of this chapter was written, everyone has heard of the researches of M. Pasteur and of his method of preventing or even curing hydrophobia by inoculation of an attenuated virus. The following is a brief account of these remarkable investigations.

Starting from the belief that hydrophobia in man and rabies in dogs and other animals is a contagious disease, Pasteur ascertained that the fresh cord and bulb of a rabid dog, when pounded up with sterilized broth, makes a highly contagious materies morbi; and this, injected under the skin or into the veins, or, most certainly and rapidly of all, under the dura mater, will reproduce the disease in a dog, cat, rabbit, or any other susceptible animal. As above stated, the symptoms vary for different species and even for different individuals; but after death the cord will again furnish the contagium, so that the identity of the disease is proved.

Next he set to work to attenuate the virus, as he had that of anthrax (cf. *supra* p. 375); and devised a plan of drying the cords in a warm, dry, sterilized atmosphere (20° C. is the temperature chosen, or about 68° F.); by this means the virus is rendered less powerful, and becomes progressively weaker day by day. When broth, charged with these dried cords, is introduced into a dog, he undergoes the disease in a mild form, and can then without danger be inoculated with a stronger virus, until at last he is

efforts got down a pint of ale. He then muttered that he was as thirsty as ever, wherefore another cup was passed to him, but on attempting to sip from it he failed. After this he drove into the town, dashed through the streets at a gallop, and became maniacal and unconscious. Dr Nicholls was called to him and found him with a rope tied round his legs, struggling furiously, and beating his arms and head. He was uttering a peculiar noise, between a howl and a scream. His face was livid and covered with a cold sweat; his jaws were clenched; he was foaming at the mouth. Convulsions like those of tetanus then came on and continued at intervals for about twenty-four hours, being, however, controlled to some extent by inhalations of chloroform. The opisthotonos was extreme, the body resting on the head and heels for a minute at a time. On the evening of the sixth day he partially regained consciousness, and asked for drink, which he gulped down in small quantities, the greater portion being expelled from the mouth. Two days later he displayed a remarkale horror of anything white, such as a bandage, a basin, a white glove, &c., turning aside and becoming convulsed as soon as he saw them. By about a week from this time he was well. If the disease was not hydrophobia, what was it?

* Mr. Youatt did not practise excision, but trusted entirely to cauterisation with nitrate of silver. He himself was bitten seven times, and operated on 400 persons besides, among whom he had only one death, which he ascribed to fright.

rendered "refractory" to the most recent and intense contagion, or to the effects of direct inoculation from a rabid dog by biting.

The efficacy of this prophylactic or "preventive" treatment (called "vaccination" by M. Pasteur) is not a matter of question. If protected and unprotected dogs or rabbits are bitten by the same rabid dog or cat, the former escape and the latter die.

It would therefore be theoretically possible by inoculating all living dogs to "stamp out" hydrophobia for ever; just as, if vaccination were made compulsory everywhere for a generation or two, smallpox would become extinct and vaccination itself no longer needful. But this is as impracticable for hydrophobia as it is at present hopeless for variola. All that can be done by way of prophylaxis is to diminish the number of dogs by taxation of the owners, to destroy masterless dogs, and to enforce the use of muzzles.

But Pasteur next tried whether it is not possible to anticipate the march of the disease, by taking advantage of its undoubtedly long incubation-period and using attenuated virus with a shorter period, so as to overtake the original virus and fortify the organism against it before its natural effects appear. Trials of this "protective" method on dogs and rabbits already bitten proved encouraging; and at last, when urged to do something to save a child named Joseph Meister, who had been severely bitten by a mad dog, Pasteur, as he tells us with great anxiety, inoculated him with attenuated virus on July 6th, 1885. The result proved satisfactory; the boy recovered, and many other persons bitten by rabid (or often, in all probability, by non-rabid) dogs applied to Pasteur, and received "protective" inoculation. The numbers increased enormously, patients arriving from all parts of France, from Italy, Russia, England, and even from America and India. A certain number of deaths with undoubted hydrophobic symptoms occurred, particularly among several Russians who had been bitten by rabid wolves. Pasteur's results were not completely confirmed by independent investigators, *e. g.* Professor von Frisch, of Vienna, and he was violently attacked, not only by fanatical opponents of experimental pathology, but by certain French physicians. The difficulties in the way of a sound conclusion are serious. For, first, no one knows how many of the people who have been inoculated had really been bitten by a rabid dog at all. Secondly, when a bite is inflicted on a part covered by clothing, the venomous saliva is generally wiped off, and so the person bitten escapes. Thirdly, many who were bitten had been well treated by caustics, and thus the virus may have been, and no doubt frequently was, destroyed before they were inoculated. Lastly, the proportion of men or animals who, from some "insusceptibility" (*i. e.* some individual unknown cause), escape the disease even when the virus is fairly injected is unknown, though judging by analogy from syphilis and from vaccinia, probably a small number by comparison.

The terrible question has also to be considered whether the intended protective inoculation may not, if unwittingly employed on persons who have not really been infected before, produce a fatal form of the very disease against which it is supposed to protect.

It cannot be said that these questions are fully answered, or that all the difficulties have been surmounted. But while these sheets are going through the press a report has been presented to the President of the Local Government Board by a Committee appointed for the purpose of investigating this

subject,* which goes far to prove to unprejudiced critics that not only has Pasteur discovered an efficient "preventive," *i. e.* strictly prophylactic treatment of hydrophobia—a fact of the utmost scientific interest and importance—but that he has also succeeded in applying the same method to the "protective" or therapeutical treatment of the same dreadful malady when it has already been contracted.

Among a total of 2682 persons inoculated at the Pasteur Institute, forty are said to have died of hydrophobia. Seven of these deaths occurred among those bitten by wolves, a peculiarly dangerous kind of rabies, and met by a more rapid and probably more hazardous process of inoculation.†

Excluding these, of 2634 persons bitten (or supposed to be bitten) by mad dogs, only thirty-three died, a percentage under 1·5, and far less than that assigned by any computation to cases treated in other ways or not treated at all.

Of 233 persons bitten by undoubtedly rabid animals (as proved by inoculation from their cords or by rabies appearing in other animals bitten by them) only four died, instead of perhaps fifty and almost certainly twenty.

Of forty-eight persons bitten by rabid wolves, only nine died, probably not a third of the natural mortality.

Of 186 persons bitten on the face (the most dangerous part) by undoubtedly rabid dogs, only one twentieth died, instead of the previously probable proportion of at least a fourth.

Ninety cases were personally investigated by Dr Burdon Sanderson, Dr Brunton, and Mr Horsley, who went to Paris for the purpose. Among them no death had occurred. In thirty-one there was no clear evidence that the dog which had bitten the patient was rabid; but in twenty-four the bite had been inflicted on an uncovered part of the skin by undoubtedly rabid dogs.

Lastly it appears certain that the ordinary inoculation as now practised by M. Pasteur is perfectly safe, although there is room for fear that the *méthode intensive* may be itself attended with a certain risk.

Whether the protection will last a lifetime, or longer than the two years or more which have now elapsed since it began to be practised, cannot of course be at present known. It is possible that further experience may modify the conclusion above stated; but no dispassionate critic can withhold his admiration from the insight and ingenuity, the zeal and perseverance, which M. Pasteur has displayed, or can doubt that humanity is indebted to him for what, on the lowest estimate, is a more than promising method of treatment of a frightful and otherwise hopeless disease.

* The Committee consisted of Sir James Paget, chairman, Sir Joseph Lister, Sir Henry Roscoe, Professor Burdon Sanderson, Dr Quain, Dr Brunton, and Dr Fleming, with Mr Victor Horsley as Secretary.

† The ordinary method was to inject on the first day of treatment virus derived from a cord dried for fourteen days; on the second, that of a cord dried for thirteen days; on the third, that of a cord dried twelve days; on the fourth, that of an eleven days' cord; fifth, ten days; sixth, nine; seventh, eight; eighth, seven; ninth, six; and tenth, five days. These injections were afterwards made more frequent, and the increase of strength more rapid. But for very severe cases, like those of wolf-bites, Pasteur now uses a *méthode intensive*, injecting on the first day of treatment virus derived from cords dried for fourteen and thirteen days; on the second, for twelve and eleven; on the third, for eleven and ten; on the fourth, ten days twice; on the fifth, nine days twice; on the sixth, nine days again; on the seventh, eighth, and ninth, eight days; on the tenth, eleventh, and twelfth, seven days; on the thirteenth and fourteenth, six days; and on the fifteenth and last day of treatment virus of maximum intensity derived from a cord of only five days' drying.

THE PAROXYSMAL NEUROSES

Introductory remarks—Relation to other neuroses and to each other.
MIGRAINE—*Nomenclature—Symptoms—Pathology and causation—Treatment.*
EPILEPSY—*Definition—E. minor—E. major: aura: fit: sequelæ—Pathology, seat, and causation—Diagnosis—Treatment of the fit and of the intervals.*
PAROXYSMAL VERTIGO—*Symptoms—Auditory vertigo—Menière's disease.*
PAROXYSMAL INSANITY—*Peculiarities—Danger of violence or suicide—Relation to epilepsy—Somnambulism—Night terrors.*

WE have now to consider a remarkable series of affections, which, although they differ widely in their symptoms, nevertheless have close mutual relations, and present many points of resemblance, both in their causes and in the circumstances under which they occur.

They all occur in paroxysms, and at periods which (at least in some cases) are more or less regular. Many of the persons who are subject to them are in perfect health during the intervals between the seizures. We may distinguish these affections as "paroxysmal neuroses;" or, adopting the expression of Dr Edward Liveing, as "nerve-storms."

The chief among them are Migraine, paroxysmal Vertigo, Epilepsy (including both the *petit mal* and the *haut mal*), Catalepsy, Somnambulism, and Paroxysmal Insanity. With these several others should probably be included :—Tic douloureux, Angina pectoris, paroxysmal Gastralgia, spasmodic Asthma, spasmodic Croup, aud Laryngismus stridulus. For reasons of convenience, however, these are treated elsewhere.

No modern writer has so clearly pointed out the relations of these various diseases to one another as Dr Edward Liveing, who in his treatise on 'Megrim or Sick Headache,' insists on the fact that in the same patient they are often transformed, one into another, in the course of time. Most other writers have dealt with them from too narrow a standpoint; they have aimed at including too much under some single affection, such as epilepsy, rather than at giving a comprehensive view of the whole group.

Common features of the group.—An important character of most of these diseases is that they are essentially *innate* and *hereditary*. This, however, is common to many other neuroses as well; and so also is the circumstance that in different members of the same family the inherited tendency may show itself in different ways—one child being epileptic, another asthmatic, a third subject to migraine, and so on. Indeed, it would be incorrect to represent the paroxysmal neuroses as a perfectly isolated group of affections, bearing no relation whatever to other nervous diseases. On the contrary, there are unmistakable evidences of such a relation; as, for instance, between Epilepsy on the one hand, and Chorea and Hysteria on the other. But by studying Migraine, Vertigo, and Epilepsy together, one is led to more just conclusions than by viewing them either as altogether independent affections, or, on the other hand, as unconnected members of the large class of neuroses in general.

In more than one of the affections now to be described, each attack is made up of a regular *succession* of phenomena. In migraine it often begins with a peculiar affection of sight; afterwards follow in turn numbness of the fingers, vertigo, headache, vomiting and sleep. In epilepsy there is perhaps first a sensation passing up from one of the limbs to the head, and then successively: complete insensibility, a general tonic spasm, a series of clonic convulsions, and a prolonged stupor. We can hardly fail to regard such seizures as dependent upon the gradual extension of a morbid change from one part to another of the nervous centres; and it seems highly improbable that any part of them should essentially depend upon a mere alteration in the cerebral blood supply, as supposed by many writers.

Another point in which most of these affections agree is that the attacks gradually *culminate* in a certain pitch of intensity and then subside; and yet another is that there is a kind of *compensation* between the frequency and severity of the paroxysms, a slight one being followed by another at an unusually short interval, while a severe seizure often ensures a long period of repose.

The seizures themselves are often directly traceable to *causes* which are similar for all the members of this group. In describing the different varieties of "nerve-storm," one has again and again to mention gastric or hepatic disorder, irritation of the brain from dentition or disease of the teeth, exhaustion from deficient food or excessive bodily exercise, affections of the sight, smell, or hearing as giving rise to a first attack, or to the succeeding ones also. Many of these neuroses bear a relation to puberty and the other great epochs of life, to the appearance of the catamenia in women, to pregnancy and to the puerperal state. Most of them are apt to break out for the first time at some particular age; migraine and epilepsy about puberty, laryngismus stridulus and spasmodic croup during infancy, as we have seen that tetany and chorea are diseases of childhood. Some of the paroxysmal neuroses tend to disappear spontaneously when a certain period of life is reached; thus, migraine often ceases to recur after the age of fifty.

Another feature which is common to the paroxysmal neuroses is their tendency to undergo *metamorphosis* in the same patient as time goes on. This is particularly the case with epilepsy; its attacks are liable to be replaced by vertigo, catalepsy, mania, or other forms of nervous disturbance. Indeed, if we had convenient English names for the *petit mal* and the *haut mal*, it would be well that they should be described separately, for their relation to one another is not very much closer than to some of the other affections treated in this chapter. The connection between migraine and epilepsy is certainly much less intimate, but certain cases have been regarded as proofs that they also are related.

MEGRIM.*—*Definition and nomenclature.*—An attack of this disease in its most typical form begins with a peculiar dimness of sight; after a little while the fingers of one hand may become numb and tingle, or the patient may experience some difficulty of speech; presently the power of vision is restored, but a more or less severe pain in the head comes on, which may last for some hours; before it ceases vomiting often takes place. The same

* *Synonyms.*—Hemicrania (*ἡμικρανία*), whence Fr. Migraine and Eng. the Megrims—Paroxysmal Sick-headache—Bilious headache (in part)—Clavus hystericus—Hemicrania periodica and Hysteria cephalica refer rather to supra-orbital neuralgia than to Megrim.

succession of phenomena recurs again and again at more or less regular intervals.

Great confusion prevails with regard to the nomenclature of this complaint. Many names for it are in use, but each of them is properly applicable to only one of the various forms it may assume, and several of them would naturally be taken to include other and distinct affections.

The pain is often limited to one side of the head. To such cases the name "hemicrania" has been applied since the days of the old Greek writers; and that word has undergone corruption into the French *migraine* and the English *megrim*. In strictness, therefore, all these terms are inapplicable to any case in which both temples ache at the same time, but we shall presently see that such cases constitute the majority, and certainly cannot be separated from the others. Still less can those instances be isolated in which the pain is intense and limited to a very small point, as if a nail were driven into the skull—*clavus hystericus*.

Again, the defect of vision has by some writers been styled Hemiopia, or Hemiopsia, from its affecting only one half of the visual field. We have seen that this symptom occurs as the result of organic lesions of the nervous system (*supra*, pp. 573, 618, 682). Aiming at still greater accuracy of expression, Dr Hubert Airy has recently proposed to call it *teichopsia* (τεῖχος, a city wall, ὄψις, vision) from a remarkable feature of the disordered vision to be presently described. But as this visual affection, even when it is present, generally constitutes only a small part of the complaint, it would be far better to use the name for the symptom only. Lastly, we have the English terms *bilious headache*, *sick headache*, and *sick giddiness*. But many cases present no indication of gastric or hepatic disorder; and, on the other hand, there is a different form of headache in which such disorder plays a far more important part. Evidently one name should be given to all the varieties which the same disease may assume. And, if we can forget its etymology, the word migraine appears to be by far the most suitable for the purpose. Dr Liveing, indeed, has already extended its signification in this way.

Symptoms.—There is one symptom of megrim which invariably precedes all the others if it occurs at all; this is the *affection of sight*. The patient first notices that he cannot see distinctly some part of what he is looking at. The portion of the visual field which is thus blotted out is originally very small. It may be at the exact centre of the field, but more generally it is a little to one side of it, although so near that in reading from a printed page he has to glance slightly away from the word he wishes to see. The dim spot is not black, but seems like a faint cloud, of the same colour as the rest of the surface upon which the eyes are directed. It quickly begins to enlarge, and gradually overspreads more or less of one lateral half of the field. It often acquires a peculiar zigzagged outline, with angles like those of a fortification; this is why Dr Airy proposed the name of "teichopsia." The surface within seems to have a peculiar undulatory motion which has been compared to that of a boiling liquid; and the angles themselves appear to flicker or to revolve. The form of the cloud is originally oval, but as it grows bigger, a gap forms in that side of it which is towards the centre of the field, so that it becomes horseshoe-shaped. One area of the curve then seems to touch the point of exact vision; and in this the angles are much smaller and closer together than in the other area, which spreads away into the outer part of the field of sight. Sometimes the cloud is uncoloured; some-

times it presents brilliant gleams of red, blue, and other colours. If the eyes be closed, or if the person should go into a dark room with his eyes open, the whole figure appears to be faintly luminous. As it increases in size, the middle of it clears up and accurate vision then is regained. On a printed page, for instance, a few letters can now be plainly recognised in the midst of the glimmering horseshoe-like curve. Before long the latter likewise disappears, and the person can see as well as ever. The whole process occupies from ten to twenty minutes, or at most half an hour.

While the oval cloudy patch and its zigzagged border are visible, they are seen in their minutest details by both eyes alike. To this rule a single exception has been recorded by Sir John Herschel, who once satisfied himself that his left eye was alone affected. But I cannot help thinking that he may have been mistaken; for, as Dr Airy points out, everyone is at first inclined to suppose that one eye only is concerned, namely, that of the side on which part of the visual field is blotted out. The fact that the impairment of vision is thus almost constantly referred to both retinæ of course proves that the seat of the affection is above the optic chiasma. And limitation to one half of the field is just what we should expect from our knowledge of the anatomy of the nervous centres, the halves of which are in great measure isolated from one another, each communicating with corresponding halves of the two eyes. Sir John Herschel, indeed, has stated that in his own person he once observed "the shadowy pattern of a fortification which passed completely across the field of vision from left to right." If this was so, we must suppose that on that occasion the disturbance (of whatever nature) which is the cause of the affection extended over to the opposite side of the brain at an early period of the attack. We shall hereafter see that at a later period this commonly occurs.

In some cases, as soon as natural vision is restored, the attack is at an end. But in the great majority, a more or less severe headache comes on at this period. It is generally said that a precisely similar headache may also arise without having been preceded by any affection of sight. It is, however, permissible to hint a doubt whether this really occurs, or at least whether it is not far more rare than has hitherto been supposed.

A very curious circumstance in regard to the visual affection is that some of the best and most careful descriptions of it have been written, not by medical men, but by astronomers and natural philosophers. Wollaston, Arago, Sir David Brewster, Sir John Herschel, Sir Charles Wheatstone, Du Bois Reymond, Sir George Airy, and Professor Dufour, of Lausanne, may be mentioned as having been liable to this paroxysmal defect of sight, and as having carefully noted its phenomena; and no similar malady has, within the present century, been the subject of two papers admitted into the 'Philosophical Transactions,' as well as of communications to the 'Philosophical Magazine' and other scientific journals at home and abroad. It may be a question whether persons who are not accustomed to employ the eyes for minute observation would notice the dimness of sight, or regard it as of sufficient importance to be mentioned to their physician. Indeed, when it commences at some distance from the centre of vision, I believe it is sure to be overlooked, unless the patient's attention is specially directed to its occurrence. And this may, perhaps, be the reason why Professor Du Bois Reymond does not mention it in describing this form of headache as he has experienced it himself.

The *pain* of megrim varies greatly in severity both in different cases,

and in the same case at different times. It commonly begins at some one spot in the brow or temple and gradually spreads all over these regions. Sometimes it remains confined to a single point, which is generally over the frontal or parietal bone on one side. It was to these cases that the special name of *clavus* was formerly applied. A strict limitation, however, is altogether exceptional. According to Dr Liveing, it is not even the rule that the headache should keep to one half of the head. He finds that in the majority of cases it affects the whole forehead and both temples, although with more severity on one side than on the opposite. It often extends to the orbit, and is referred with special intensity to the back of the eye. More rarely it passes behind the ear to the occipital region. Some writers describe it as of a stabbing, cutting, or boring character; others as throbbing, and especially as undergoing augmentation with each beat of the heart. It is generally much increased by every bodily movement that the patient makes, by exposure of his eyes to light, and by every noise that he hears. He therefore lies down, and keeps the room as dark and quiet as possible. But Dr Wilks knows of one patient in whom the recumbent posture aggravates the pain, and who will sit up all night rather than lie down until the attack has passed off; and Dr Liveing speaks of cases in which the pain is so intolerable that the sufferer cannot lie in one position for any length of time, but is obliged to get up and move about. It has been said that the patient can sometimes make the visual affection more marked, if not increase the headache, by lying on one side rather than on the other; namely, the side opposite to that on which the dimness of sight is observed. But this appears to be seldom the case.

The headache scarcely ever remains long at the same pitch of intensity. Generally it goes on gradually augmenting in severity until it reaches a culminating point, after which it begins to decline. Its increase is usually steady, but sometimes this takes place by fits and starts, with more or less distinct remissions.

When the pain becomes very intense the patient often begins to feel *nausea;* and presently he retches and is sick. Anything that the stomach may contain is rejected, including sometimes a considerable quantity of undigested food. But frequently it is empty; and then the retching is ineffectual, or some mucus at first is brought up, and afterwards a bilious fluid. After the occurrence of free vomiting the pain often quickly passes off, and thus many persons regard it as curative, saying that they get relief as soon as they can be sick.

But in some individuals, however severe the headache may be, sickness seldom or never occurs. In them the pain gradually passes off of its own accord. Very frequently it lasts for the remainder of the day; the patient at length becomes worn out and drops off to sleep; and when he wakes on the following morning he finds that his suffering is over, all that remains of it being a slight soreness of the forehead or temple. Some persons lose the pain if they can sleep for a short time, even during the early part of an attack. Dr Liveing mentions a gardener who, if he could at the commencement leave his work and lie down under the shade of a tree, would wake at the end of half an hour as well as ever. Lastly, in some rare cases, the complaint terminates by epistaxis, by a copious secretion of tears, by profuse perspiration, or by the flow of a large quantity of pale urine.

Less common symptoms.—An attack of megrim may be attended with other symptoms beside those which have been hitherto described. In

certain cases, which are generally of considerable severity, common *sensation* is impaired in one hand, especially towards the ends of the fingers; and a feeling of tingling, thrilling, or formication may also be experienced. Sometimes the surface of all one arm and of the corresponding leg seems to have gone to sleep, or numbness may extend to the mouth, the lips, tongue, or throat. Dr Liveing says that all these parts are affected bilaterally. Dr Anstie noticed in his own person that, even in the intervals between the attacks of pain, the power of distinguishing impressions was permanently less in the skin round the inner angle of the right eye than on the opposite side; during and after the paroxysms the impairment of sensation was always more marked, and also affected a more extensive area.

Occasionally *muscular power* seems to be more or less distinctly impaired. Ptosis and strabismus from paralysis of one of the recti muscles have each been present in cases which have been regarded by good observers as of this nature. Some patients have been known to drop things which they were carrying in the hand; but Dr Liveing suggests that this may be due to a loss of the sensations which should guide the muscles, rather than to a true muscular palsy. Another symptom which is sometimes, but not often, present is *giddiness* or vertigo.

Again, in some cases the faculty of *speech* is disordered. The patient has difficulty in finding the expression which he wishes to use, or in constructing a coherent sentence. He may even substitute one name for another; and an instance is recorded by Dr Liveing in which not a single word could be uttered. This may occur without any confusion of thought, or the patient may be painfully conscious that his memory is failing him and that his intellect is embarrassed. Actual hallucinations are very rare. But there are often much general mental depression, and a vague and unac countable sense of anxiety and dread.

Such serious symptoms are uncommon. Hence the patient who is subject to migraine may fail to perceive that they have any connection with his habitual complaint. The period at which they commence is generally before the headache, and after the affection of sight has continued for some time.

An oppressive *drowsiness* is sometimes noticed, and the patient may even lie in a half unconscious state, not heeding when he is spoken to; but this symptom rather accompanies than precedes the pain in the head.

In some instances there are also symptoms which indicate an implication of the vaso-motor system of nerves. The temporal artery becomes enlarged, and its tortuosities are much more plainly visible on the affected side of the head; it feels hard and like a cord to the touch. The conjunctivæ may be much reddened. The *pupil* is sometimes altered in size, but observers are not agreed as to the character of the change. Du Bois Raymond says that in himself it is always dilated; Piorry and Latham describe it as being contracted. The eyeball is said to appear retracted in some cases. In one instance Möllendorff found with the ophthalmoscope that the background of the affected eye was of a bright scarlet colour, the optic papillæ red and œdematous, the central artery and the veins enlarged and tortuous. These facts have a theoretical interest as indications that the sympathetic nerves play an important part in the production of migraine.

Again, after the subsidence of these paroxysms, certain very curious changes are sometimes observed in the tissues of the affected parts, which can only be ascribed to an interference with their nutrition consequent on

the nervous disturbance. One such change is a localised *greyness of the hair*. Anstie relates that when he himself had a severe attack the eyebrow would show a distinct patch of grey opposite the supra-orbital notch, but that subsequently the individual hairs regained their natural colour. He found that as many as eleven out of twenty-seven patients showed more or less greyness of the hair of the forehead and temple on the side on which they suffered most pain. In other cases the hairs become brittle or fall out. Some years ago I was consulted by one of our students, who wished me to examine the hairs from his eyebrows with a microscope to see if I could detect any fungus. More than half of each eyebrow, at its outer part, had become denuded of hair; and this condition was more marked on one side than on the opposite. I at once inquired whether he was liable to migraine and found that this was the case, and also that it was especially severe on that side on which the eyebrow was the more deficient. In a few weeks, under treatment for the neurosis, the hairs began to grow again.

According to Anstie, more or less thickening of the solid tissues occurs in many cases as the result of repeated attacks of migraine; he even observed periosteal swelling, which had a close resemblance to syphilitic nodes, but which he believed to have no such origin. He also speaks of iritis, glaucoma, opacity and ulceration of cornea as resulting from neuralgia of the fifth nerve; but it is not certain whether such effects occur in cases of true recurrent migraine. An erysipelatoid eruption ought perhaps, however, to be mentioned; for Anstie relates more than one instance in which a patient suffered from two or three successive attacks of this kind, in connection with neuralgic pain. The upper eyelid is sometimes greatly swollen.

Another affection which is frequently consecutive to megrim is *xanthelasma* of the eyelids. Except in cases of jaundice xanthelasma almost invariably affects the eyelids, and occurs nowhere else.* It always begins near the internal canthus, and generally first in the upper lid; and a curious circumstance is that (according to Mr Hutchinson) it constantly appears on the left side earlier than on the right. This observer found that most of the patients in whom he noticed xanthelasma of the eyelids had suffered from frequent sick-headaches, and some of them very severely. The cutaneous affection seldom appears in those who have not reached the age of thirty-five or forty years. I have seen it develop itself in three successive generations; and Dr Church has placed on record a family tree in which it recurred again and again, but unattended with any liability to migraine.

The general *circulation* is interfered with in severe attacks of migraine. Möllendorff has found the beats of the heart reduced to fifty-two or even forty-eight per minute. The pulse at the wrist becomes small and contracted. The hands and feet are cold. The face is pale and haggard, and dark borders appear round the margins of the orbits.

The side affected.—It seldom if ever happens that megrim, in all its attacks in the same patient, attacks the same side of the head. But, as a rule, there is one side rather than the other which is especially apt to be affected. Tissot, however, mentions the case of a lady who had it alternately on each side with great regularity. Wollaston and Sir George Airy may also be mentioned as having been equally liable to the affection on either the right or

* The rule is not absolutely true. See cases reported in the 'Pathological Transactions' for the year 1882 (vol. xxxiii, p. 372, and third table, p. 383). I have seen two myself, in addition to these.

the left side. With regard to the mutual relations of the various phenomena which make up an attack there are some differences of opinion. Dr Latham describes the headache as beginning, and as more intense, on the side *opposite* to that on which the dimness of sight is noticed, but Dr Liveing deduces from the observations which he has collected that they often both occur on the same side. Each writer says that when the affection of sight and the numbness in the fingers are present together, it is on the same side of the body. Lastly, Dr Liveing has pointed out the fact—interesting in connection with what has been said as regards aphasia from organic changes—that when the speech is interfered with in migraine there is very generally numbness in the fingers, and that this always affects the right hand, either alone or in association with the left. He has not met with a single instance in which sensation was impaired in the *left* hand only, and in which there was any affection of the speech.

Pathology.—Megrim is undoubtedly one of the paroxysmal neuroses. As regards its anatomical seat, the disturbance which causes the affection of sight must occur somewhere above the optic chiasma. Indeed, this was long ago pointed out by Wollaston, who observed that the hemiopia which he described would probably be found to arise in the thalamus of one side. And, as Dr Liveing says, the only correction that this statement seems to require at the present time consists in the inclusion, within the area of disturbance, of the ganglia of the sensory nerves, down to the nucleus of the vagus. The order in which the symptoms follow one another in the several attacks renders it likely that the affection generally starts in the region of the thalamus and corpora quadrigemina or geniculata, and passes downwards and backwards along the sensory tract. The numbness and tingling are, perhaps, due merely to disturbance in the thalamus itself. But when disorder of speech and impairment of memory are present, one can hardly doubt that the "storm" must have spread upwards towards the convolutions. That it may also extend to the opposite side of the brain, along some of the commissural fibres, appears to follow from the facts that the numbness in the tongue and throat is sometimes bilateral, and that the headache is frequently so.

As to the exact nature of the change in the sensory tract which gives rise to migraine, nothing definite can at present be stated. The most recent hypotheses refer it to vaso-motor disturbance. Thus the theory of Dr Latham (1872) is that in the early stage the affected side of the brain is anæmic; that the contraction of the blood-vessels of this hemisphere is itself due to a morbid activity of the sympathetic nerve; and that this in its turn results from a defective control or inhibition on the part of the cerebro-spinal system, which he supposes to be enfeebled. In the stage of headache he supposes that there is a secondary hyperæmia, consequent upon exhaustion of the vaso-motor apparatus. Du Bois Reymond had previously (1860) maintained that, at least in his own case, migraine was due to a tetanus of the muscular coats of the vessels of the affected side, in the territory of the cervical portion of the sympathetic. On the other hand, Möllendorff (1867) and Wilks (1869) maintain that the complaint is caused by a paralysis of the very same nerves, with dilatation of the vessels and consequent hyperæmia.* The former writer lays great stress on the fact (which had been pointed out nearly a century ago by Dr Parry, of Bath) that compression of the carotid on the affected side of the head often

* Dr Eulenberg thinks that both theories are right, and that certain cases are due to spasm, others to paresis of the vaso-motor nerves.

removes headache as if by magic, though only for a time. But diminishing the blood supply to one side of the brain may very well suspend for a time the disturbance in it which is felt as pain, and yet the disturbance need by no means have been caused by an overflow of blood. Moreover, as Dr Liveing points out, the statements of different observers with regard to the condition of the pupil are so diametrically opposed that no other inference seems possible but that it must really differ in different cases; while as for the dilatation of the temporal artery, the flushing of the face, the redness of the conjunctiva, the injection of the fundus of the eye, there is direct evidence that each of them is only occasionally, not constantly present; so that the only possible conclusion seems to be that all these vaso-motor phenomena are accidental rather than essential characters. As Dr Liveing remarks, there is a clear analogy between the paroxysmal neuroses, and certain minor consensual and automatic movements: such as sneezing, coughing, and gaping, to which may be added ejaculatio seminis—a short convulsion, as it has been termed, and in certain cases accompanied by a true epileptic attack; yet no physiologist thinks of referring any of these to vaso-motor disorder.

Thus it would seem that at present we can form no clearer conception of an attack of migraine than that it is a "nerve-storm," the result of an irregular accumulation and explosive discharge of nervous irritability.

Ætiology.—The *hereditary* character of megrim is well marked. Dr Liveing found that in twenty-six cases out of fifty-three it was said to be a "family complaint;" and the twenty-six patients in question had among them forty near relations who were liable to it. In many cases it is transmitted without the slightest change of type, and sometimes it passes from a parent to those children only who in other respects resemble him. Sometimes, however, a daughter inherits megrim from an epileptic mother.

As to the relative liability of the sexes, women appear to be more prone to this complaint than men. Eulenberg believes that five women have hemicrania to one man.

The *age* at which it commences is generally about the seventh or eighth year, at the beginning of the second dentition, but sometimes it is the period of puberty, and sometimes that of early adult life. It rarely occurs for the first time in a person over twenty-five or thirty. The more marked the hereditary tendency the greater the probability of its beginning in childhood. At about the age of thirty, persons often suffer from it much more severely than previously; and, as Anstie remarks, at this period the attacks cease in many instances to be accompanied by vomiting, so that the complaint is no longer regarded as mere "sick headache," and the patient consequently seeks medical advice, perhaps for the first time. When fifty years of age are reached, or even rather earlier, it commonly happens that the liability to migraine ceases; old people seldom suffer from it.

In some persons the attacks of migraine recur with regular periodicity, and this has led to the supposition of some more or less remote *malarial* origin. The period is sometimes a fortnight, sometimes a month, sometimes longer still. There are, however, cases in which it is much shorter. We shall presently see that the immediate exciting cause of the paroxysm is very often excessive fatigue from brainwork. The constant repetition of this may render the attacks correspondingly frequent. Thus, a bank clerk for a considerable time had an attack regularly every week-day, but was free on Sundays; and some years ago a governess was under the

author's care who had a headache every night. In cases of this kind some of the more characteristic features of the complaint are very apt to be missing; but their relation to true migraine can often be established by the account which the patient gives of his previous state of health. Further observation may perhaps show that a headache which is persistent, and lasts for a great length of time, may grow out of the paroxysmal affection. Such a case would be strictly parallel to one of epilepsy, in which the so-called status epilepticus is developed.

In the cases just referred to the complaint may return every day, or every other day, as regularly as the paroxysms of an intermittent fever; and this fact, together with the striking therapeutical influence of quinine, often makes it difficult to exclude the possibility of miasmatic poisoning. But, on the other hand, these circumstances, taken by themselves, are far from justifying the conclusion that a migraine is really due to such a cause, and deserves the name of "brow ague." I do not myself believe that in persons living in London this origin of the complaint can ever be established satisfactorily; and it is probable that even in districts where marsh miasm prevails, cases of simple migraine and of other forms of neuralgia are often wrongly ascribed to it, just as I believe the same thing to have happened in the case of intermittent hæmatinuria. But it appears to be certain that it is sometimes really the cause; and in some parts of Spain a miasmatic migraine is said to be endemic. Again, Dr Macculloch has stated that this kind of headache may occur as a substitute for ague during the whole of one relapse of the disease, and that he has seen a "double tertian" ague, in which the headache and the ague fit occurred regularly on alternate days.

In England, however, it is universally believed that migraine, instead of being essentially a nervous malady, is the result of "*bilious*" disorder. And, when the attack is accompanied by vomiting, this is supposed to expel a materies morbi, in the shape of vitiated bile. Until one has happened to discuss the matter with some non-medical friend or patient of average intelligence and education, one can hardly conceive how firmly fixed the belief in question is in the mind of everyone who has not been taught the contrary. A notion of this kind could only be derived from the medical science of a former age; but one might well wonder how the teachings of a previous generation of physicians should have left behind them so absolute a conviction. The truth, however, is that it is a relic of one of the most ancient doctrines in the history of the healing art,—that of the four Cardinal Humours, one of which was "yellow," and another "black" bile. There is, therefore, no ground for suprise at the hold which the theory has on the popular mind, not only in England but wherever traditional physiology has been taught.

But although it is certain that migraine is never solely due to disorder of the chylopoietic viscera, there is yet no question that some error of diet is often the direct exciting cause of an attack in a person who is liable to it. I am not now referring to the diffused headache and giddiness which are apt to be more or less constantly present in persons who suffer from dyspepsia or from the so-called congestion of the liver; this has doubtless been often confounded with true migraine. But what is conclusive as to the reality of the influence of improper food is the fact that some persons at least can always bring on an attack by eating particular articles of diet towards the end of the interval between one headache and another; whereas, for a few days after a paroxysm, they might partake freely of the very same things

without suffering in any way. Dr Fothergill nearly a century ago stated that he had found nothing more apt to cause "sick headache" than "melted butter, fat meats, spices, meat pies, hot buttered toast, and malt liquors when strong and hoppy." A medical man who had suffered all his life from the complaint told Dr Liveing that he could never take the smallest quantity of wine nor eat the smallest fragment of burnt pastry without bringing on a headache. Many persons speak of butter and pork as particularly frequent exciting causes of migraine; and, making every allowance for the influence of preconceived opinions, it seems likely that such statements are not entirely imaginary.

In women, again, the recurrence of the *catamenia* is often an exciting cause of attacks of migraine, which, perhaps, generally precede the flux, but sometimes accompany or even follow it. Not unfrequently each monthly period brings with it a series of more or less distinct paroxysms of headache. A striking illustration of this connection is afforded by a case related by Dr Liveing, of a woman who was very liable to the complaint when menstruating, but who throughout repeated pregnancies was always entirely free from it. This writer also mentions an instance in which the headache and the catamenial discharge recurred simultaneously at fortnightly intervals.

But *fatigue* is a far more important exciting cause of migraine than either of those which I have mentioned. In some persons a straining effort, such as lifting a heavy weight, will bring it on, and in others the exertion of running is apt to have the same effect. Many patients are exceedingly liable to be attacked by it after protracted labour, such as a hard day's washing; or after prolonged exercise, particularly if the stomach be not duly supplied with food. Another frequent cause is severe mental work, but, above all, anxiety and worry. A long railway journey is apt to be followed by a paroxysm in some individuals, and in others merely driving in the streets of London has the same effect. Many persons always have a sick headache after a day's sight-seeing, or after passing an evening in a crowded concert-room or ball-room; and in some susceptible individuals an attack may be brought on by glaring lights, loud noises, or the strong odours of certain flowers. Dr Airy mentions the case of a person in whom the peculiar affection of sight was occasionally caused by looking at a striped wall-paper or a striped dress; and Sir John Herschel states that he incurred it as the result of allowing his mind to dwell upon a description of the affection.

In several of the conditions already alluded to as exciting causes of migraine, one element is *visual exhaustion.* This is true, for instance, not only of over-study, but also of railway travelling and the like. And, many years ago, Piorry propounded the theory that the complaint, or at least one variety of it, was the result of irritation of the optic nerves, from straining efforts to see very small objects, or from want of care in regulating the amount of light. Now, this view is altogether untenable, if applied to all cases of migraine, or even if limited to that form of it in which symptoms referable to the visual apparatus preponderate, the headache being slight. But it is perfectly true that when the eyes are structurally imperfect the forced use of them may be the immediate cause of attacks of migraine. The defects which lead to this result are chiefly those of the transparent or refracting media of the eyes; their direct effect is the production of spasm of the ciliary muscles, and with this is associated an irritation of nervous filaments, which may diffuse itself over a wide area within

the distribution of the fifth nerve. Every practitioner now knows that hypermetropia is a frequent cause of attacks of dimness of sight, headache, and giddiness, which recur when the eyes are used for near work for any length of time, particularly under artificial light. A student suffering from similar symptoms discovered that they were the result of the employment of too-powerful concave glasses, which he had chosen without proper advice in order to correct a moderate degree of myopia, and which he wore even when reading or writing. In this connection, too, astigmatism must not be overlooked. Another cause of such symptoms is weakness of the internal recti muscles. Three years ago a bank clerk sent to the author who had previously more than once had to give up work for a period of two or three months on account of cerebral symptoms. These had been thought very serious; but on examination by Mr Higgens it was discovered that the internal recti muscles failed to make the eyes converge properly upon near objects; and when suitable glasses were supplied, he soon lost all his complaints. In practice, therefore, one should make it a rule never to prescribe for any kind of frontal headache without eliminating the possibility of its being caused by imperfection of the eyes.

Lastly, affections of the *teeth* must not be overlooked as causes of migraine, at least if clavus be included as a form of it (see p. 422). One of the cases recorded by Mr Salter is that of a young lady, who for eight years was subject to attacks of headache, confined to a space of about the size of a crown-piece, rather to the left of the vertex. They sometimes recurred three or four times a week, beginning after breakfast and lasting all day; they were attended with great prostration. The affected spot became hot, and pressure with the hand gave relief to the pain. At length the patient conceived the idea that the left upper canine tooth, which was known to be impacted in the palate, was in some way connected with her sufferings. It was removed, and she never afterwards was attacked by the headache.

Diagnosis.—In its typical form migraine presents little or no difficulty of diagnosis. I have seen one or two cases in which a syphilitic periostitis of the margin of the orbit has produced recurrent pains of somewhat similar character; but this could not escape the notice of any but a very careless observer.

When the phenomena of the attacks are ill-developed, however, one may not find it easy to determine whether they belong to the disease now under consideration, or to some other neurosis. There is reason to believe that migraine and the *petit mal* of epilepsy are really linked together by transitional forms. Another form of migraine about which one may fall into error is that in which an affection of speech is a main symptom. Some months before his death, the late Dr J. J. Phillips had a severe attack of headache, attended with marked aphasia. When he had recovered I one day happened to discuss with him the question whether it could have been of the nature of migraine; but his fatal attack of apoplexy, which doubtless was the result of embolism, afterwards began in precisely the same way and presented the very same symptoms. It seems doubtful whether Dr Liveing is right in regarding as a mere paroxysmal neurosis a case in which attacks of loss of speech and right hemiplegia recurred, persisting for a week or more at a time.

Relation to other neuroses.—Migraine is so common a complaint, that one can hardly attach much importance to the mere fact that some other

neurosis occasionally develops itself in those who are subject to it, as indicating that there is any real relation between them. Dr Liveing, however, believes that a transformation sometimes occurs between megrim and epilepsy; and he relates cases in which persons who had suffered from the former afterwards became affected with the latter disease; but it is to be noted that some of them had relations who were epileptic. This writer also refers to an instance in which migraine became replaced after a certain period by asthma; and to another in which a constantly recurring gastralgia disappeared and was followed by a typical migraine, while this in its turn was succeeded by a kind of spasmodic croup. He also relates a case in which attacks of sick headache were followed after a time by angina pectoris; and another in which insanity developed itself.

Probably no medical man can have suffered again and again from migraine without the thought being forced upon him that such attacks must indicate such a defect of cerebral organisation as might subsequently result in serious mischief. Dr Liveing quotes Calmeil as having remarked that both the intellectual faculties and the moral disposition of the patient are sometimes impaired by the repeated occurrence of migraine. He also refers to the cases of Parry and Wollaston, both of whom, after having long been subject to this complaint, died of organic cerebral disease. But such results are in the highest degree rare and exceptional.

Treatment.—This involves two distinct questions: (1) the management of the patient during the intervals between the attacks of migraine, with a view to prevent their occurrence or diminish their severity; and (2) the treatment of the attacks themselves.

(1) Under the first head hygienic measures are of primary importance. One must insist upon the importance of daily exercise, short of fatigue, in the open air; one must prohibit an excess of brainwork, and take care that the patient is as far as possible shielded from domestic worry and anxiety. The state of the digestive organs must be carefully inquired into; and due weight must be allowed to any indications of dyspepsia or lithæmia, or to evidence that the attacks are brought on by errors of diet. But in many cases it is a mistake to restrict the patient too closely to what is termed plain food. Rather he should be advised to take a freer quantity of fat, in the form of butter or cream. Alcoholic beverages, however, should generally be avoided, or sparingly indulged in. In severe cases a change of climate is often advisable, and particularly a sea voyage.

One of the good results which may fairly be anticipated from the discussions which have taken place within the last few years as to the nature of migraine, is that those who have the care of young people should look out for the early manifestations of the complaint during childhood or after puberty, and insist upon the avoidance of over-study and of undue excitement in those who seem likely to suffer severely from it. Until recently migraine was almost universally looked upon as being at once incurable and almost unworthy of notice on the part of a medical adviser. But the truth is that if systematically taken in hand it is very amenable to treatment; and those who suffer from it know best how serious a matter it is to them, interrupting, as it frequently does, all the engagements, the pleasures and the duties of life.

Among medicinal agents, Dr Liveing says that he has sometimes found the regular administration of belladonna and hyoscyamus of great service.

Cannabis indica is also recommended; and may frequently be prescribed with decided advantage; but bromide of potassium is perhaps still more generally useful.

In many cases the preparations of iron (especially the sesquioxide and the saccharated carbonate) are very serviceable, and this even though there may be no anæmia. Strychnia in small doses (one twentieth of a grain) is said to be another valuable remedy. The author has prescribed arsenic with marked success; it was long ago recommended by Dr Bright and Sir Thomas Watson. Quinine is said to be less serviceable than it is in many forms of neuralgia, except in cases of malarious origin.

The remedies hitherto mentioned possess physiological properties which explain in some measure their beneficial action in the prevention of attacks of migraine. But there are other medicines of which the good effects are less readily intelligible. One such is iodide of potassium. Dr Todd is said to have found this more successful than anything else; and Dr Liveing mentions a case in which the attacks were so frequent and severe as to render the patient's life a burden to him, and in which five grains of the iodide three times a day set him almost free from them. In other cases chloride of ammonium seems to answer better than anything else. Valerian and valerianate of zinc are said by Dr Liveing to be sometimes of great value.

(2) The treatment of the paroxysms of migraine must necessarily depend on their severity; it is only in very violent attacks that the patient is likely to seek for medical advice. Of his own accord he will keep in a darkened room and maintain absolute quiet. If the feet be cold, they should be wrapped in blankets, and a hot bottle should be placed in contact with them; or they may be immersed in hot water to which some mustard has been added. Hydrate of chloral may then be administered in a dose of from twenty to thirty grains. Dr Anstie speaks of it as being of the greatest possible value in quickly bringing sleep to the patient, who when he wakes up may be free from pain. In some cases, and particularly when connected with neuralgia of dental origin, butyl-trichlor-aldehyde-hydrate ("croton-chloral") is still more decidedly efficacious. Bromide of potassium also is very useful; a scruple, or half a drachm, taken when the sight begins to be affected, sometimes cuts short the attack. At this period a cup of strong tea or coffee is in some persons capable of producing the same effect; even sipping hot water gives relief in some cases. But the best remedy of all appears to be guarana. This drug—prepared in Brazil from *Paullinia sorbilis*—has been principally recommended in England by Dr Wilks; half a drachm of it may be taken, mixed with water; or about twenty or thirty minims of the liquid extract. Even this, however, is sometimes altogether useless. Dr Anstie mentions the extract of cannabis indica in a dose of a quarter to half a grain as being very serviceable in the migraine of the young; he says the dose should be repeated in two hours if sleep be not obtained. In some cases a full dose of brandy or a glass of sherry or champagne is very effectual; but, as Dr Anstie points out, there is great danger in allowing a patient to fly to such remedies for the purpose of allaying pain.

Locally some measure of relief may be afforded by the pressure of a handkerchief tied tightly round the brows. Dr Liveing mentions a case in which plunging the head into cold water was often effectual. In some cases it has been found advantageous to apply to the seat of pain a little piece of

cotton wool, on which a few drops of ether have been poured, and which is covered with a watchglass; and the bisulphide of carbon has been used in a similar manner. Trousseau speaks highly of the application of extract of belladonna to the painful temple; and Anstie says that a diluted ointment of veratria is often very serviceable.

Another measure which appears sometimes to be highly successful is the application of the constant galvanic current. Anstie recommends that it should be passed from one mastoid process to the other. Only three or four cells should at first be employed, and never more than ten; it should be applied for but half a minute at a time, and repeated once or twice a day. Giddiness is very apt to be produced unless it be used with great caution.

EPILEPSY.*—*Definition.*—Convulsions which closely resemble the fits of epilepsy may accompany the onset of variola and other exanthemata; they occur shortly before life is extinguished by hæmorrhage; they result from Bright's disease; and they are produced by various organic affections of the brain.

But, in remarkable contrast with the various cases just referred to—in all of which the fits are accidental, occasional, or solitary—there are other cases in which convulsive fits of precisely the same kind return again and again for years together, and often without being traceable to any cause, except in some instances an inherited tendency to nervous disorder. One cannot but regard such cases as essentially distinct from the others, and needing a special name; we therefore speak of them as *epileptic*, and call fits which are accidental, secondary, or symptomatic, *epileptiform*. We may (if we choose) apply the term "eclampsia" to the latter generally, instead of confining it to the convulsions which are apt to occur after childbirth.

In limiting the use of the word Epilepsy to an idiopathic and recurrent paroxysmal neurosis, one must not imagine an underlying essential disease of which the fits are only symptoms. The fits themselves constitute the disease, the only other element in its definition being the clinical fact that they tend to return at more or less regular intervals for an indefinite length of time. We shall soon see that the distinction between *epileptic* and *epileptiform* attacks is not always easily applied, and that some cases of very long standing should nevertheless be classed with symptomatic eclampsia rather than with true epilepsy. On the other hand, a single fit, which is never repeated, may belong to epilepsy in the strictest sense, although the tendency to recurrence is overcome by treatment, or is never called into activity.

Epileptic attacks vary greatly in severity. In accordance with the universal practice, we may divide them into two principal groups, the one corresponding to what the French call the *petit mal*, the other to what they call the *haut mal*. These terms have been latinised as *Epilepsia minor* and *Epilepsia major*, and it would be convenient to adopt similar expressions in English. The *petit mal* is sometimes spoken of as *epileptic vertigo*, but we shall see that there is more than one reason why this use of the term is undesirable.†

* *Synonyms.*— Επιληψία or Ἐπίληψις, a seizure (Hippocrates, Aristotle) — Morbus Comitialis (Seneca and Pliny), so called, according to the grammarian Festus, because the Roman assemblies were broken up if anyone present was attacked by a fit—Morbus sacer (Cælius Aurelianus)—Falling Sickness—Haut Mal—Fallsucht.

† "Inter notissimos morbos est etiam is qui *comitialis* vel *major* nominatur. Homo ubitò concidit: ex ore spumæ moventur: deinde interposito tempore ad se redit, et per se

Epilepsia minor.—A paroxysm of the *petit mal*, or minor epilepsy, may be a mere suspension of consciousness, sudden in its onset and scarcely more than momentary in its duration. For two or three seconds the patient is lost, but he quickly recovers himself and goes on with what he is doing. Dr Wilks mentions the case of a shopman who often had a seizure of this kind while serving a customer, and who believed that no one noticed it. In some instances such attacks are attended with muscular rigidity. Dr Chambers relates that a well-known lecturer would sometimes be seized while addressing his class, and would stop in the middle of a sentence, remaining perfectly still, with mouth open and arms extended; after a minute or two he would go on just where he left off without knowing anything that had happened. Dr Chambers, indeed, puts this case as one in which the *petit mal* assumed the characters of *catalepsy*, but it does not seem clear that it really deserved that name. At any rate its close relation to epilepsy was shown by the circumstance that the gentleman in question afterwards suffered from that disease in its ordinary form.

In other cases, if the patient should happen to be speaking at the time when he is attacked, he loses the thread of his discourse, and afterwards he cannot remember what he was saying. Or he may stagger and lean against something, or grasp at it for support. He may experience a sensation of giddiness and lie down to avoid falling, or may fail to prevent himself falling. Dr Wilks mentions one instance in which a little girl would be sitting in her chair stitching, when she would suddenly fall, but before the nurse could pick her up she would be in her seat and again at work. To such cases the name of "epileptic vertigo" would be fairly applicable, if it were not undesirable to separate them from the rest, and if there were not another *paroxysmal vertigo*, which bears no close relation to epilepsy (*v. infra*, p. 812). Moreover, some writers call any nervous affection epileptic which recurs paroxysmally; consequently the expression "epileptic vertigo" is ambiguous.

If we have the opportunity of watching an attack of minor epilepsy, we may notice that the patient's pupils become slightly dilated and that he no longer seems to be looking at anything. The face often becomes pale, and afterwards slightly flushed. Dr Reynolds says that the pulse may falter and become irregular. Some years ago, while I was one day listening with the stethoscope to the heart of a man whom I had never seen before, its beats suddenly ceased. I looked up and saw that his face had turned deadly pale. He said that he was going to faint, and reclined back on the chair from which he had risen. I could feel no pulse at his wrist, and for an instant I thought that he might be going to die, but I had hardly time to ring my bell when the colour returned to his face, and I found that his heart was beating again. Presently there was a little twitching of the muscles in one or both hands. In a minute or two he was able to tell me that he often had "fainting fits," and that some years back he had been subject to epileptic seizures. It appeared clear that the attack which I had witnessed was one of minor epilepsy. In a large majority of cases the so-called fainting fits which are apt to occur in children are really of an epileptic nature.

It is, however, certain that in some instances the circulation in the face and limbs goes on without interruption during an attack of the *petit mal*. Dr Reynolds testifies to this from repeated observations.

ipse consurgit. . . . . Modò cum distentione nervorum (*i. e.* convulsions) prolabitur aliquis, modo sine illâ" (Celsus, 'De Med.,' lib. iii, cap. xxiii).

Not infrequently, an attack which is in all other respects one of "minor epilepsy" is accompanied by some slight convulsive movement. There is a transient strabismus; the mouth is drawn to one side; the whole head is turned towards one shoulder; or the body generally becomes for an instant rigid. Cases of this kind form links between the two main varieties of the disease, and show how closely they are related to one another. A further proof is the fact that in perhaps the majority of cases in which attacks of the *petit mal* occur again and again, they are after a time replaced by those of the *haut mal*. Or the patient may suffer alternately from the one and from the other.

Prodroma.—Before describing the phenomena which constitute a regular attack of the *haut mal*, we must mention certain sensations which some patients experience at the commencement, and which may precede the other symptoms so as to afford a warning of the approach of the seizure. To all such phenomena the name of *epileptic aura* is commonly given by a convenient extension of its original meaning; it was first applied to a feeling, as of a draught of air, passing over the surface until it reached the head, whereupon the patient became insensible. Sometimes a creeping or a dragging sensation is experienced, which begins in the fingers of one hand, or the toes of one foot, and rapidly moves upwards. Dr Bazire relates a case in which it first affected the wrist and then extended downwards to the tips of the fingers, the nails feeling as though they were pulled off at the roots. Dr Radcliffe had a case in which it was a painful sensation, always referred exactly to the foramen cæcum at the base of the tongue. Other cases have been recorded in which an aura has consisted of a painful sensation at the epigastrium, attended with nausea. Or again, there may be loss of sensation in one side of the face, or in one limb. Nothnagel mentions an instance in which numbness always began in the right shoulder and side of the head, and spread downwards into the right arm, the right leg, and the right half of the body. When an aura passes up a limb its path seldom corresponds with the course of any particular nerve. In some rare cases it seems to start from the seat of a former injury. Sir Thomas Watson quotes a case in which it proceeded from an old cicatrix in the side. Dr Wilks alludes to one instance in which it was referred to a painful corn on the toe. He also states that a little girl under his care localised her aura in a sore spot on her face, and that her father assured him that the application of laudanum to that part would sometimes arrest the fit.

It other instances it would seem that an epileptic aura is due to an affection of the vaso-motor nerves of some part of the body. The patient perhaps experiences a sensation of coldness or weight in a limb; and the part is found on examination to be pale and cold to the touch, and to have its sensibility distinctly blunted. Trousseau says that when an aura occurs in a finger it is sometimes a little swollen, so that the rings on it which before were loose suddenly become tight.

In other cases, again, an epileptic attack is preceded by a profuse secretion of tears, or of saliva, or of sweat, as in several cases that have come under Nothnagel's observation.

Or, what has been termed a "motor aura" may occur, in the form of tremor or of slight spasms. The eyelids may twitch, or some muscles of the face or of a limb. Or complex movements may be performed, the patient perhaps turning round, or running some distance. It is said that a

transient paralysis of one limb may take place under the same circumstances.

Again, the epileptic aura may be referred to one of the special senses. Joseph Frank is said to have met with an instance in which it always took the form of a sweet taste. Still odder is Gregory's case of a man who always fancied he saw a little old woman in a red cloak; she seemed to come up to him and to strike him a blow on the head, whereupon he lost all recollection and fell down. Sometimes the only warning is a vague sensation of fear. Dr Reynolds was told by a gentleman that what always passed through his mind was, "This is what I had foreseen. I knew it would come on here; I ought to have avoided it by remaining away,"—although in reality he had not before had any suspicion that a fit was impending.

An epileptic aura may last for a few seconds or some minutes, or even (it is said) for two hours. Nothnagel mentions that when its duration has been more than ten minutes he has been able to ascertain that there was distinct loss of sensation in the part affected by it. Sometimes it occurs and passes away without being followed by a fit. In one of Nothnagel's patients a vaso-motor aura in one leg is said to have come on as many as six or even ten times daily, whereas she had only about one epileptic attack in a week.

Symptoms of the fit.—An attack of the *haut mal* begins by the patient suddenly—almost instantaneously—falling unconscious, with strained and rigid muscles. As he is seized, he may utter a sharp shrill cry; or he may be perfectly silent, the respiratory movements being arrested by spasm; or he may emit a smothered groaning sound. In many cases the face becomes pale; but in some its colour remains unchanged. The heart goes on beating and the carotid arteries continue to throb. But the radial pulses are sometimes imperceptible, this being in all probability due to the contracted state of the muscles of the upper limbs.

The *tonic spasm* which occurs at this period of an epileptic or other kind of fit is peculiar; it usually affects one side of the body more powerfully than the other; the head is turned round towards one shoulder, and the eyeballs are strained in the same direction. The pupils constantly become dilated; but Dr Reynolds says that in one instance he observed a momentary contraction before dilatation began. They are generally, if not always, insensible to light. Touching the conjunctiva commonly excites no reflex movements, but according to Romberg it is sometimes followed by closure of the lids; this observer also states that sprinkling the surface of the body with cold water may in some cases cause shrinking movements. Soon the face flushes, and acquires a dull red or dusky hue. This appears to be in great measure due to the fact that the large veins of the neck undergo compression, and that the flow of blood through them is interrupted by the spasmodic contractions of the sterno-cleido-mastoideus and neighbouring muscles. Spasm of the glottis seems also to occur.

The stage of tonic spasm may last only two or three seconds, or it may be prolonged to thirty or forty seconds. Trousseau says that in some rare cases it extends over two or three minutes, in fact until the patient dies asphyxiated. It is followed by a stage in which *clonic spasms* form the most striking feature. These also are generally more marked on one side than the other. The fingers of one hand are alternately flexed and extended; the like movements simultaneously occur in the other joints of the arm, and

even in the corresponding lower limb, and the eyes are twitched violently towards the affected side. The opposite limbs may escape entirely, or be affected with less powerful convulsive movements. The pupils may now oscillate between a state of contraction and one of dilatation. Dr Reynolds says that they are not always altogether insensible to the influence of light. The jaws are forcibly contracted, and the tongue is often caught between the teeth, and bitten on one side. The blood which comes from it is mixed with a secretion that is poured abundantly into the mouth and air-passages; and a red foam is blown out through the clenched teeth. The face is now always of a livid purple hue. There is often profuse sweating. The urine and fæces are apt to be involuntarily ejected, and emission of semen may occur.

This second stage—of clonic spasm—may last two or three minutes; according to Dr Reynolds, even ten minutes. Trousseau, however, says that it scarcely ever continues longer than four to six minutes; as he remarks, one is very apt to be deceived as to its duration, unless one actually reckons the time by a watch. As the convulsive movements pass off they become slightly altered in character. They are no longer wholly meaningless. The patient often draws a deep sigh; he may endeavour to change his position and may look at those about him with a bewildered, or suspicious, or sorrowful expression. Dr Reynolds makes of this a third stage, which, however, seems to be hardly needed. He says that the pupils are now contracted.

When the fit passes off, the patient may at once regain his consciousness; or he may be more or less confused for a time; or, more frequently, he becomes drowsy and passes into a deep sleep or stupor, which may last several hours, and is often attended with guttural stertor of the breathing. During this time, if he can be roused at all, he is generally very irritable and peevish. Slight clonic spasms not infrequently recur.

Exceptional forms.—An attack of major epilepsy, however, does not always conform strictly to the above description. It is sometimes attended with but very slight convulsive movements, or perhaps with none at all. Such cases were formerly described as examples of a form of apoplexy, or as "apoplectiform cerebral congestion" (cf. *supra*, p. 597). Even when an epileptic fit occurs during sleep, one can often make out its real nature by the fact that the tongue next morning is found to be sore, or that the urine has been passed involuntarily, or even the fæces. As Trousseau points out, if a patient who had never before had difficulty in retaining the contents of his bladder at night, should now and again find that he has wetted his bed, this mere fact should arouse the fear that he may be an epileptic. Dislocation of the shoulder, discovered on waking in the morning, may signify an epileptic fit during sleep. A similar inference may sometimes be drawn from the presence of minute scattered red petechial spots, like fleabites, which occur chiefly on the forehead, throat, and chest. These may frequently be observed after an ordinary fit of the *haut mal;* and sometimes they follow a masked attack. A point which may be mentioned incidentally is that when petechial hæmorrhages into the skin occur after an epileptic attack in a person who has not before been subject to fits they may be wrongly regarded as an evidence that the patient is suffering under blood-poisoning or that an exanthem is about to appear. Dr Wilks has related a case in which this mistake occurred.

We have seen (*vide supra*, p. 612) that tumours and other local organic

diseases of the brain frequently give rise to fits which are unattended by loss of consciousness, and consist mainly of convulsive movements, perhaps limited to certain parts. These have been very properly distinguished as Jacksonian convulsions, after Dr Hughlings Jackson. It might be expected that similar fits would sometimes occur in genuine epilepsy, either alternating with ordinary attacks of the *haut mal* or preceding them, as is so commonly the case with those of the *petit mal*. All observers, however, are agreed that this is exceedingly rare. Two instances are recorded, the one by Trousseau, the other by Nothnagel. The former occurred in a young man aged eighteen, who was liable to convulsions of the facial muscles, affecting only the left side, and not accompanied by loss of consciousness nor by any other epileptic phenomena. But the clinical history was that the disease had first set in six years previously with violent fits of the *haut mal*, and that these had gradually become milder and passed into those of the *petit mal*, which presumably occurred alternately with the attacks of mere partial clonic spasm. Nothnagel's case was in a boy, aged sixteen. Dr Reynolds, who gives to this form of the disease the very appropriate name of "abortive epilepsy," furnishes a list of references to various writers as having described it. After looking up most of them, it does not appear that the writers in question took care to exclude cases of cerebral tumour and the like. However this may be, exceptions are very rare to the rule, that there is local organic disease of the brain, whenever attacks of clonic spasm recur paroxysmally without loss of consciousness.

Recurrence.—The frequency with which the attacks of epilepsy return varies greatly in different cases. A patient may have one fit without ever having a second; and yet it is possible that it was epileptic in the strictest sense of the word. Or after an interval of two or three years he may be again attacked. In some cases the paroxysms return once, or twice, or three times a year; in other cases more or less regularly once a month. In women they are, on the whole, more apt to occur at the catamenial periods than at other times. But Dr Reynolds says that monthly recurrence is actually more common in the male than in the female sex; and it is very rare indeed for a woman to be liable to fits of an epileptic character only while menstruation is in progress. More frequently the interval at which the attacks recur is less than a month. According to Dr Reynolds, in half the cases that he analysed the average period of return was between fourteen and thirty days. Lastly, there may be one paroxysm, or even more than one every day. A common thing is for two, three, or more fits to occur on the same day or within two or three days, and then for the patient to be free from them for several weeks. They are then sometimes said to recur in *series*. When they return with very great frequency during a lengthened period they almost always belong to the minor form of the disease. For attacks of major epilepsy, if they repeat themselves several times a day, must, with the stupor that follows them, take up a considerable part of the twenty-four hours, and this necessarily impairs the patient's health, so that it cannot go on indefinitely.

There is, however, a special modification of the disease in which the fits follow one another in rapid succession, so that before the patient has recovered completely from one of them another comes upon him. This has by modern French physicians been called the *état de mal épileptique*, and in England some writers have made use of the equivalent expression, *status epilepticus*. When it is at its height the convulsions follow one another with

extraordinary rapidity. Dr Crichton Browne in describing such cases says that the limbs are scarcely laid to rest after one fit before they are tossed and contorted by another, and even in the intervals there are frequent muscular twitchings. The patient lies perfectly unconscious; his heart beats rapidly and tumultuously; his respiration may either be quick and shallow or slow and laboured. His features are swollen and livid, and his lips purple. His body is bathed in profuse perspiration, and the temperature is raised to 105°, or even higher still. Dr Merson found it in one case 107·8°. This condition often terminates fatally; indeed, it constitutes one of the principal ways in which epileptic patients die. Charcot observes that in such cases bedsores over the sacrum develop themselves rapidly. He also mentions that a temperature as high as 105·8° is sometimes reached without death ensuing.

Complications.—Certain occasional results of an epileptic fit have yet to be described. One is a loss of muscular power in one arm, or in both the arm and leg on one side, generally that side which was the more convulsed. Dr Todd described this under the name of "epileptic hemiplegia;" it may either pass off in a few hours or last several days. One must not forget that diminished mobility of the arm after a fit is sometimes due to a very different cause, namely, to dislocation of the shoulder, produced sometimes by muscular spasm, sometimes by direct injury, the patient having struck that part in falling. I myself once nearly overlooked this accident. A patient came with her arm hanging helpless. I was pointing out to the students that a transient paralysis often follows an epileptic attack, when I happened to notice that touching the limb gave pain. I grasped the deltoid muscle, and found that the head of the humerus was out of place. At one time a woman who was liable to epileptic fits used frequently to come to the hospital to have her shoulder set. In her case the fact that the same dislocation occurred again and again showed that spasmodic contraction of the muscles was its cause. Still more serious injuries sometimes arise during a paroxysm. The skull may be fractured by the patient dropping down on the pavement or on a stone floor; or he may be severely burnt if he should happen to fall against the bars of the grate; or he may in a crowded thoroughfare be run over. Persons who are liable to epilepsy cannot be too closely looked after.

Delirium has not hitherto been mentioned as accompanying epileptic fits, but in some cases it is one of the most important features. Indeed, in a person subject to the disease, the attack may be wholly replaced by one of paroxysmal insanity, such as will be described in the next chapter. Or, again, the epileptic character may be so far maintained that the patient falls down, but quickly gets up again and attacks those about him with the utmost violence. An instance of this is recorded by Trousseau, who remarks that if there had been no witnesses of the commencement of the attack, the patient would have been the subject of a criminal prosecution, since he not only assaulted the passers-by in the street, but insulted the soldiers who held him and spat in their faces. In other cases the epileptic stupor is succeeded by maniacal delirium, during which suicide may be committed, or murder. Still more frequently a fit is followed by loss of memory, incoherence of ideas, and perversion of intellect, which may last several days. It is not surprising therefore that when the paroxysms recur very frequently and at short intervals, the mental powers should become permanently impaired. Patients who have been subject to the disease for some time commonly

acquire a peculiar dull heavy aspect, and this, with the circumstance that they almost always have widely dilated pupils, often enables one to tell at the first glance what is the matter with them. They are apt to be exceedingly irritable in temper, or morose, gloomy, and desponding. Sometimes, however, the mental state is worse immediately before the epileptic attacks than at any other time; when a fit occurs it seems to afford temporary relief to the brain, and the patient afterwards feels lighter and more cheerful than for a long period before.

Impairment of the intellect is by no means confined to patients who have already suffered for a long time from epilepsy. In children it often happens that a series of fits, continued for a few successive hours, produces a permanent state of imbecility, or of mania. A considerable proportion of those who are admitted into asylums for idiots are children who were in reality born with full powers of intelligence, and learned to talk as soon as others; but, having been attacked by epilepsy when perhaps four or five years old, they have since lost sense and intellect, have become dirty in their habits, passionate or violent in temper, and unable to recognise their parents. Such cases are frequently brought to the out-patient departments of the London hospitals.

In adults, on the other hand, permanent impairment of intelligence occurs only when the disease has been of some standing. Indeed, it is indisputable that some individuals retain their full vigour of mind after having been liable to fits for years. Julius Cæsar, Mahomet, Peter the Great, and the first Napoleon are commonly referred to as illustrations of the fact that the repeated occurrence of epileptic attacks does not necessarily injure the intellectual powers, but in each of these cases the fits were only occasional, and the evidence of their nature is far from being complete. Dr Reynolds combats what he terms the "prevalent belief" that mental deterioration is necessarily associated with epilepsy, and he states that in rather more than one third of all the cases which he examined there was average intellectual power; but as his investigations of course apply only to the state of the several patients when he saw them, one cannot infer from them that the intelligence remained perfect during the whole of the patients' lives. And the records of every lunatic asylum afford abundant illustrations that dementia ultimately shows itself in many persons who had been epileptic for a long time before the mind gave way. According to Dr Reynolds the later the age at which the fits commence the greater the probability that the intellect will suffer. The severity of the individual attacks has less influence in bringing about such a result than the frequency of their occurrence. Esquirol long ago pointed out that dementia more often occurs in persons who are liable to the *petit mal* than in those who have attacks of the *haut mal*.

Pathology.—With regard to the pathology of epilepsy much uncertainty of opinion still prevails. So soon as an attempt was made to distinguish the functions of different parts of the nervous centres, it was suggested that in a fit there was a torpor of the brain, associated with excitement of the spinal cord. But the regular order in which the phenomena of the paroxysms succeed one another is evidently inexplicable under such a simple hypothesis. And with the advance of physiology the theory arose that the disease might have its seat in some particular part of the brain, which would be the starting-point of the attacks, and which might fairly be termed the "epileptic centre." Sir William Gull used to speak of epilepsy as a "function;" and by this he meant that the orderly development of the various

symptoms which constitute the seizure must depend upon structural nervous arrangements, like those involved in the more complicated physiological actions. Von der Kolk suggested that the medulla oblongata was the seat of epilepsy, and recent writers, including Reynolds and Nothnagel, have expressed a similar opinion, only including within the area of disturbance more or less of the pons Varolii, or of the cervical part of the spinal cord.

We must, however, either confine the supposed "epileptic centre" within very narrow limits or else include in it the entire length of the spinal cord. We cannot imagine that spasmodic movements of the eyes or of the face bear to such a centre any closer relation than do those of the upper or even of the lower limbs. And if we once give up the idea of fixing the seat of the disease in a definite spot within the medulla oblongata, a little consideration will lead us to include in the affected area the mass of the cerebral hemispheres. There is no other way in which we can so simply explain the facts that consciousness is suspended and that delirium and excitement are often present. Dr Wilks may be quoted as one writer who has always strongly expressed the opinion that epilepsy is an affection of the whole of the brain.

The views entertained by Dr Hughlings Jackson are widely different. This observer has studied with the greatest care those epileptiform and other convulsions which are caused by local diseases, such as tumours of the surface of the brain. He has laid great stress on the fact that whereas a "destroying lesion," affecting a particular convolution, is capable of causing paralysis, a "discharging lesion" of the same part gives rise to convulsions, which may implicate the opposite face, arm, and leg in a definite order. His way of accounting for this is to suppose that in particular convolutions movements are "represented," which involve the action of many different muscles (cf. *supra*, pp. 624, 626).

In 1873 Dr Ferrier performed a series of experiments on the lower animals with the express object of throwing light on Dr Jackson's theories of epilepsy. Fritsch and Hitzig had shown in 1870 that the surface of the brain is sensible to the galvanic current, and they had laid down the seat of cortical centres for the muscles of the neck, of the face, and of the upper and the lower limbs, and the recent experiments of Schäffer and Horsley have put most of the physiological facts on a solid basis (*v. supra*, pp. 622, *et seq.*).

Apart from these experimental results, it would seem, from clinical observations, that when convulsions are set up by local disease of the surface of the brain, the fact that certain parts are especially implicated in the spasms may sometimes enable us to indicate the seat of the lesion (*v. supra*, p. 625). But it is quite another question whether any similar conclusion is warranted, when there is no other evidence of local disease of the brain.

It must also be borne in mind that the spasmodic movements produced by disease—such as are observed in a convulsive attack of whatever kind—are very different in character from the slow and orderly actions to which galvanic stimulation of the brain gave rise in those of Dr Ferrier's experiments, from which alone conclusions as to the functions of particular convolutions could be drawn. It is true that spasmodic contractions, and even complete epileptiform fits, were often observed; but these received further explanation, as will presently appear. The cases to which reference is now made are those in which we read, for example, of a cat raising the shoulder and adducting the forepaw, as if to strike; of a rabbit munching with its lips and jaws; and the like. In an epileptic fit the affected part is

violently jerked backwards and forwards, in a way altogether different from what occurs in its natural movements; and surely this suggests that the convulsive movements do not depend merely upon "discharge" of the cortica centres, but are the results of impressions transmitted downwards from them to the corpus striatum, or some lower plane of the cerebro-spinal centres.

The case is far stronger when, instead of localised spasms, a complete epileptic paroxysm occurs, in which all parts of the body are convulsed, and consciousness is for the time suspended.

Dr Ferrier has discussed this question very fully in his treatise on 'The Functions of the Brain.' His theory is that in the cortex of the brain there are individual centres for each separate muscular action involved in the epileptic convulsion, that they are related to each other in a constant and definite order, and that the attack is due to the discharge of these centres in a tolerably uniform manner. Much, he says, depends on the primary source of the irritation, but adds that from whatever part of the hemisphere this proceeds, whether from a motor centre, or from the sensory areas towards the back of the brain, the order most commonly observed is that the centres discharge from before backwards, beginning with those of the head and eyes, and ending with those of the leg. He goes on to say that epileptic convulsions can be produced with as great readiness by application of the irritation to the sensory areas as to the motor centres themselves; and, again, that it is doubtful whether consciousness becomes lost when the motor centres of the brain are alone implicated.

These statements of Dr Ferrier seem to deprive Dr Jackson's views, in their most special form, of the support gained from experiment. But Dr Ferrier's own hypothesis appears less probable than the older opinion that, even when irritation of the surface of the brain causes an epileptic fit, the actual motor impulses start from the basal ganglia or from centres still lower, in the pons, the bulb, or the cord. Surely, the orderly development of the phenomena of the paroxysms is more easily explained in this way.

Again, it is well known that there are cases in which epileptic fits are set up by irritation of spinal nerves, by diseased teeth, or (as in Brown-Séquard's experiments) by pulling the hairs on the face of a guinea-pig, of which the spinal cord has previously been injured. All such cases are obviously much more easily explained on the view that the parts which are the seat of "discharge" in the attacks are the lower centres, than on the theory that they are the highest centres of all, situated in the cerebral cortex.

A further argument on the same side may be found in the analogy of another paroxysmal neurosis. A tumour in the brain, at a distance from what one can suppose to be the seat of migraine, may excite repeated attacks of that malady. One certainly cannot imagine that these are due to "discharge" of the part which is immediately affected by the tumour; they must be due to an influence transmitted downwards to the thalamus or to other sensory ganglia.

At the present time a view is widely prevalent, according to which all but the initial phenomena of an epileptic attack result from vaso-motor disturbance. It is supposed that the cerebral arteries undergo spasmodic contraction, and that the consequent anæmia of the brain causes the patient to fall down insensible. It is well known that the face commonly turns pale at the commencement of a seizure. There is ophthalmoscopic evidence that the retina, which derives its blood supply directly from the internal carotid artery, also becomes anæmic. Dr Jackson and Dr Charles Aldridge have

proved that the optic disc is pale or even white, and that the arteries which traverse it are much diminished in size. It is to be noted, however, that on the single occasion on which the ophthalmoscope has been used before the clonic convulsions ceased, the disc was at first pink, so as to be undistinguishable from the surrounding choroid; and that it only afterwards became white, and then slowly.

But the most substantial support of the notion that the brain is essentially anæmic during an attack of epilepsy has been afforded by the well-known essay of Kussmaul and Tenner. It had before been known that both in animals and in man loss of blood was followed by convulsions. These observers showed that the like result could be brought about by ligature or compression of the four great arteries supplying the encephalon. In reality, however, their experiments only proved that a deficient supply of blood to the brain might be one cause of attacks of an epileptiform kind. They themselves pointed out that ligature of the trachea, rendering the arterial blood rapidly venous, had the same effect; and in either case they referred the convulsions to sudden interruption of the nutrition of the brain. Their theory evidently does not in any way confirm the more modern opinion that when an epileptic fit has commenced in the medulla oblongata, the patient becomes unconscious as the result of some reflected influence upon the cerebral arteries, rendering the brain anæmic.

For after all, the feature of epilepsy most difficult of explanation is the tendency of the fits to recur at more or less definite intervals. And no way of accounting for this seems to be so satisfactory as that which refers it to a gradual accumulation of energy in the nervous centres, which is dissipated during the attacks. As van der Kolk points out, it often happens that after a severe fit an epileptic patient remains free longer than usual; but if he should have only a slight attack, he soon afterwards has another, perhaps on the following day. Again, in some cases each fit is preceded by a gradually increasing irritability of temper and restlessness, which disappear or are notably diminished after it has taken place. Other patients, as Trousseau remarks, become gay, loquacious, and excited for some hours before an attack; and yet others complain of failure of memory, of torpor, and of physical and mental prostration. Nothnagel mentions the case of a lady, generally a light sleeper, who always knew that she was about to have a paroxysm, when she happened to sleep more heavily and longer than usual; nevertheless she would wake up feeling quite well, and would not be attacked until later on in the day.

We are thus brought in regard to epilepsy to the same point which we reached in discussing the nature of migraine: that it is essentially a paroxysmal neurosis, recurring at more or less regular intervals. And, as in the case of migraine, in all probability the alterations in the blood supply to the brain, which undoubtedly occur during the epileptic attack, are accidental concomitants rather than essential to the development of any of its symptoms. This view is not inconsistent with the fact that the inhalation of nitrite of amyl is sometimes of service in the treatment of the disease; for when there is an aura the attack can sometimes be overcome by a decided impression on the part to which the aura is referred, and the inhalation may fairly be supposed to act in a similar way, and to cut short the paroxysm by arresting one of its phenomena. Moreover, venesection is sometimes useful, apparently under the same circumstances as those in which the nitrite does good. Yet they must produce contrary effects.

To sum up, then, we may perhaps at present be content to adopt the language of some modern writers, and say that epilepsy is "dependent upon an unstable condition of the nerve-tissue in some portion of the nervous system, permitting occasional discharges." This, in reality, is not stating more than that the disease is a "nerve-storm." Just as in migraine, teichopsia may be followed in succession by numbness in the fingers, by headache, by vomiting, by sleep, so in epilepsy tonic spasms give place to clonic convulsions, and these, again, to stupor or coma. It appears more satisfactory to refer this sequence of phenomena to the gradual extension of some morbid condition from one part of the nervous centres to another, than to ascribe it to modifications in the blood supply.

This account of the pathology of epilepsy can hardly be regarded as complete without some reference to the remarkable experiments of Brown-Séquard, who found that in guinea-pigs and some other animals section of the spinal cord, or even of one or both sciatic nerves, was followed after some weeks by well-marked epileptiform fits, which returned again and again, and could at any time be excited by slight irritation of the cheek (as, for instance, by gently pulling the whisker) or of the side of the neck. The exact bearing of these interesting observations, however, seems even now to be doubtful, so far as concerns their applicability to the disease with which we are acquainted in the human subject. They do not afford any support to the idea that the epileptic aura really starts from a distal part of the body and travels along sensory nerves, setting up the paroxysm when it reaches the encephalon. There is every reason to believe that the aura is itself part of the attack, and due to a change in some region of the brain which has close connections with the roots of certain cutaneous nerves; the disturbance is accordingly referred to the peripheral distribution of these nerves.

Ætiology.—In passing on to consider the causes of epilepsy, we have in the first place to consider *hereditary predisposition.* Dr Reynolds made inquiries which led him to the conclusion that in 12 per cent. of his epileptic patients the same disease had occurred in other members of their families. But this was not all. For in a much larger number of cases, other affections of the nervous system could be traced in persons derived from the same stock as the patient. Sometimes it was one of the other paroxysmal neuroses, such as migraine; sometimes insanity, hysteria, hypochondriasis, or mere nervousness. Thus in a family in whom nervous disorders prevail, one child may be epileptic, another insane or idiotic, another hysterical, and so on. Dr Reynolds says that in nearly 30 per cent. of his cases of epilepsy he obtained a history of some nervous disease having occurred in at least one near relative. Drunken habits in parents are also believed to be often concerned in inducing epilepsy in their offspring; but then the inclination to intemperance is often itself a sign of the neurotic tendency. Some writers have supposed that a disposition to tubercle, that rickets, or that general malnutrition may be concerned in the causation of epilepsy; but this is rendered very doubtful by the exceeding prevalence of such conditions. The intermarriage of blood relations does not seem to have any tendency to cause this disease in the offspring, provided that neither parent is predisposed to the disease.

As to predisposition of *sex* and *age*, epilepsy appears to affect males and females in about equal proportions, notwithstanding the remark of Celsus, "*sæpius viros quam fœminas occupat.*" See some sagacious remarks of Dr

Reynolds ('System,' vol. ii, p. 295) on the fallacies of statistics in this respect.

The *time of life* at which it is most apt to begin is between the tenth and the twentieth years. Dr Reynolds found that in 106 out of 172 cases the first fit occurred between these limits of age, and in by far the larger number it was within the still narrower period of from thirteen to seventeen years. The more marked the inherited predisposition, the earlier is the average age at which the disease develops itself, and it appears in girls sooner than in boys. Most of these facts seem to point clearly to the conclusion that the development and commencing activity of the sexual organs are in some way concerned in the causation of the disease. It has been said that in women the time at which the menses cease to appear is again apt to be attended with the development of epilepsy, but Nothnagel says that this is not the case. However, according to Dr Reynolds, the period between twenty-five and thirty-five years of age is one at which there is a comparative immunity from first attacks; and they often occur in persons about forty. Exceptional instances are recorded in which epilepsy has begun at an advanced age; one, for example, by Trousseau, which began at about sixty-nine.

In other cases, instead of the predisposing cause of epilepsy being an inherited tendency, it is an acquired condition.

Prolonged anxiety of mind, grief and destitution have been supposed to lead to it. Habits of *intemperance*, *sexual excesses*, and the practice of masturbation are believed to be more directly concerned in bringing it about. Indeed, both a first fit and the succeeding ones have in some individuals occurred only when they were actually intoxicated, and in others only during coitus. With regard to masturbation, all modern writers speak very guardedly, so far as concerns the origin of epilepsy, although there is no doubt that this degrading habit has a most depressing effect upon the health of immature youths, and is capable of causing a variety of nervous symptoms, some of them of a serious kind.*

We have seen that in a large proportion of cases of epilepsy no definite predisposing cause can be discovered. And even where there is a marked inherited tendency our knowledge of other hereditary complaints would lead us to expect that in the majority of cases the first attack, and perhaps even the subsequent ones, would be immediately traceable to some definite *exciting cause*. Experience, however, hardly confirms such an anticipation. There is, indeed, much difficulty in eliciting the real facts in many instances. On the one hand, the parents of children affected with epilepsy are much disposed to conceal a family tendency to that or any other serious neurosis; and patients themselves are apt to be unacquainted with the real state of health of relatives older than themselves. And, on the other hand, there is a powerful tendency to attribute the disease to any accidental circumstance which can by possibility be brought into relation with the first attack. Thus, Trousseau says that when a fright was assigned as the cause of epilepsy he often found on inquiry that it had really occurred months or even years before the fits began, or that it was trifling in character. He himself, however, relates a case in which it appeared clear that the original cause of

* I do not believe in the existence of any unfelt irritation starting from the genital organs of modest young women, and giving rise to epilepsy or any similar disease. And I regard as an abomination the operation of clitoridectomy, which was some years ago practised upon a theory of that kind.

epilepsy was the terror caused by the sight of a quarrel between two men, one of whom was wounded and fell down dead. In that instance the first attack (of the *petit mal*) occurred within a few days, and subsequently the *haut mal* developed itself.

According to Dr Reynolds, it is only in one out of every eight cases of epilepsy that the inquiry as to the ætiology of the disease fails altogether, by neither any predisposing nor any exciting cause being discovered. And since an inherited tendency (which is the great predisposing cause) could be traced in but one third of the cases there must evidently be a large number in which a supposed exciting cause, such as fright, is the only one that can be detected. We have then a very important point to determine, namely, why the first fit should be followed by others. For, it is to be observed that in some cases the disease has been consequent upon attacks which are commonly regarded as purely accidental in their origin. Thus, Dr Hughlings Jackson mentions a case in which a convulsive fit at the onset of scarlet fever proved to be the forerunner of habitual epilepsy.

Again, it is said that the simulation of the disease by impostors has ended in their becoming really subject to it; and, if true, this is a still stronger fact in the same direction. Brown-Séquard found that the guinea-pigs in which he artificially set up epilepsy transmitted it to their offspring.

On the other hand, some very remarkable cases have occurred which show that even when epileptiform fits have occurred at intervals for a very long period, as the result of irritation of the nervous centres by some cause acting on a distant part, the liability to their recurrence may cease entirely when the cause in question is removed.

Thus, Mr Tomes relates the case of a farm-labourer suffering from epilepsy whose mouth was examined and the molar teeth of the lower jaw were found to be decayed, the fangs of some of them alone remaining. Although he had been treated for the fits during six weeks and complained of no pain, these teeth were removed, and were found to be enlarged from exostosis. During the eighteen months that followed he had not a single fit, although for many weeks before the operation he had had two or three daily. Another case, recorded by Dr Ramskill, is that of a boy who for eighteen months had had epileptic fits, and in whom it was noticed that before the fits he used to rub his left cheek on account of an indefinite uneasiness, not amounting to pain. On examination a molar tooth considerably decayed was found; this was removed, and from that time the boy did not have another fit, although he remained under observation for four months. It is to be said, however, that during that period belladonna was administered. But the most extraordinary case of all is one, related by Trousseau, of a young clerk, who for several years had been subject to monthly attacks of epilepsy; remedies had been tried in vain at the Hôtel Dieu, when Dr Foville suggested the extraction of some carious teeth which ached constantly. The suggestion was acted on, and from that day the fits disappeared.

Trousseau also relates the case of a man, aged forty, who on several occasions, at very short intervals, was seized with violent epileptic attacks. Dr Monnier found that he had been passing fragments of *tænia*, and gave him large doses of castor-oil; a whole tapeworm came away, and from that time the convulsive fits ceased.

Again, an *injury* to the head may be the starting-point of habitual epilepsy. Nothnagel gives the case of a boy who when eight years old fell

from a height of twelve feet upon his head upon a hard floor. He was stunned for a quarter of an hour, and ten minutes after recovering consciousness he had a characteristic epileptiform fit. There was a slight scalp wound which healed in a few days. After six weeks he had a second attack, and from that time they recurred at periods which became shorter until he had them at intervals of from four to twelve days. He was twenty-one years old at the time when Nothnagel wrote, and his intellect and memory were already somewhat impaired. A slight scar remained, but this was not painful nor adherent. Nothnagel seems not to have thought that surgical interference would have done any good; and perhaps he was right. But in the 'Lancet' for 1873 two cases will be found recorded, in each of which a piece of the skull was removed by the trephine on account of epileptic fits following an injury to the head. Both of them occurred at Guy's Hospital, the one under Mr Cooper Forster, the other under Mr Bryant. The former patient had had a blow on the head four months before his admission; it left a slight swelling, from which a little pus exuded when it was incised. His first fit occurred the day before he came into the hospital. But a week later he was having four or five fits every hour, and his temperature was 103°. The operation was then performed, and the piece of bone which was removed was very dense, three eighths of an inch thick in one place, and rough on the outer surface. A fortnight afterwards he could walk the length of the ward, and he never had another fit—at least until the time when the report of the case ceases, which was two months from the date of the trephining. In the other case the accident had occurred five years before, and attacks of the *petit mal* had occurred at intervals of about a week during the whole period. The cicatrix was still tender, and occasionally painful. Medicinal treatment having been tried without any good result, the trephine was applied, and a piece of bone, which was much thickened, removed. The fits at once became less frequent and seemed to occur only when he was depressed from want of food in his miserable home, or when he was exhausted by diarrhœa. The very same medicines which he had before taken with no benefit were resumed. After a time he became able to earn his living, and when the case was reported (sixteen months later) a confident hope was expressed that he was permanently cured.

Relation to other convulsive fits.—One point in favour of a real distinction between true epilepsy and symptomatic eclampsia is the observation of Dr Tyler Smith that puerperal convulsions are by no means of very frequent occurrence in women who are habitually subject to epileptic fits. But it is clear from the foregoing paragraphs that there may in practice be great difficulty in applying the distinction in question. And another instance of the same difficulty is afforded by infantile convulsions. On the one hand, it is said that persons subject to epilepsy in adult life are often found on inquiry to have had fits in early childhood. Nothnagel expressly states that this is often the case with children born of epileptic parents, and with those who in later years themselves become epileptic. But, as Dr Jackson points out, there are at present no facts to show what is the proportion of those who having had infantile convulsions afterwards escape epilepsy. It can hardly be doubted that they form the immense majority. The fits of infants were formerly attributed to irritation of the nervous centres from teething or disorder of the alimentary canal. But the more closely such supposed causes are inquired into the less clear does their relation to the convulsive attacks appear to be. The tendency of modern observation

is rather to associate infantile convulsions, as we associate laryngismus stridulus, with rickets. And whatever part in their causation one may suppose to be taken by external sources of irritation, it is certain that another very important part is taken by inherited or acquired conditions of the nervous centres, disposing them to convulsive discharge. It may well be that in infancy the brain, being imperfectly organised, yields to influences which in after-life it successfully resists. Certainly it would be a serious error to say that the occurrence of fits in childhood involved danger of epilepsy in adult life. And yet it seems clear that no absolute line of distinction between them can be drawn; nor, in general, between habitually recurrent epilepsy and the various forms of eclampsia, or of epileptiform fits due to external irritation.

The difficulty is in a great measure avoided if we regard all these affections as members of the same group of paroxysmal neuroses.

Anatomy.—The only morbid changes which are to be found in the brain in epileptic patients must be regarded as *effects*, and not as *causes* of the fits. Perhaps the most important are dilatations of the capillary blood-vessels in the medulla oblongata. These were first described by van der Kolk, who maintained that there was a definite relation between their seat and the symptoms observed in the paroxysms; the nucleus of the hypoglossal nerve and the olive (*corpus olivare*) presenting dilated vessels when the tongue had habitually been bitten; whereas in cases in which the tongue escaped he found them chiefly in the nucleus of the vagus. He also described an albuminous exudation into the bulb, causing at first induration of its substance, but afterwards fatty degeneration and softening.

The skull is often exceedingly thick and dense in those who have long suffered from epilepsy, the membranes may be opaque and the brain generally indurated; but these changes seem to bear a closer relation to the impairment of intellect which is so generally present in cases of long-standing than to the primary paroxysmal attacks.

Diagnosis.—This involves several different questions, some of which have already been dealt with, or will be considered in other chapters. For example, one may have to distinguish an attack of minor epilepsy from one of *cardiac syncope*, and an attack of major epilepsy from one of *hysteria*.

But when a person is in a fit, which, if really epileptic, would belong to the major form, we have often first to ask whether it is possible that he is *malingering*. Now, an impostor is likely to choose for his fits a frequented spot, where he is sure to be seen. When he throws himself upon the ground it is in such a way as not to hurt himself; he may even put out his hands to break his fall. He probably overacts his part, crying out many times instead of only once, throwing his limbs violently about, and making the attack last much longer than a genuine paroxysm of epilepsy. There is, of course, no pallor of his face at the commencement; his skin, instead of being cold, is warm and covered with sweat; his pulse, even if quickened, does not become irregular; his pupils are not dilated, still less are they insensible to light. He does not keep his eyes wide open, nor are the eyeballs distorted; generally he only separates the lids a little from time to time, so as to watch the bystanders. If an attempt should be made to raise the upper eyelid he resists it, and instead of his conjunctiva being insensible, the slightest contact excites forcible contraction of the orbicularis muscle. On the other hand, he may readily allow the thumbs to be drawn away from the palms, and afterwards close them again. But according to Dr Marc, in

a person really attacked by epilepsy the thumbs require force to loose them; yet, when once extended, they remain so until the end of the attack, or at least until a fresh convulsion begins. Again, in a simulated fit the tongue is seldom or never bitten; there is no foam about the mouth, unless indeed this is imitated by a piece of soap in the cheek. The muscular strength of a malingerer is not increased. If a little snuff be blown into his nostrils he cannot help sneezing; in a real fit no such effect is produced. Another test is the absence of sensibility to pain; a test commonly adopted by policemen and others is to press the thumbnail forcibly beneath that of the person supposed to be in a fit. As already mentioned, there is reason to believe that repeated attempts to feign epilepsy have sometimes ended in the development of a genuine attack.

An educated medical man would naturally succeed in imitating a fit better than one who has no professional knowledge. Thus Trousseau relates that Esquirol, who did not believe that an attack could be simulated so as to deceive, was once talking this very matter over with him and with Calmeil at the asylum at Charenton, when the latter fell on the floor in violent convulsions. Esquirol examined him for a moment and then said, "Poor fellow, he is epileptic!" upon which Calmeil got up and asked him whether he still retained his opinion.

But assuming our patients' attacks to be genuine, one has next to consider whether they are really epileptic or epileptiform. This question is often settled by the clinical history; the case may, perhaps, be of many years' standing, and the fits may have gradually developed from the minor into the major form. But one must bear in mind that attacks may recur at intervals for a great length of time, and present all the characters of true epilepsy, and yet not be symptomatic. Trousseau's case has been already mentioned, of the clerk who had been liable to epileptiform seizures, for several years, at monthly intervals, and in whom they ceased when some carious teeth were removed. The same writer relates the case of a lady, aged seventy-one, who for thirty-one years had been subject to attacks recurring with daily increasing frequency, so that she at length had as many as twenty-one in the twenty-four hours. Her forehead and nose presented characteristic signs of former syphilitic disease; and the administration of mercury and iodide of potassium greatly checked the fits.

Generally, however, the question of syphilis need hardly be entertained unless the fits are comparatively of recent origin; and the same may be said of Bright's disease, lead-poisoning, chronic alcoholism, and the various organic affections of the nervous centres. The diagnosis between these diseases and true epilepsy must depend mainly upon the presence or absence of the other symptoms which severally characterise them, and upon the condition of the patient between the attacks.

Treatment.—In the treatment of epilepsy two things have to be considered: 1, the management of the attacks themselves; and 2, that of the intervening periods, with a view to prevent their recurrence.

So far as concerns *the paroxysm* the most important point is to ascertain whether the patient has any warning of its approach. If there should be a distinct aura, starting, perhaps, from the hand or foot, one can often, by compressing the limb above, arrest the fit after it has commenced. Some years ago there was, in the Evelina Hospital, a girl who constantly wore round her wrist a piece of cord; this was pulled tight as soon as she felt the sensation which indicated that she was about to have an attack, and

not a single one developed itself during several weeks ; after a time bromide of potassium was administered, and the aura then ceased to recur. Many instances of a similar kind have been placed on record by different observers. Dr Bazire mentions the case of a woman whose fits were always preceded by spasmodic closure of the left hand ; by forcibly extending the fingers, and keeping them open, an impending attack could be warded off. A patient of Dr Reynolds had jerking of the left leg, which was drawn up behind him, when his attack began ; it was arrested by extension of the muscles. Another plan which has been recommended consists in cauterizing the surface from which the aura proceeds.

Even when an epileptic fit is not ushered in by any symptoms beyond pallor of the countenance and tonic spasms, it would appear that the prompt inhalation of nitrite of amyl is sometimes capable of arresting it. Dr Crichton Browne has related some instances of this. One is that of a man who started up suddenly in bed, with his eyes fixed and his head turned to one side ; these symptoms were known to indicate the approach of an attack ; but the nitrite was administered, and the patient at once fell back on his pillow in a half-fainting state, but without the slightest agitation of the muscles. In another case a fit had actually begun in the ordinary way with rigid stretching of the hands by the side, and turning up of the eyeballs, when the nitrite was held to the mouth and nostrils, and in twenty seconds complete recovery had taken place.

Even when an epileptic attack has fully developed itself, there is evidence that it may sometimes be cut short by compression of the carotid artery in the neck. This procedure must be supposed to diminish to some extent the blood supply to the brain. It was first suggested by Dr Parry, of Bath, towards the end of the last century. He relates a case in which it proved successful. A man who had been liable to epilepsy for two years was one day beginning to have a fit : his eyes were assuming a vacant stare, and convulsions were beginning about his throat, when Dr Parry made strong pressure over the right carotid artery ; upon this the convulsions ceased, and the attack proceeded no further. He instructed the patient how to compress the vessel, and the latter afterwards assured him that when he had sufficient warning he was often able to prevent the epileptic paroxysms. At Guy's Hospital this practice was often adopted by the late Mr Stocker, and sometimes with striking results. But it was most useful in cases with a strong hysterical element. The plan which Mr Stocker used to adopt was to press both thumbs into the neck, one on each side, towards the spine ; in doing so he doubtless compressed many other parts beside the carotid arteries, and the pain caused may well be supposed to have been concerned in the rapid restoration of the patient to consciousness, at least when the case was hysterical.

When one is called to a patient in an epileptic seizure, one must see that his clothes are loosened, especially about his neck, and one must take care to prevent his injuring himself. We may endeavour to keep the tongue from being bitten by putting a piece of india rubber between the teeth, but this involves the risk of its falling back into the throat and causing suffocation.*

In the *status epilepticus* it would appear that the best remedy is the

* Epileptics who are liable to attacks in the night should be very careful to remove false teeth from the mouth before going to bed, lest they should become impacted in the pharynx during a paroxysm.

inhalation of the *nitrite of amyl.* Dr Crichton Browne has recorded ten cases in which he employed it, and eight of them terminated in recovery. The effects of the remedy were of the most striking character. For instance, a man, aged thirty, had from May 6th to the 10th had from twelve to sixteen fits a day; on the 11th he was in a most critical condition; he lay on his back, breathing stertorously, with livid purple features, and streaming with perspiration; the pulse was 140, the temperature 103°. It seemed useless to make trial of the nitrite, but as a forlorn hope he was made to inhale five drops every hour. His breathing at once became less laboured, and he had only three more fits that day, and on the 12th there were gleams of consciousness; the pulse and temperature fell; on the 14th he could answer questions, and by the 17th the fits ceased, and he passed into his usual state of health, and was able to take part in domestic work. Dr Browne adds that he has found no other plan of treatment of nearly the same value in the status epilepticus as inhalation of the nitrite of amyl; although other measures may occasionally be useful. The withdrawal of a few ounces of blood has sometimes suddenly restored to consciousness patients who were in a state of profound coma. Some years ago a very striking instance of the same kind occurred at Guy's in the practice of Dr Wilks.

For the *prevention* of epileptic fits in those who are liable to them—in other words, for the cure of the disease, epilepsy—one remedy appears to surpass all others in efficacy,—this is the *bromide of potassium.* Sir Charles Locock in 1857 was the first to recommend it, and he spoke of it as being especially serviceable in women in whom the attacks coincided with the menstrual periods. But subsequent observations have shown that there is no such limitation of its curative power. It is given in doses of from ten to thirty grains three times daily, and it must be continued for months, or even for two or three years. Its effect is sometimes to free the patient for ever from the liability to recurrence of the attacks. In other cases it suspends them for a time, or diminishes to a marked extent their frequency and their severity, but when after a time its administration is suspended they become as bad as before. In yet other cases it does some good for a time, but seems to lose its power, although the patient may go on taking it without interruption. Lastly, in a very few instances it appears to be altogether useless. No explanation has yet been found for the varying effects of bromide of potassium in different cases. It has been stated that it is comparatively ineffectual when the seizures occur only during the night, but according to Dr Reynolds this is by no means always true.

Most persons can take ten to twenty grains of bromide of potassium three times daily without suffering any ill-effects. But when it is given in large doses it is apt to cause very striking symptoms after ten days or a fortnight, and the condition so produced has been called *Bromism.* According to Dr Bazire (Syd. Soc. translation of 'Trousseau's Lectures,' vol. i, p. 100) it is characterised by headache, apathy, impairment of the special senses and of common sensations, loss of sexual appetite and vigour, enfeeblement of muscular power in the limbs, tremor of the hands, and impaired action of the heart. A young American lady came under the editor's care, who, after taking bromides for several years to cure epilepsy, had fallen into a dull, stupid, listless condition, sitting for hours without moving and incapable of joining in society. A very definite symptom of

bromism is anæsthesia of the velum palati, uvula, and pharynx, which may be tickled without producing any efforts of deglutition.

Another remarkable effect is the production of a cutaneous eruption. This commonly resembles acne more or less closely; it consists of pustules surrounding hair-follicles, but these are arranged in patches or groups, and dry up into large scabs, beneath which the skin becomes red and thickened. One case of this kind is depicted on the forty-third plate of the Sydenham Society's 'Atlas.' In that instance the scalp and the extensor surfaces of the limbs were especially affected by the eruption, but the face and the legs are more often its principal seat.

One is not infrequently consulted by a patient who has just had a first epileptiform attack. It is then one's duty to prescribe the bromide without delay. The probability of its usefulness may fairly be inferred from its ascertained efficacy in the large proportion of the cases in which its value can be fairly tested. If one were to lay the matter in all its bearings before the patient himself, he would certainly wish to take the medicine regularly for a considerable length of time, even though, in the event of his remaining free from further attecks, it must always remain doubtful whether or not there was a real necessity for treatment.

But sometimes the bromide of potassium fails to check the recurrence of attacks of epilepsy, although given in the largest doses and with the utmost perseverance. One must then have recourse to some other remedy; perhaps the best is *belladonna*. This was especially recommended by Bretonneau and by Trousseau. At first a quarter of a grain of the extract should be given twice or three times a day; and the dose should be gradually increased. At one time a patient of the author's took two grains three times daily for a great length of time, with marked benefit. Trousseau insists on the necessity that this medicine should be continued for a long period, if its value is to be fully tested; a year, he says is sometimes scarcely sufficient for the discovery of its influence; and if in the second year there should be some improvement it may be worth while for the patient to go on taking it for three or even four years. He speaks of it as completely curing the disease in some very rare cases; but Dr Reynolds says that he has never known it do more than diminish the frequency of the seizures.

The salts of *zinc* are useful in some cases of epilepsy. Dr Wilks speaks of having had under his observation more than one case in which the patient was always better when under their influence. Dr Reynolds speaks favourably of the oxide, but has seen no good result from the sulphate. The nitrate and the oxide of silver have been recommended, but are probably useless. And one must not forget that, taken for a length of time, this drug will stain the skin of a bluish-black colour. It is believed that these preparations may be administered with safety if the course is not allowed to be continued for more than six weeks. Twenty years or more ago an American was to be seen about London who had been castrated and afterwards completely blackened by nitrate of silver in the hope of curing his epilepsy. The result was negative so far as the disease was concerned. His skin looked as if it had been polished with blacklead.

In some cases the introduction of a *seton* at the nape of the neck has led to the suspension of epileptic fits, at least for the time. About this Dr Wilks speaks decisively. A patient of his in Mary Ward, subject to epileptic fits, had a seton put in her neck. The fits ceased entirely. After more than a year the seton was removed and they re-appeared. It was again

inserted and again the convulsions ceased. The application of ice to the spine, as recommended by Dr Chapman, has been tried in numerous cases by Dr Reynolds, who reports that it did no good whatever.

The food of patients suffering under epilepsy should be digestible, and should be taken with regularity. Dr Wilks speaks of having seen cases in which reducing the quantity of meat has been followed by a decline in the number and severity of the fits; but in other instances a generous diet has been advantageous. In a lady, who by Dr Radcliffe's advice, lived on vegetarian diet for some years, the effect in checking the fits both in frequency and severity was marked. Some definite experiments in regard to the influence of animal food have been made at the West Riding Asylum by Dr Merson, who kept a number of patients for a month on a diet including much meat, and then for the same period on one in which there was no meat, or *vice versâ*; there was no very marked difference in the number of fits, but several of the patients were much more dull and languid when taking animal food than they were when kept on a diet mainly farinaceous in character.

Both the mind and the body of epileptic patients should be kept in exercise, short of fatigue. The limbs should never be allowed to get cold, particularly at night. Dr Reynolds says that in many cases nocturnal seizures have been prevented by a simple mechanical contrivance placed under the upper half of the mattress, so as to keep the head and shoulders well raised.

Paroxysmal Vertigo.—Another affection which may occur in paroxysms is vertigo or giddiness. The expression "paroxysmal vertigo," indeed, is not altogether free from ambiguity; for the analogous one, "epileptic vertigo," is commonly used as a synonym for that less severe form of epilepsy—the *petit mal*. The latter, however, is not always, nor even usually, attended with giddiness, and it is distinct from the complaint which we are now considering.

Two forms of paroxysmal vertigo are recognised by systematic writers. In one the patient feels as though he himself were made to turn round and round, or were against his will impelled forwards, or backwards, or to one side. In the other he fancies that objects are revolving round him. In each case he remains perfectly conscious, and all that occurs is duly registered in his memory. If the attack is not the first, he may be well aware that his sensations are without foundation; yet by the strongest effort of his will he may be incapable of overcoming their effect. In a first attack he may be completely deceived. A patient, who happened to be a railway official, when travelling on a particular occasion, beheld one side of the carriage suddenly rise four or five feet, and throw him into the opposite corner. He naturally supposed that there was a serious accident; but in reality he had not moved from his seat. Dr Ramskill relates that a patient of his, who was attacked while in the street, felt the pavement uneven, with alternate depressions and elevations over which he seemed to be obliged to lift his feet. At the same time the shop windows seemed to him to be moving forwards, and the passers-by to be racing after one another. He also felt giddy in himself; and as a matter of fact the two forms of vertigo just described cannot be regarded as distinct affections. In most cases both of them are experienced, either at the same time or in succession. The gait is unsteady or reeling; the patient feels afraid of running against other

people or surrounding objects; he catches hold of some support; he may even lose his balance and fall to the ground. Sometimes the act of closing the eyes removes the sensation of vertigo completely for the time. Nausea very commonly accompanies the attacks, and even vomiting.

Paroxysmal vertigo is often connected with impairment of the sense of hearing, the patient being more or less completely deaf on one or both sides, and generally experiencing sensations of buzzing or singing in the ears. In 1861, Ménière recorded in the 'Gazette Médicale' some remarkable instances of this kind; and of late much attention has been drawn to such cases under the name of Ménière's disease or labyrinthine vertigo; but the prevalent interpretation of their pathology is far from proved.

In the first place, it is certain that affections of the middle, and even of the external, ear may give rise to attacks of giddiness, faintness, and sickness. For example, in the 'Archives of Ophthalmology and Otology' for 1871, both Knapp, of New York, and Bremner, of Zürich, mention cases of aural catarrh, in which such symptoms showed themselves; and Toynbee many years ago asserted that cerumen accumulated in the external meatus might by its pressure on the membrana tympani produce similar effects.

In the great majority of cases, however, the more accessible parts of the organ of hearing are free from disease. If the deafness is of one ear, a tuning-fork is not heard on that side, even when placed upon the teeth or upon the top of the head. It is hence inferred that the seat of mischief must be the internal ear.

At this point some very interesting physiological observations appear to find their application. Many years ago Flourens discovered that in pigeons and rabbits section of the semicircular canals causes strange disturbances of equilibrium. More recently Mach, of Vienna, and Crum Brown, of Edinburgh, have shown good evidence that the function of these structures is to furnish the impressions which form the principal basis of our knowledge as to the relation between our movements and those of surrounding objects. They have even shown what are the several disturbances of equilibrium which irritation or destruction of each canal may be expected to produce. And accordingly it has been proposed to employ the name "labyrinthine vertigo" as synonymous with Ménière's disease. Charcot observed a case in which the lesion was chiefly in the left ear, and in which the direction of reeling was principally forwards, but sometimes backwards, while occasionally there was a sense of rotation on a vertical axis, always from left to right. This last would, according to recent writers, be due to irritation of the left horizontal ampulla, while movements forwards and backwards would respectively answer to irritation of the posterior and superior canals. Destruction of the same parts would, however, produce precisely the converse effects, and thus there is no difficulty in accounting for the fact that some patients have shown a tendency to reel towards the side on which they were deaf. In either case the actual movements are supposed to be the reflex results of the impressions conveyed to the coordinating centre from the various canals, which under normal conditions balance one another, but which no longer do so when some of the canals are diseased or injured.

Even when morbid changes in the meatus or tympanum are obviously present, it is commonly supposed that the direct cause of vertigo is disorder of the labyrinth; for pressure upon the fenestra ovalis can easily be conceived to cause increased tension in the semicircular canals. Thus all cases

of "auditory vertigo" might come to be regarded as alike examples of Ménière's disease or labyrinthine vertigo.

But it would be a great mistake to suppose that Ménière himself merely wished to draw attention to the fact that vertigo was apt to occur in those who suffer from deafness or from some disease of the internal ear. What was really new in his paper was that he endeavoured to show that sudden apoplectiform symptoms (including at least a transient loss of consciousness) might occur in a person previously healthy, and be followed for the first time by deafness, and that the cause of such attacks might be an affection of the internal ear. He relates several cases of patients who fell down insensible, and who, when they recovered, were found to be deaf; and a similar instance has been recently recorded by Knapp.

The only one of Ménière's cases in which a *post-mortem* examination was made is the tenth and last of his series. A young women, while menstruating, undertook a night journey outside a coach. She suddenly became completely deaf, and was admitted into Chomel's wards. The principal symptoms were constant vertigo and vomiting. She died on the fifth day. At the autopsy no disease could be discovered in the nervous centres; but the semicircular canals in each ear contained a reddish plastic substance. Surely this observation is very inconclusive. Cases in which an autopsy fails to reveal a satisfactory explanation for cerebral symptoms present during life are, after all, not very rare; and it seems rash to assume that the state of the labyrinths was the real cause of the fatal illness in Chomel's patient. Moreover, as Bremner points out, even if full value were allowed to the case in question, one could hardly take it as demonstrating the nature of those other cases in which cerebral symptoms come on suddenly and rapidly pass off. In these it has been supposed by some writers, including Knapp, that hæmorrhage takes place into the semicircular canals. They do not seem to have found any difficulty in the fact that, since the deafness is often simultaneous in the two ears, the blood must be effused on both sides at or about the same time. But this appears to render the explanation very improbable. It is true that hæmorrhage into both retinæ occurs in cases of Bright's disease; but surely not so as to cause sudden and total blindness.*

A different view has been suggested by Dr Wilks; namely, that when there is no affection of the meatus or tympanum, the deafness and the cerebral symptoms are both in some cases due to changes in the nervous centres. It is evident that such an explanation is particularly applicable to cases of Ménière's disease in the stricter sense of that term; the sudden loss of hearing in both ears may fairly be attributed to an affection of the auditory centre; and the giddiness to a similar affection of the centre for equilibrium, which is probably adjacent, since its most important afferent nerves are those which come from the semicircular canals. Whatever peculiarities in the direction of the vertiginous tendencies may be observed, such as have been supposed to depend upon affections of particular ampullæ, they can all be referred to corresponding changes in the centre, for in this the functions of each canal must be fully represented. The analogy of the other paroxysmal neuroses seems to support very powerfully Dr Wilks's view. We have seen that impairment of sight is a frequent symptom of migraine,

* The editor once saw retinal hæmorrhage in the course of chronic Bright's disease cause sudden and total blindness; but in that patient the other retina had been long affected without his knowledge.

and that it is certainly due to an affection of the brain and not of the eyes Indeed, "cloudiness before the eyes" and "obscuration of the visual field." are mentioned as having been present with the veritgo in some of Ménière's and Knapp's cases; and it may be that in these instances the attacks presented a combination of the two neuroses, the nerve-storm spreading beyond its usual limits and encroaching upon the area concerned in migraine. So also it seems to me that, in the "apoplectiform" cases, such as Ménière described, the simplest way of accounting for the loss of consciousness is to suppose that the disturbance diffused itself over the hemispheres, as it probably does in epilepsy.

Another strong argument in favour of Dr Wilks's view is afforded by the fact that bromide of potassium may remove both the giddiness and the loss of hearing at the same time; and Mr Hinton recorded under the name of Ménière's disease a case in which paroxysmal vertigo and sickness had been associated with only transient deafness, and in which all these symptoms together were brought back by the administration of quinine.

Knapp has observed that in certain cases the impairment of hearing is particularly marked for certain musical tones, those of the middle octaves being distinctly perceived, while those of the lower, and still more those of the higher octaves are heard very imperfectly. He suggests that this is a proof that the seat of the affection is in the labyrinth; but the force of the argument is not obvious. Much more weight must be allowed to an observation of Charcot's, that some patients experience sensations of vertigo and buzzing in the ears, only so long as the deafness is partial, and lose these symptoms as soon as it becomes complete. But even if we should have to admit that in these instances the internal ear is really the part primarily affected, it would by no means follow that the same thing is true of the "apoplectiform" cases, nor that the vertigo is anything but a neurosis. Why may not the same symptoms be produced in many different ways, and yet be always themselves the immediate result of one particular kind of nervous disturbance? Just as migraine may be excited by a variety of causes, so may paroxysmal vertigo.

Indeed, the analogy between these two neuroses must now be carried a step further. We discussed in detail the question whether migraine is ever due to disorder of the digestive organs, and found reason to conclude that such disorder certainly plays a part in its causation. Now, writers describe *vertigo a stomacho læso* as distinct from other varieties of giddiness. It may be that the stomach is not so often concerned in the production of vertigo as the liver, under the condition which Dr Murchison has called lithæmia. But without deciding this point the question is whether the giddiness due to disorders of digestion is different in kind from that which depends upon other conditions, such as deafness. Dr Wilks has remarked that the vertigo caused by derangement of the liver occurs chiefly when the patient stoops or lays his head upon the pillow, and ceases when he stands upright. But it will presently appear that this distinction is not constant; so that in many cases there is nothing in the character of the nervous symptoms themselves to show that they depend upon one rather than another of the various causes to which they may be attributed.

In some cases the ingestion of food which disagrees with the patient leads so quickly to swimming in the head that the connection cannot be overlooked. Dr Murchison speaks of a medical friend of his, who has long suffered from gout, and who, whenever he drinks a cup of tea or a glass of

champagne, is seized with sudden giddiness. His head feels empty, and neighbouring objects seem to whirl about him; he would fall did he not lay hold of something to support him. After a few seconds or minutes the attack passes off. In other patients the vertigo lasts longer. Dr Ramskill relates the case of a merchant who was one day quietly walking in the City from one office to another, when he was seized with giddiness, so that he reeled, and had to lay hold of a post which was near at hand. In a few hours, after a free evacuation of the bowels, he became better, but he felt weak and shaken, and complained of a heavy diffused headache. About three hours before the attack he had eaten hastily, and with imperfect mastication, a breakfast of which sausages and Devonshire cream formed a part; and to this the vertigo was ascribed, no doubt with justice. Yet, during the following month, the same patient had five similar attacks, not one of which could be traced to any such cause, he having in the meantime become very particular as to his diet. Dr Ramskill even goes so far as to say that in "stomach vertigo" it is the exception for one to be able to trace any positive signs of stomach disorder. The proof is that the complaint is cured by treatment directed to the regulation of the digestive organs. Thus, a second medical friend of Dr Murchison's, who had never had gout, and in whose case the only recorded indication of lithæmia was that his urine was often loaded with lithates, was seized with dimness of sight every night while writing. He took iron and quinine and other tonics, but without any benefit. He was advised to give up practice for a time, and try the effect of a change of air; but while he was making up his mind to so serious a step, he took a few grains of blue pill, whereupon his symptoms at once disappeared. So, again, Trousseau quoted from Boerhaave's commentary the case of a man who, during two years, was always seized with vertiginous symptoms whenever he attempted to stand up. In vain had the ablest practitioners attempted to cure him. He had an attack of gout from which he had never before suffered, and from that time the giddiness ceased.

To complete the chain of evidence which proves that vertigo is one of the paroxysmal neuroses, there remains the fact that it may replace other members of this group. Giddiness is occasionally present during the paroxysms of migraine; and Dr Liveing refers to two cases, in each of which an attack of intense vertigo, of short duration, appeared several times to replace the ordinary sick headache. The connection of vertigo with epilepsy is, perhaps, closer still. As I have already mentioned, giddiness is one of the symptoms of many cases in which fits take the form of the *petit mal;* and that affection is very often only the precursor of *epilepsia gravior.*

There are, however, cases in which this neurosis remains unchanged in type for many years. Dr Ramskill speaks of them as "essential" vertigo, and states that he has met with two instances in which the complaint appeared to be transmitted by inheritance. One of his patients suffering from vertigo had a father living, and then aged seventy-one, who had himself been subject to it for thirty-five years; he also had asthma. Another patient complained for three years of giddiness for which no cause could be discovered, and which resisted all kinds of treatment. It is true that in that case the giddiness after a time became almost continuous; and *persistent* vertigo is probably more often due to anæmia from disease of the arteries of the brain than to any other cause. But it seems probable that in exceptional cases any of

the paroxysmal neuroses may at last cease to occur in separate attacks with intervals. For example, a case is recorded by Charcot ('Progrés Méd.,' ii) of a woman, aged fifty-one, who had for six years suffered from continuous vertigo, which did not intermit even at night, and was so severe that she could not walk, nor even stand, for the slightest movement of her head made her clutch at surrounding objects for support. The complaint had lasted twenty-six years; but for a long time it was purely paroxysmal, and the attacks were comparatively slight. She had disease of the tympanum on each side.

The late Mr Hinton published nine cases of vertigo, usually of a paroxysmal character and associated with deafness, tinnitus, and nausea or vomiting, in the 'Guy's Hospital Reports' for 1873 (Third Series, vol. xxiii, p. 193). In some of these the hearing was affected on both sides, and in two (Cases 3 and 9) the perception of certain musical sounds was definitely impaired.

A classical instance of vertigo, nausea, deafness and pains in the head, coming on as a paroxysmal disease early in life and persisting until old age and senile imbecility, is that of Dean Swift. Dr Bucknill and Dr Legg have argued in favour of its being a typical case of Menière's disease; but we have of course no knowledge of the condition of the labyrinth. His account of the origin of his attack in a fit of indigestion while he was living with Sir William Temple would denote *vertigo a stomacho læso*.

In the *treatment* of vertigo, bromide of potassium is of more service than any other medicine, and its value is sometimes more striking than even in epilepsy itself. The aural surgeons use chloride of ammonium for those cases which are associated with deafness. A careful inquiry must be made for symptoms of dyspepsia or of lithæmia, and Dr Ramskill recommends that alkalies and vegetable bitters should be used in all cases on the chance of their being of service. It is a good plan to give the bromide with a few grains of carbonate of soda, and with equal parts of the infusions of rhubarb and calumba. Wine is valuable in senile vertigo. Charcot's patient was cured in from two to three months by fifteen grains of quinine daily; but in a very characteristic case of auditory vertigo lately under the editor's care, the patient had, by Professor Charcot's advice, taken quinine in full and repeated doses without the least benefit.

Paroxysmal Insanity.—There remains a paroxysmal neurosis in which the attacks take the form of transitory mania or other mental disorder.

A striking instance is recorded by Dr Maclaren in the 'Medical Times and Gazette' for 1876. The patient was a slight gentle-looking lady, aged forty-three, with winning manners and a soft quiet voice. She was characterised by exalted religious feelings and morbid sensitiveness. She would be reading her Bible and talking gently to her attendant, when suddenly, without a moment's warning, she would throw the book out of the window and make a rush to run her head into the fire; or she would, perhaps, turn on the attendant and try to strangle her. She would then struggle on, keeping all the time perfectly silent or uttering only an occasional word of Scripture until she was exhausted, or until by a kind of awakening she became restored to her former condition. Sometimes in the attacks she would expose her person. During the intervals she had no recollection whatever of what she had done; at the most she could slightly recall the impulse which led her to attempt some act of violence.

One peculiarity of the paroxysms in Dr Maclaren's case was that in each of them the patient made efforts to get at one particular picture, which at other times excited no emotion whatever. Thus it seemed that she followed out in successive attacks trains of thought of which she had no knowledge during the intervals; and her condition might so far be fairly termed one of "dual consciousness." Fabret has laid stress on the fact that all the paroxysms in the same patient may be exactly alike in their suddenness and extreme violence. He also remarks that it is characteristic of this affection that repeated blows are struck by the patient, and several persons injured.

By Fabret the affection now under consideration was described under the name of epileptic insanity—*furor epilepticus*. But the adjective is ambiguous, since it has been used as a general name for all mental disorders which may accompany or follow epileptic attacks. Moreover, it would appear that a liability to that disease is not always present. Dr Maclaren's patient had never had epilepsy, although she was a member of a family in which neurotic affections prevailed. Paroxysmal insanity is therefore a better name; but there is no question that the affection is one which bears a very close relation to epilepsy. As a rule, patients who have seizures of this kind have before suffered from attacks of the *petit mal* if not of the *haut mal*. Dr Hughlings Jackson, indeed, has recently expressed the opinion that a transitory epileptic paroxysm really occurs each time before the mental symptoms develop themselves. In other words he thinks that the affection is identical with that form of mania which I have described as sometimes following an epilectic fit. This view rests upon a theoretical basis. He supposes that a necessary condition for the occurrence of the mental disorder is the removal of the control of the highest centres, which are exhausted by having discharged themselves during the fit (cf. p. 686).

Even when paroxysmal insanity occurs in those who are really subject to frequent epileptic fits, one may be unable to ascertain the fact at the time. It often happens that a patient in this condition is brought to a public hospital by the police, and, as Dr Jackson remarks, it may be impossible for one to say whether he is an epileptic, or drunk, or suffering from meningeal hæmorrhage. He records a very interesting case of a woman who was brought to the London Hospital in a maniacal state, with a deep gash in the left arm, by which the elbow-joint was opened, and all the soft parts in front of it were cut through. She accused different people of having inflicted this wound upon her, but it was ascertained beyond doubt that she had done it herself. She had been cutting bread for her children's tea when she suddenly sent them all out of the room. A short time afterwards she was found lying in a pool of blood on the floor. On the following day she was rational, but furious mania returned several times during the next week. On inquiry it was ascertained that she had been subject to epilepsy, in both the minor and the graver form. The patient doubtless received the suggestion which led to the infliction of the wound from the circumstance that she had a knife in her hand at the time.

The sufferer from this form of insanity is particularly liable to find himself in the hands of the police for some offence committed during the paroxysm; and although to a skilled medical observer it may be perfectly evident that he is irresponsible, there may be much difficulty in making this clear to others. Dr Jackson has done much towards the eluci-

dation of such cases by studying other instances in which the acts performed are not criminal but absurd; they may be characterised by precisely the same degree of adaptation of means to ends, and yet they leave no trace on the memory. Thus one of his patients had been talking about supper, and it had been agreed that he and his wife should have some cold fowl, and her sister some cocoa. Soon afterwards he felt the symptoms of an attack and sat down on a chair against the wall of the kitchen, where he happened to be. He remembered nothing further, but his sister-in-law came in and found him standing by the table mixing cocoa in a dirty gallipot, which was half filled with bread and milk for the cat, and stirring the mixture with a mustard-spoon, which he could not have obtained except by going to the cupboard for the purpose. If the object fetched had been a knife, and if he had inflicted some injury with it, this purposive action would have seemed a strong point against him.

Dr Jackson, however, himself admits that the form taken by a man's mental automatism during the paroxysm depends very much on his natural disposition. A savage and suspicious man would, when his highest faculties were temporarily in abeyance, be more likely to kill someone than to mix cocoa for his sister-in-law. Indeed, the actions performed during a state of unconsciousness are sometimes exactly those which would have been performed if the individual had been in full possession of his faculties. The patient last referred to had on another occasion ordered dinner at an eating-house, when his mental condition underwent a change, and he remembered nothing more until he found himself at his desk in the office, feeling rather confused. He had to go to the place and ask whether he had had his dinner, and he then found that he had eaten it and paid for it, and that neither landlady nor waiter had noticed any peculiarity about him.

In other cases the patient goes through ordinary actions in an absurd way. Thus another of Dr Jackson's patients, while in an omnibus, was one day observed to blow his nose upon a piece of paper, and when he got out he gave the conductor £2 10*s.* instead of the usual coppers. The same physician had a patient, who one day found the extinguisher of a candle in his waistcoat pocket. For some years he invariably looked at his watch after each fit, and the supposition is that he must, in an attack, have mistaken the extinguisher for his watch.

Fabret long ago remarked that an impulsive tendency to wander about is characteristic of this form of insanity. Trousseau gives some instances. A gentleman was attending a literary meeting at the Hôtel de Ville, when he ran out and walked for some minutes on the quays, avoiding with success both the carriages and the passers-by. When he recovered, he found that he had neither great-coat nor hat; he returned to the room and resumed, with a perfectly lucid mind, the historical discussion in which he had before taken an active part. The same gentleman, being a magistrate, was presiding at a provincial tribunal, when he suddenly got up, muttered a few unintelligible words, and went into another room. He was followed by the usher, who saw him make water in one corner, after which he returned to his seat. Another patient of Trousseau's, an architect, used often to have an attack while walking across a narrow plank at a height from the ground. He never met with any accident, although he would run rapidly over the scaffolding, shrieking out his own name in a loud abrupt voice. A quarter of a minute afterwards he would resume his occupation and give orders to his workmen, without any recollection of what had occurred to him.

In attacks of this kind the state of the patient is often said to be that of a man walking in his sleep; and the expression "*day*-mare" has been invented for them by way of contrast with *night*-mare. True somnambulism (at least when it is not a manifestation of hysteria) has every claim to be regarded as one of the members of the group of paroxysmal neuroses (cf. *infra*, p. 830).

The same is the case, too, with the "night terrors" to which some children are liable, who start up an hour or two after going to sleep, screaming with fright, and unable for some minutes to recognise the parents or nurse. That these various attacks should occur only at night is after all nothing more than occurs in many cases of epilepsy itself.

HYSTERIA AND HYPOCHONDRIASIS

HYSTERIA—*A real and distinct disease—Mental and moral perversions—Hysterical affections of sensation and of movement—Contractures—Globus hystericus—Flatulence—The hysterical fit—Catalepsy—Sleep-walking—Ecstasy—Convulsions—Mania—Hystero-epilepsy—Anorexia and marasmus—Ætiology and Pathology of hysteria—Diagnosis—Prophylaxis—Treatment—Hysteria in men and boys.*

HYPOCHONDRIASIS.—*Distinctions from hysteria—The supposition of imaginary diseases, commonly abdominal—Distinction from melancholia—May mask real disease—Treatment—Prognosis.*

HYSTERICAL AFFECTIONS—disorders of function, or pains, or perverted sensations—are seated in the most diverse organs of the body. In books on surgery many complaints are mentioned under the same name, which befall the joints, the mammary gland, or various regions of the trunk or limbs. Such a use of the term "hysterical" implies that there is one disease common to all the affections in question; and that disease—*hysteria*—so far as its general characters are concerned, is the subject of this chapter.

But first is hysteria a distinct and definite malady? For there are some who refuse to admit its claim. In this they find support from two sides. (1) On the one hand, the name* (derived from the Greek word ὑστέρα, a womb) suggests the erroneous opinion that the organs of generation in the female play the principal part in causing hysterical affections. This, indeed, would not in itself matter, for many maladies have names etymologically unjustified. What creates the difficulty is rather the circumstance that a fundamental difference of opinion exists at the present time between physicians of different schools as to the extent to which uterine or ovarian complaints are to be regarded as the cause of the disease now under consideration. Those who see little or no connection between them have been tempted not only to do away with the name, but to ignore the clinical association of the symptoms as manifestations of a single malady. (2) On the other hand, it must be admitted that, more than most other diseases, hysteria is difficult to define. As a neurosis independent of any appreciable structural change, its definition would naturally be based upon either its causes or its symptoms. But the former are still the subject of discussion, and the latter are singularly variable and inconstant. Moreover, the curability of most hysterical affections, and the fact that they are generally contrasted with organic diseases, have caused a tendency to apply the term vaguely to cases of unascertained nature, but which are regarded as of a trifling character and likely to be of transient duration. In this way the words hysteria and hysterical have often been deprived of all real meaning.

Very little study at the bedside, however, is needed to convince one that the various affections called hysterical are really the expression of a special

* Ὑστερία (*i. e.* womb-sickness)—Fits of the mother—The vapours.

morbid condition, for which the name of Hysteria is as convenient as any other, besides having the sanction of antiquity and established usage. One finds that two or more of these affections are commonly present in the same patient at the same time, or that a person who has suffered from one is exceedingly apt to be afterwards attacked by others. Then, again, there are marked peculiarities in the age, sex, disposition, and other conditions of such patients. Above all, there is in most cases a particular mental state which can easily be recognised apart from other indications of the disease, and which may be regarded as its fundamental character.

Mental condition.—The principal features of the mental state which characterises hysteria are an exaltation of the emotional faculties, and a perversion of the will or a loss of the proper balance between it and the judgment. The patient seems to have little or no power of controlling her own feelings. The most trifling occurrences excite her to rapture or plunge her in despair. She may conceive a violent affection for some persons, and an equally irrational aversion from others. She perhaps exhibits great attachment to some pet animal, while other creatures no less harmless excite in her repugnance and disgust. She torments all those about her with her caprice and her egotistical claims upon their attention and forbearance. Her will may seem to be altogether in abeyance; she may appear to be unable to make up her mind to the smallest effort, and may lie in bed all day from sheer want of energy. But in some particular direction she will perhaps show the most obstinate determination and tenacity of purpose. She has a great craving for sympathy, and is exceedingly anxious for her delicacy of health to be recognised. In pursuance of this object she will apply corrosive acids to her skin, swallow needles day after day, or run them into all parts of her body and make attempts (almost always abortive) to poison or drown or hang herself. The stoical endurance of pain which she exhibits under such circumstances is sufficient to show that a mere defect of volitional power cannot be the essential element of hysteria. Perhaps the best way of expressing the facts is to say with Dr Reynolds ('Syst. of Med.,' vol. ii) that the will is no longer called into exercise by the judgment or reasoning faculties, but only by some one dominant idea or emotion. Thus, as Jolly observes ('von Ziemssen's Handbuch,' vol. xii), if one can by stimulating her enthusiasm or spirit of emulation supply a hysterical patient with a sufficient motive to undertake any good work, such as nursing, she will often carry it out with more than ordinary perseverance and energy. In regard to the will, as well as to the emotions, the peculiarity of hysteria is that there is a loss of the due balance and proportion between the several faculties.

Moral perversion.—The moral tone of a hysterical woman is often seriously impaired, particularly in certain directions. Morbid sexual inclinations have often a strong hold upon such a patient, and she will plan and scheme to induce her medical attendant to make a vaginal examination or otherwise to gratify her perverted feelings. Sir Thomas Watson relates a case in which a young woman contrived to make a hospital surgeon believe that she had a stone in the bladder; and the imposture was not detected until she had been tied up in the position for lithotomy in an operating theatre full of students. I have met with an instance in which a patient who had been supposed to have a hydatid in the liver, and who had before been punctured without result, allowed the operation to be repeated, and almost immediately produced a piece of the stomach of a rabbit (or some other small animal) which she declared she had vomited, and which she no

doubt thought would be mistaken for a hydatid membrane; a few hours later she sent for her medical man again, to remove from her vagina another piece of the same substance. Others have been known to drink their urine and then to bring it up again, pretending that none was passed in the natural way. The fact that such cases have occurred makes one hesitate to accept as authentic such cases as have been described on the authority of Charcot as hysterical ischuria. There is often great difficulty in detecting impostures of this kind. Charcot himself relates a case of Boyer's in which a woman pretended that urine came from her umbilicus, ears, eyes, and breasts, and that she vomited fæces; and it was not until she had been placed in a straight waistcoat that little balls of fæces ready prepared for swallowing were found in her bed. Hysterical young women also simulate phthisis and pretend to spit or vomit blood.*

Some of the *factitious affections of the skin* are by no means easy of diagnosis. In the museum of Guy's Hospital there is a model of the right breast of a girl, which is reddened and has on it a number of large raised tubercles, some of them bigger than peas. The first time that patient was an inmate of the hospital the cause of the affection remained undiscovered, although it was evident that some irritant was being intentionally applied. But more than a year afterwards she was again admitted, and Mr Birkett, happening to visit the ward at an unusual hour, found a piece of lint, strewn with powdered cantharides, which she had put on the breast. Sir William Gull related a similar case in which he detected with a lens a glistening fragment of the same powder on the skin itself. Some years ago a girl was admitted into Miriam Ward who had on her chest and breasts a number of whitish gangrenous-looking patches, of irregular sizes and forms; these successively shrivelled up into brownish scabs, which soon became detached, leaving the skin beneath reddened and rather scaly. On one finger, in the groove between the skin and the nail, there was found a bright yellow stain, which affected both structures to about the same extent, and which seemed indisputably to have been caused by a drop of nitric acid. It therefore seemed clear that the patches on her chest were likewise produced by the acid, although they did not show the characteristic yellow colour. It happened strangely enough that shortly before she came in there was in the ward another patient affected in exactly the same way but (there was no reason to doubt) with spontaneous gangrene. She also was a girl, aged eighteen, but apparently free from suspicion. The museum of Guy's Hospital contains models taken from each of these patients.

Other forms in which hysterical patients exhibit their craving for notoriety and their willingness to deceive are illustrated by the cases of the Welsh fasting girl, and Louise Lateau, the Belgian Nun.

Among the particular manifestations of hysteria some are frequently met with, some are very rare. One, "the hysterical fit," has always been regarded as especially typical of the disease; and two others, the "globus' and a flatulent distension of the belly, are perhaps still more common and scarcely less distinctive. One must make diligent inquiries after all these in any case the nature of which appears doubtful. Before describing them, however, it will be convenient to mention some symptoms which are less often seen.

* A remarkable case of the kind obtained some notoriety about thirty years ago by the publication of a book called 'The Female Jesuit in the Family.' The patient, a typically hysterical governess, imposed on the Protestant credulity of the household.

Perversions of sensation.—Not the least curious of these are perversions of the special senses. The patient perhaps complains of intolerance of light and insists on having the room kept darkened. Here, however, imagination often plays a great part. Dr Reynolds relates the case of a woman who had for weeks been lying with her hand before her eyes to keep out the light of a dull London sky. When he brought a candle close to her in order to examine the pupils she shuddered, knit her brows, and held both hands between it and her eyes. But as soon as her attention was distracted to the state of her front teeth, the brows became relaxed, the hands were removed, and she bore the light without inconvenience. In other cases the patient is distressed by the slightest sound and will allow no one to speak above a whisper. Yet such a person may herself exclaim in a loud voice or make a great noise in stirring the fire without seeming to mind it. In other cases there appears to be an actual exaltation of perception. A hysterical girl may seem, as Dr Reynolds says, "to hear through stone walls;" but in such matters deception is often practised, as when such a patient pretends that she can see with her eyes shut.

Jolly quotes from Amman the case of a woman who discovered the presence of some cherries in another room by their odour, and who could distinguish one individual from another with her nose; and such persons often detect by their taste the presence of a perfectly infinitesimal trace of any flavour that happens to be disagreeable to them. It is perhaps a perversion of the gustatory sense that leads girls affected with hysteria to eat cinders, sealing wax, lead-pencil, and the like; this perverted appetite used to be called "pica." They are sometimes equally fond of repulsive odours or flavours.

The sense of touch, again, may be unduly acute. In almost every case of hysteria there is at some part or other of the body an over-sensitiveness to painful impressions—a *hyperæsthesia*, or rather, in strict language, a *hyperalgesia*. Sometimes the patient complains that it hurts her very much to have the skin over one or more of the spinous processes pressed upon or even touched; sometimes she has extreme tenderness of the breast, or of the edge of the costal cartilages.

Another frequent effect of hysteria is impairment of common sensation, "*dysæsthesia*" or "*anæsthesia*." This may either be limited to ordinary tactile impressions or it may include those of heat and cold; in other instances it concerns only the perception of pain, and then the name of "analgesia" is sometimes applied to it. Gendrin went so far as to declare that sensation was more or less defective in every case throughout the whole course of the disease; and other writers have asserted that loss of feeling in some part of the skin is invariably left behind by hysterical fits. Their statements, indeed, are too absolute, but the symptom is undoubtedly often present, and Lasègue and Charcot are probably right in saying that it would be more frequently observed if it were carefully looked for, a person being sometimes unaware that she is unable to feel pain in some particular part, until her sensibility is tested. In many cases, however, subjective sensations of tingling, pins and needles, &c., are experienced and complained of by the patient. It is important to note that there is no opposition between hyperæsthesia and anæsthesia, such as might appear from the names. A part of the skin may be exquisitely tender, and the seat of burning pain when touched; and yet at this very part the power of transmitting definite tactile impressions may be greatly impaired.

Anæsthesia, or defective sensation, is much more frequently observed in some regions than in others; Jolly mentions the backs of the hands and feet, and the parts above the outer malleoli, as its favourite seat. Sometimes it affects exactly one half of the body, leaving the other free. To such cases Charcot has given the name *Hemianæsthesia*. He quotes Briquet as having stated that this occurs on the left side more often than on the right side in the proportion of seventy cases to twenty. The limitation of the parts in which there is loss of feeling is often remarkably sharp, corresponding almost precisely with the median line of the face, neck, and body. It differs from hemiplegia in this character and by the greater implication of sensation than of motion. The mucous membranes are affected in a similar way. Taste may be wanting in one half of the tongue, the sense of smell may be impaired, and there may be a considerable degree of amblyopia, with limitations of the visual field for the several colours—a symptom which Galezowski calls *Achromatopsia*. According to Jolly, reflex excitability is also wanting; sneezing cannot be induced by irritating the nose, nor retching by tickling the fauces on the affected side. Lastly, Charcot says that the side on which sensation is impaired is cold and pale and that there may even be a difficulty in drawing blood from the skin by pricking it with a needle.

Altogether apart from hemianæsthesia, loss of feeling in the mucous membranes is common. Anstie laid considerable stress on the frequency with which it may be found, if looked for, in the back of the pharynx. According to that writer ('Lancet,' ii, 1872), whenever a person who has not been taking a bromide can without retching let one pass the finger down to the epiglottis, the diagnosis of hysteria is exceedingly likely to be correct. Dr Reynolds mentions that he has known several cases, occurring in married women who were still bearing children, in which there was absolute anæsthesia of the vulva and vagina. Impairment of sensation in the bladder is supposed to be sometimes the cause of retention of urine in hysterical patients, but in other cases it appears to be due to a prurient desire to have a catheter introduced. Jolly relates the case of a patient who inflicted terrible burns on herself by taking live coals out of the fire and pressing them with both hands into her vagina, but who seemed to experience no pain whatever.

Hysterical anæsthesia is probably never permanent. It may last for several weeks and even for months, but sooner or later it always passes off.

Paralysis.—Another manifestation of hysteria may be paralysis of movement. Aphonia and dysphagia often result from hysterical loss of power in the laryngeal and the pharyngeal muscles respectively; and the former is highly characteristic of the disease, so that its presence gives great aid towards the diagnosis of doubtful cases. Not uncommonly hysterical paralysis takes the shape of paraplegia or of hemiplegia. The former affection was described at p. 459. The latter likewise presents characters of its own. Much more frequently it occurs on the left side than on the right. The proportion in Briquet's cases was as forty-six to fourteen. An important distinction between it and the paralysis due to disease localised in the opposite side of the brain is that in hysterical hemiplegia the side of the face and tongue always remain entirely unaffected. This was long ago remarked by Dr Todd, who also pointed out that the patient in attempting to walk "drags the palsied limb after her, as if it were a piece of inanimate matter, and uses no art of circumduction nor effort of any kind to lift it

from the ground; the foot sweeps the ground as she walks." Dr Reynolds supplements this by saying that a woman suffering from hysterical hemiplegia cannot help lifting the great toe, like a healthy person, when she endeavours to put the foot forwards; and, again, that a paralytic patient looks at her feet, a hysterical patient at the persons who are watching her.

In some cases one limb is alone affected with hysterical paralysis; or, much more rarely, only part of a limb. I do not remember to have seen any one muscle affected singly, with the exception of the levator palpebræ; but hysterical ptosis is not uncommon.

Hysterical paralysis may be very transitory, lasting only a few weeks or days, or not longer than a few hours. Sometimes it afterwards reappears either in the same limbs or in others. But there are cases in which it persists for months or even years. I do not know that it is ever permanent, except in some of the cases in which rigidity sets in, to be mentioned in the next paragraph. It may disappear suddenly, under the influence of a sudden emotion or shock; or it may pass off very gradually. The removal of the patient to a hospital ward—away from sympathising friends and relations—is often followed by a more or less rapid recovery. In several cases under Dr Wilks's care this result has been brought about rather by moral influence than by any special treatment. The patient is told that she is expected to begin to move the paralysed limbs; day by day she is encouraged to do more and more; after a time she is taken out of bed, and dressed, and put into a chair; and before long she walks as well as ever.

Contractions.—Another and a very curious effect of hysteria is a chronic spasm or rigidity of one or more of the limbs, associated with a complete loss of power of voluntary movement. This has recently been studied with much care by Charcot, who has published in his 'Leçons' several capital drawings illustrative of the distortions which it produces. Some English writers have since adopted into our language the French word *contracture* for such cases. The affection often follows immediately upon a severe hysterical fit; and the paralysis and the rigidity then generally develop simultaneously. But in some cases the latter does not come on until there has been loss of power for a considerable time. In certain cases only one leg or one arm becomes contracted; but sometimes both legs are affected, sometimes both the arm and the leg on one side, sometimes three of the limbs, or even all four. When the affection assumes a hemiplegic type, and the rigidity follows the loss of power after some interval, it might be thought that there would be a difficulty in distinguishing it from the "late rigidity" of hemiplegia due to organic disease of the brain; but in the hysterical affection the contraction comes on, not gradually, but suddenly, and as the immediate result of a fresh hysterical fit. Moreover, the positions assumed by the individual limbs are peculiar. The arm becomes semiflexed. But, according to Charcot, the leg always falls into a state of rigid extension, the knee and ankle being straightened and the toes stretched down to the utmost extent possible while the sole is turned inwards. Thus the condition of the foot resembles that which exists in talipes equino-varus. Charcot also mentions that the adductors of the thighs are forcibly contracted; and Jolly relates a case in which one leg became powerfully flexed, especially at the knee-joint. In a marked case of hysterical paraplegia (which afterwards completely recovered) the girl's legs were both forcibly flexed at the knees and hips, so as to resist the utmost power that could be safely used to straighten them,

until chloroform was administered. She afterwards recovered completely, and became an excellent nurse.

The rigidity in these cases is not relaxed in the most profound sleep; nor does it undergo any variations in degree at different times or periods of the day. The spasm is by no means confined to a single set of muscles, for one is not able to force the patient's arm (which is semiflexed) into a position of complete flexion, any more than to extend it. By making the patient inhale chloroform, however, if its action is carried far enough, one can temporarily remove the spasm, the affected parts becoming perfectly supple in all but very exceptional cases.

Anæsthesia of the affected limbs is generally present. The muscles remain well nourished, and retain their electro-contractility at least for a considerable time. But when a limb has been contracted for some years without intermission, a little general wasting may occur, and the reaction of the muscles to galvanism may be somewhat impaired. The termination of hysterical "contracture" is almost always in recovery; and in many cases this takes place quite abruptly. One of Charcot's cases is very instructive. A woman was first attacked, at the age of thirty-four, with loss of consciousness after a moral shock; she then fell into the fire and burnt her face severely. After this she had several seizures, some of them hysterical in character, others rather epileptiform. Four years afterwards she had a very severe fit, attended with convulsions and followed by an apoplectiform stupor with stertorous breathing; this was at once followed by left hemiplegia. Rigidity of the left limbs set in abruptly after an interval. In the following year the right limbs also became contracted; and later still the jaws were fixed, so that an œsophageal tube had to be used. Her right arm, however, became free; and Charcot maintained that recovery was still possible. One evening, six years from the commencement of the contraction of the left arm and leg, she had an attack in which she imagined herself to be about to die. She cried out, became agitated, and with her right arm pushed aside those who held her. Her strong desire to get to the window for air was resisted; she became more and more angry; and first her right leg lost its rigidity, then her left leg, and finally her left arm. She got up and walked; and in eight hours her cure was complete. A slight crackling in the joints was all that remained of the complaint.

Such a case has an obvious bearing upon those in which cures are attributed to miraculous agencies. They are seldom related in such a way as to enable one to speak positively as to the exact form of paralysis which had been present. But Charcot quotes an article by Littré in which detailed histories are given of certain persons who were cured at the end of the thirteenth century by pilgrimages to the tomb of St Denis, where the relics of Louis IX had recently been deposited. Three of these were young women who had been suddenly attacked with contraction of one leg or of the arm and leg on one side, and who also had anæsthesia. They were cured suddenly, under circumstances certainly very likely to act upon the imagination. In fact the analogy seems to be complete.

In some cases of hysterical "contracture," however, recovery never takes place. Charcot mentions several instances which he believed to be hopeless. One is that of a woman, aged fifty-five, whose legs had been contracted for sixteen years. Under chloroform her knees could still be relaxed; but her feet remained in a condition of equino-varus. This observer has made an autopsy in one case in which all four limbs had been contracted for ten

years ; and he discovered sclerosis of the lateral columns in nearly the whole length of the cord. The patient was a hysterical woman, and the rigidity passed off several times and returned again, before it became permanent. There seems no reason to doubt that the case originally belonged to the same class as the rest. Charcot accordingly supposes that sclerosis may come on secondarily in such cases. The points on which he would lay stress as indicating the probability of an unfavourable termination to a case of long-standing contraction of a limb are that particular groups of muscles should be wasted in an extreme degree and affected with fibrillary tremor like what is seen in progressive muscular atrophy ; that there should be a great diminution in the contractility, as tested by faradisation ; and that the rigidity should remain to very considerable extent when the patient is under the influence of chloroform. On the other hand, he does not attach any importance to a little general loss of substance in the affected limbs, to a slight impairment of the electrical reaction of the muscles, or to the presence of spasmodic movements, which last are not uncommon in such cases, as in those of ordinary hemiplegia (see p. 576).

Globus.—Among the most common and characteristic symptoms of hysteria there is one—the so-called *globus hystericus*—of which there is a difficulty in saying whether it is merely a morbid sensation, or depends upon spasmodic contraction in the œsophagus and pharynx. The former opinion has been maintained by Eulenburg and Reynolds, the latter by Jolly. To the patient herself the globus is a sensation, most commonly a feeling as if there were a ball or lump in the throat, which she cannot swallow or get rid of in any other way. She may even put her finger into the pharynx, and make herself sick, although she knows there is not really anything that can be dislodged ; or she may drink mouthful after mouthful of water to wash it down. The exact nature of the sensation varies a good deal in different persons. In some it is likened to that which would be caused by a bean sticking in the throat ; others feel as if a worm or some other living animal were creeping about in its interior. With others, again, it is rather a feeling of constriction about the neck as if the clothes were too tightly fastened. The relation of such a feeling to the state of emotional excitability which characterises hysteria is shown by the fact that a precisely similar sensation is felt by healthy persons when suddenly seized with grief or terror. Indeed, children before a fit of crying experience a sensation of a lump in the throat which is probably identical with the more marked form of globus. In some hysterical women the lump is not merely felt in the throat, but seems to rise up from the chest, or even from the epigastrium. Rumbling movements in the bowels are also complained of by many of these patients, and perhaps this is the best argument in favour of the opinion that the globus is also due to peristaltic contractions in the gullet.

Flatulence.—Another frequent symptom of hysteria is a rapid distension of the intestines with gas. The abdomen may suddenly swell up, so that the patient is obliged to take off her stays and undo all the fastenings round her waist. Jolly remarks that this form of tympanitis bears no relation to meals. In several instances he has seen it return each morning regularly and attain its maximum about midday. The gas may ultimately escape either by the mouth or from the rectum. Jolly says that it is commonly quite free from odour ; and this, he adds, explains the fact that the distension sometimes subsides without any apparent escape of wind in either direction. How such an enormous quantity of gas can be produced in the stomach and bowels is as

yet altogether unknown. It is sometimes undoubtedly air which has been swallowed ; sometimes probably gases, liberated from the food and expanded in accordance with physical laws by relaxation of the muscular walls of the stomach and bowels. The chemical character of the gas needs more exact determination.

In some cases hysterical tympanites persists for years together, the abdomen being as tense as a drum. Bamberger alludes to a case of this kind, in which the patient passed through an attack of cholera without alteration in the size of the abdomen, but in which an abundant flow of saliva, coming on spontaneously, once led to its temporary subsidence.

The hysterical fit.—There remains to be described that which was for a time regarded as the chief symptom of hysteria, the hysterical attack or "fit of the mother." But the truth is that in the majority of cases no attacks occur during any part of their course ; according to Briquet in three cases out of four. Moreover, when they do occur, they present infinite varieties of symptoms. Sometimes they are of so trifling a character that it is obviously unnecessary to send for medical advice. The patient perhaps has an unusually painful globus, and then begins to cry and sob, or falls into violent laughter. Or she may laugh and cry alternately, until she is exhausted. Such an attack, for which the vulgar name is "hysterics" and was "vapours," may occur singly, or there may be several in the course of two or three days. A very constant feature of them—and, indeed, of all kinds of hysterical fits—is that when they pass off the patient voids a very large quantity of colourless urine, of low specific gravity, and in fact almost pure water.

But in many cases a hysterical attack is accompanied by violent move ments of the body and limbs, which may be distinctly convulsive in character. Perhaps the patient screams out, and then falls into a state of opisthotonos, remaining supported for some minutes together only by the back of her head and by her heels. Or she may throw her limbs in all directions, beating them on the ground, or striking her own chest with her closed fists, tearing her hair, kicking those about her, and struggling with all her might to release herself from restraint. Or, again, she may thrust one arm high in the air ; or forcibly bend her arm over the chest, and her thighs across one another ; some of the fingers and toes being at the same time stretched apart to the furthest possible extent, while others are as strongly flexed. The respiration is much quickened, and it may be very noisy, but it is not actually stertorous. Her features undergo the strangest contortions and grimaces. Her eyes are generally closed. The eyelids quiver, and the eyes themselves, the pupils of which are of natural size, are often turned upwards, particularly if an attempt is made to separate the lids by force. The state of the eyes thus differs altogether from that which belongs to an epileptic fit, for in it they are commonly fixed wide open, and have their pupils greatly dilated.

There appear to be all degrees of impairment of consciousness in hysterical attacks. At the commencement the patient almost always knows what is happening, so that she is able to place herself out of the way of injury. Sometimes she keeps on screaming during the greater part of the fit, or shouts out "fire," "thieves" or "murder," or pours forth a continuous torrent of the foulest language. Sometimes she has visual hallucinations, addressing furious objurgations to imaginary persons, or fancying that she is surrounded by wild beasts or spectres. Sometimes she seems to

be altogether unconscious; but even then by careful watching one may often make out that she really notices what is going on, casting occasional furtive glances at the bystanders from between the half-closed eyelids or modifying her behaviour under the influence of their remarks.

Catalepsy.—In some instances a hysterical attack is attended with very remarkable symptoms, which have long been regarded as belonging to a special neurosis named catalepsy (κατάληψις = a seizure or arrest). Its characteristic features are that the patient, although she may be deprived of sensibility and of voluntary motion, remains fixed in whatever position she occupied at the commencement of the fit, and yet that her muscles offer no such resistance to external force as would prevent the limbs from being easily bent or extended by another person, or the body from being placed in any posture. The condition of a cataleptic patient may be compared with that of a lay figure, such as artists use; if she is sitting up her arms can be put at the most awkward angles with the trunk, and will remain without falling, at least for a time; if she is recumbent her spine may be bent upon the pelvis so as to form an obtuse angle with the thighs, and will retain that posture. The name of *flexibilitas cerea* is sometimes given to this peculiar state of the muscles; they have been found by Rosenthal to have their electric sensibility and contractility either normal or decidedly increased. It is, however, a mistake to suppose that during the cataleptic state the muscles are capable of resisting the force of gravity for an indefinite length of time. On the contrary, the limbs, if extended, slowly fall again to the side. Dr Chambers quotes a case in which an impostor was detected by the simple plan of attaching a weight to the extended hands. The patient (as she was supposed to be) supported it without moving, and this was taken as showing that she was not labouring under a genuine attack of catalepsy; ultimately she confessed the fraud. The eyelids of a cataleptic patient may either be widely open or shut; in the latter case, if opened, they perhaps very slowly close again. The pupils may contract under the influence of light.

These details of the "cataleptic" paroxysm appear to belong to this place, because it probably never occurs in a well-marked form except in persons who either are obviously hysterical or who at least may be strongly suspected. A case of the minor form of epilepsy was quoted (p. 793) in which it assumed a cataleptic character; but in that case the *flexibilitas cerea* does not seem to have been observed. Fully-developed catalepsy does not appear even to accompany epilepsy in a person of the male sex, or in a woman presenting no hysterical tendencies. The attacks seem almost always to be traceable, at least in the first instance, to a fright or to some other powerful emotion. Dr Chambers mentions the case of a girl, a patient in St Mary's Hospital at Paddington, who when Covent Garden Theatre was burnt down was awakened by the flashing into the ward through the uncurtained windows; on the following morning she was attacked with catalepsy. It is noticeable that the most striking instances of catalepsy which stand recorded are to be found in the medical works of a former generation, published at a time when the modern complex conception of hysteria was not dreamt of, and yet the reports of these cases often contain clear proofs that the patients were in the highest degree hysterical. The celebrated history related by Dr John Jebb nearly a century ago is a case in point. The subject of it was a young lady who suffered from hysterical risings in the throat and flatulence, and who was highly suscep-

tible to every change in the weather. A very suggestive feature is that although she was prepared for Dr Jebb's visit when he first went to see her, she was seized with the disorder as soon as his arrival was announced. "She was employed in netting, and was passing the needle through the mesh, in which position she immediately became rigid, exhibiting in a very pleasing form the figure of death-like sleep. . . . The positions of her fingers, hands, and arms were altered with difficulty, but they preserved every form of flexure they acquired, nor were the muscles of the neck exempted from this law, her head maintaining every situation in which the hand could place it as firmly as her limbs. . . . About half an hour after, the rigidity and statue-like appearance being yet unaltered, she sang three plaintive songs in a tone of voice elegantly expressive, and with affecting modulation." The seizures sometimes lasted as long as five hours.

In other cases similar attacks have been of still longer duration. The condition of the muscles has then been generally different from the *flexibilitas cerea* of catalepsy proper; either the limbs oppose considerable resistance to all attempts to change their posture, or they are completely flaccid, falling into any position whatever under the mere influence of gravity. Such patients may most fairly be said to be in a state of trance. Writers describe this condition as sometimes lasting for days and weeks at a time. The face may then be perfectly pale, the breathing so feeble as to be imperceptible except on the closest examination, and even the heart's pulsations and pulse at the wrist discoverable with great difficulty. These are the cases which have led to the popular belief that death is sometimes only apparent, and that there may be a danger of persons being buried alive. And it cannot be denied that a patient in such a condition might easily be allowed to die by careless or ignorant attendants, or might be buried before death.

Sleep-walking.—In other hysterical persons the attacks assume the form of somnambulism or of ecstasy. The former is a condition which may be compared to an acted dream. It is probably sometimes a transformation of epilepsy or some other paroxysmal neurosis; and when not allied to those diseases it is almost always a manifestation of hysteria. The somnambulist, without seeming to wake from a state of sleep, walks all over the house and even out of doors. She balances herself without difficulty on narrow planks, and manages to avoid all obstacles in her path. She takes no notice of anyone whom she may meet, there is much difficulty in rousing her, she remains for a time bewildered, and she has no recollection of what she has been doing. Marvellous stories are told of the feats which are accomplished by persons in this strange condition. But one must not forget how untruthful hysterical patients often are, how prone to take a pleasure in mystifying and deceiving those about them, and how apt to be encouraged in such a course by the admiration and wonder which their performances call forth, particularly when they are sufficiently exaggerated.

Ecstasy.—The state of ecstasy is one in which a person becomes regardless of all external circumstances and engrossed with some particular emotion or idea. She may remain motionless, with staring eyes and fixed expression, or she may repeat a few words with ceaseless monotony, or dance or spin round and round with extraordinary vigour and persistency. In the "Tarantulism" of the Middle Ages, and in the epidemics which have more recently affected religious enthusiasts in Scotland and America, men suffered as well as women. But there seems to be no doubt that, underlying the

attacks, there was a loss of balance between the different faculties of the mind, with an exaltation of the emotional susceptibilities exactly like what we have seen to be essential to hysteria.

Other varieties of hysteria.—For certain cases, in which movements of one particular kind are incessantly repeated for an extraordinary length of time, the special name of *malleation* has been invented (cf. *supra*, pp. 722, 723). Some years ago Dr Wilks had a girl under his care, who sat by her bedside and kept thumping at something with her fists all day long. But the most remarkable instance of this kind on record is probably that which was related by Mr Kinder Wood ('Med.-Chir. Trans.,' vol. vii). The patient, a young married woman, who had suffered severely with pain in one side of the face, began to be troubled with involuntary movements in the eyelids, which were opened and shut with excessive rapidity. After a time the hands were beaten rapidly upon the thighs, and the feet upon the ground. Then she became half raised from her chair and seated down again, these movements succeeding one another as quickly as they possibly could. Other modifications of her attacks occurred from time to time, until at last she took to skipping about the room, regulating her movements by a series of strokes on the furniture as she passed, or by movements of her lips, as though beating a tune. Someone thinking he recognised the air as "The Protestant Boys" began to sing it, and she suddenly turned and danced up to him, and continued dancing till she was out of breath. Then a drum and fife were procured, and she immediately danced up as close to the former as possible, and went on until she lost the step, or until the measure was changed, or was made so rapid that she could not keep up with it. It was presently found that a continued roll of the drum also had the effect of putting an end to her movements; and this instrument being used as soon as the attacks threatened to begin, their occurrence was at length prevented. The explanation which the patient gave of them was that "there was always a tune dwelling on her mind which, at times becoming more pressing, irresistibly compelled her to commence the involuntary motions."

Still more extraordinary movements were performed by a lady who came under Dr Abercrombie's observation, and who had suffered for two years from various nervous affections. She, while lying perfectly quiet, would suddenly with her whole body make a kind of convulsive spring, by which she was jerked entirely out of bed. Or, if sitting or lying on the floor, she would fling herself into bed, or leap, as a fish might, on to a wardrobe fully five feet high. After a time the muscles of the back and neck became affected with a wonderful semi-rotatory movement, which sometimes went on without interruption night and day for several weeks together. If the head or neck were touched, the motion was increased to a most extraordinary degree of rapidity. She was cupped in the temples, when the affection suddenly ceased with a general convulsive start of the whole body. But it returned again and again, and finally disappeared only when treatment was used to correct constipation and menstrual disorder from which she suffered.

Some years ago a similar case occurred in Guy's Hospital. A little girl, aged nine, had been knocked down by a boy five days before her admission. She was insensible at the time, and seven hours afterwards she had a fit. Subsequently she had nine other fits. They began by her making a low sighing or moaning sound, after which the upper extremity became contracted, the teeth were clenched, and with a sudden bound she threw herself completely out of bed. When she recovered con-

sciousness she had no remembrance of what had occurred. On the day of her admission she had fourteen fits, and at one time she remained insensible for two hours. A day or two later she had two fits during the visit of the physician, Dr Owen Rees; in these she clapped her hands, and her face went through a most extraordinary series of contortions. In one attack she struck a part of her head to which a blister had been applied, whereupon she at once became conscious. After she had been in the wards six days one of the fits was commencing, when the nurse told her that she must be tied down; she immediately began to cry and recovered, saying that she would have no more fits. From that time she remained well.

The final manifestation of the disease in Dr Abercrombie's patient—when the head revolved from side to side—has been dignified by certain systematic writers with a special name, that of "*rotation.*" Several instances of it are related by Sir Thomas Watson as having been recorded by different observers. The most striking of them is one related by Dr Watt ('Med.-Chir. Trans.,' vol. v) which occurred in Scotland in 1813. A girl, aged ten, who had before exhibited other nervous symptoms, was seized with a propensity to turn round on her feet like a top, with great velocity, always in one direction. This continued a month and then passed off. Afterwards she began to roll over and over on her bed, moving rapidly from one end to the other, for six or seven hours every day. Having been taken into a garden she quickly rolled along the whole length of a gravel walk, and even when she was laid in the shallow part of a river, she began to turn round as usual, although she seemed to be on the point of being drowned. She made little or no use of her arms in performing the rotations, of which there were about sixty in a minute. At the end of another month or six weeks she began an entirely new set of movements. Lying on her back she could by drawing her head and her heels nearly together, bend herself like a bow, then she would relax her muscles, and fall with considerable force on her seat. This she repeated ten or twelve times a minute for several hours daily. After a fresh interval, she became possessed with a propensity to stand on her head; and as soon as her feet were elevated perpendicularly in the air, to let herself fall again, her knees striking the bed first. This movement also she continued for fifteen hours a day, at the rate of twelve or fifteen times a minute. Blistering, purging, and leeching were employed without result; but having been taken to Glasgow from her home in the country in an open chaise, and brought back again after three days, she was seized with diarrhœa, and soon afterwards recovered entirely.

In other cases, again, *maniacal excitement* with hallucinations and delusions complicates the hysterical attack, or even constitutes its main feature. Such symptoms may be transitory, the patient becoming perfectly rational as soon as the fit passes off, but in many instances they last for a considerable time, and then it may be very difficult, and even impossible, to distinguish the case from one of insanity. Dr Blandford remarks that the points specially indicative of insanity are that the patient should betray strong amatory feelings, that she should pretend to be unable to speak, and that while seeming to be violently delirious, she should nevertheless be thoroughly alive to all that is going on, and display an exercise of volition in what she does. But many very doubtful cases find their way into lunatic asylums, and the truth seems to be that no actual boundary line between hysteria and insanity exists, the one disease being very prone to pass into the other. Dr Savage, in the twenty-first volume of the 'Guy's

Hospital Reports,' (1876) says that a large number of the female insane patients admitted into Bethlem Hospital have suffered from severe hysteria at former periods in their lives, and that many cases, at first regarded as examples of hysterical insanity, end in death or dementia. Some observers have made it a sort of test for hysteria that the mental faculties should be found unenfeebled when recovery takes place, even after a considerable length of time. And thus many doubtful cases have been at last set down as hysterical because of the suddenness and completeness with which the patients have regained their senses. Dr Savage speaks of one woman who imagined that she had the itch, and who refused to shake hands with anyone; she woke one morning quite free from delusion and full of wonder at her fancy; and he alludes to other patients who have gone to bed believing themselves eternally lost and have awakened full of hope and faith.

Hitherto we have not mentioned the occurrence, in a hysterical attack, of rhythmical *clonic spasms* at all resembling those which are seen in an ordinary epileptic fit. But spasms of this kind are not infrequently present, the other phenomena of the seizure being such as to leave no doubt as to its nature. In 1876 a girl was in Guy's Hospital whose main symptom was a rhythmical clonic spasm recurring at intervals of a few seconds in the neck, shoulders, and arms, and who had transitory fits in which she seemed to lose consciousness, while these jerking motions became much intensified and succeeded one another with greatly increased frequency. One feature in that case was the perfectly bilateral character of the spasms, and this accords with the statements of writers to the effect that in hysteria the convulsive movements are rarely limited to one side, or even greatly more severe on one side than on the other, whereas in epilepsy each of these conditions is well known to be of frequent occurrence.

Epileptiform hysteria.—In some cases of hysteria, however, fits occur which in all respects resemble those of epilepsy. The loss of consciousness is complete; the eyes are widely open and the pupils dilated; a bloody foam escapes from between the lips, and the tongue is bitten; the movements are clonic rhythmical spasms of the simplest kind, and repeated again and again with but little variation. Perhaps this should not surprise us, for there are few diseases of the higher nervous centres (whether functional or organic) which may not be accompanied by similar attacks, of the most typically epileptiform character. But writers on hysteria, having probably in view the necessity of laying down a line of distinction between such fits and the ordinary hysterical seizures, have created for the cases in question the special designation of "hystero-epilepsy;" and there have been many discussions as to whether they are transitional links between the two neuroses, or instances of their co-existence in the same individual. But, as we have seen (p. 792), the disease epilepsy is defined not merely by peculiarities in the fits themselves, but by the fact that they recur again and again without any cause, unless it be an inherited tendency to nervous disorder. If it is theoretically inaccurate to regard as epileptic the fits to which some patients are liable for months or years as the result of the irritation of a carious tooth, it must be equally incorrect to give such a name to the fits which occur in a woman only while she is under the influence of hysteria. And Charcot has recently pointed out that the so-called hystero-epilepsy differs altogether from true epilepsy in some important particulars. However closely the attacks may follow one another, they do not, as in the status epilepticus, entail danger to life with the development of a high

bodily temperature. He relates the case of a woman who for more than two months laboured under a constant repetition of epileptiform fits. Once they succeeded one another without a break from 9 a.m. to 8 p.m.; and then again from 9 p.m. onwards for an equal space of time. He estimates that she had from 150 to 200 attacks on that one day alone. As he remarks, a patient suffering under epilepsy would soon have succumbed; but in his case there was at no time the least anxiety as to the issue. The woman never passed urine nor fæces involuntarily; and got out to relieve herself in the brief intervals that occurred between the attacks. Her temperature rose occasionally for a short time to 101·3°; but the mean was 98·6°. As further distinctions between hystero-epilepsy and epilepsy Charcot alleges that the former never assumes the type of the *petit mal;* and lastly that, however perfectly epileptiform a fit may be, it can always be arrested by compression of the ovary.*

Professor Mendel has lately (1887) shown the Medical Society of Berlin an apparently typical case of hystero-epilepsy or epileptiform hysteria, in the person of a man fifty years old.

Occasional fatal event.—The rule that hysterical fits are devoid of danger to the patient's life is not altogether without exceptions. Wunderlich met with a case in which a servant girl, aged nineteen, who had for eight weeks been labouring under a succession of epileptiform fits unattended with pyrexia, fell suddenly into a state of *collapse*, and died in two days with a temperature of 109·4°. Other cases have been recorded by Meyer in which excitement of a nympho-maniacal character was followed by cramps and spasms in various muscles, and led to collapse and death after an illness of some weeks.

Again, the more chronic forms of hysteria sometimes prove fatal by *marasmus*. Dr Wilks has related in the 'Guy's Hospital Reports' for 1866 the case of a young lady who lost her sister rather suddenly by heart disease, whereupon she began to experience palpitation and sickness and pain over the heart, would take no food, declared that she had her sister's complaint and should soon follow her to the grave, and did actually die in a sort of hysterical convulsion exactly five weeks afterwards. He also gives the case of another girl who would eat nothing, but merely nibbled a biscuit and drank wine by drops. The bowels ceased to act, but masses of scybala were from time to time removed from the rectum. She became thin, and Dr Wilks at length renounced the opinion that the complaint was merely hysterical, and thought that there must be some partial obstruction in the small intestine. But when, at the end of more than two years, she died rather suddenly, he could find nothing to account for her illness. Her body was then fatter than when he first saw her, and she had in fact taken rather more food towards the last.

A somewhat similar complaint has been described by Sir William Gull ('Clin. Soc. Trans.,' vol. vii) under the name of *anorexia nervosa vel hysterica*. It is attended with extreme wasting, and is commonly supposed to be the result of some latent tubercular affection. But, remarks the writer, the emaciation is actually too great for this diagnosis to be correct; for persons with organic disease seldom become so thin as this until they are no longer

* The extraordinary gestures of these unhappy patients have been carefully studied and named, as phases of "Ecstasie," of "Clownisme," and so on. See a report of a visit to Salpêtrière by Dr Gamgee ('Brit. Med. Journ.,' Oct. 12th, 1878); and details of cases in the same No., p. 561, with Professor Charcot's lecture, ibid., p. 789. Some of the attitudes are shown to coincide remarkably with those of dæmoniacs in works of art.

able to get about, whereas it is characteristic of the hysterical anorexia that those affected with it display an excessive restlessness and bodily activity. In this complaint the pulse and the respiration are slow, and the temperature is generally below the normal. Sir William Gull's patients were chiefly young women between the ages of sixteen and twenty-three. It does not appear that any of them showed definite indications of hysteria. Some of his cases terminated fatally,* but in others a complete recovery took place, the main points in the successful management of them being the avoidance of fatigue, the administration of nourishing food at very frequent intervals, and the use of warm clothing.

Ætiology.—In passing on to consider the causes of hysteria, we find ourselves face to face with a question which even now can hardly be said to be settled, although it has been discussed for centuries—I refer to the question as to the part played by affections of the generative organs in the ætiology of the disease. As I have already remarked, its very name involves etymologically the doctrine that it is the result of uterine disorder, and the ancient Greek writers—including Plato and Aristotle—actually supposed that the womb left its place in the pelvis and wandered about the body, setting up disturbances in different parts. From their time to the present there have never been wanting theories which, according to the different standpoints afforded by the science of the age, have referred hysteria to a similar cause. The physicians who have held such views in extreme forms have, indeed, been generally professed gynæcologists rather than men acquainted with the pathology of nervous diseases; but Romberg, the great German authority on these disorders forty years ago, defined hysteria as "the reflex neurosis proceeding from sexual irritation."

By different writers various disorders of the female sexual apparatus have been made the starting-point of hysteria. Some have endeavoured to trace it to unsatisfied desire, citing in support of their views the frequency of its occurrence in young widows and in the wives of impotent husbands, others have referred it to masturbation or to excessive venereal indulgence, and others again to the common menstrual disorders. Addison published a clinical lecture in 1830 to show that its phenomena depended upon "uterine irritation." By Dr Henry Bennet ulceration and induration of the cervix uteri were represented to be morbid conditions of great potency in causing such effects. More recently Dr Graily Hewitt has assigned the principal place to flexions of the womb.

Charcot has laid great stress on the frequency with which hyperæsthesia of the ovary is present. When lecturing on this subject at the Salpêtrière he showed to his class five women (almost all the hysterical patients who were then in the hospital) each of whom had pain in, or tenderness on pressure over, one or both of the ovaries. According to him the pain is sometimes very acute, and widely diffused over the abdominal surface, with special hyperæsthesia of the skin over an area two or three inches in diameter, corresponding with adjoining parts of one iliac and of the hypogastric regions. In other cases no spontaneous pain manifests itself; indeed, the skin is anæsthetic, and the muscles can be pinched up without pain; but on making deep pressure towards the brim of the pelvis, one can feel the ovary as an egg-shaped body, which slips beneath the fingers.

* I once saw a fatal case of this form of hysteria. Extreme emaciation preceded death. At the autopsy there was confirmation of suspected vaginal irritation, but the organs were like those of starvation. The cord was normal to the eye, and histological examination revealed no lesion.—Ed.

Further pressure upon it gives rise, not exactly to pain, but to a peculiar sensation which the patient at once recognises, having felt it many times before as the starting-point of hysterical attacks; and this, passing up to the epigastrium, is followed by pain in that neighbourhood, and sometimes by nausea and vomiting. Next, if the pressure on the ovary be continued, the patient experiences palpitation of the heart with extreme rapidity of the pulse, a sensation of globus in the neck, a hissing noise in the ear of the same side, a feeling as if blows were being struck with a hammer on the temporal region, an impairment of vision, especially in the corresponding eye, and then perturbation of consciousness, ending in a regular hysterical fit. Charcot also says that when there is hemianæsthesia, or paralysis, or contraction of the limbs on one side of the body, it is always the ovary on the same side which is hyperæsthetic; and that if such symptoms are bilateral, both ovaries are affected. Lastly, he has found that when a patient is seized with a hysterical attack, even if it is epileptiform, it may be cut short by energetic compression of the ovary continued for three or four minutes.

Charcot, although he speaks of ovarian hyperæsthesia as "playing a predominant part" in the clinical history, nowhere speaks of it as the *cause* of the disease. There are, in fact, ample proofs that Romberg's definition, already quoted, is inaccurate. Thus hysteria is occasionally met with in males; according to Briquet in as many as 5 per cent. of all cases, and Dr Reynolds says that in them its development seldom bears any relation to the generative functions; he has sometimes observed it in boys at puberty, but the most marked cases which he has seen have been in men from thirty-five to fifty or upwards, who have been mentally overworked or greatly reduced in physical power. So, also, in women, although probably more than one half of those who become affected with hysteria show the earliest indications of it between the twelfth and the twentieth years, about the time when menstruation was first being established, there are yet a considerable number of girls who manifest hysterical symptoms at a much earlier period of life. No fewer than one in five of Briquet's cases occurred in girls under the age of puberty.

It is one of the most important facts in the ætiology of hysteria that, even among children, it is far more common in girls than in boys. We have seen (p. 749) that this is the case with chorea likewise, and in both diseases the preponderance of cases in females is probably due to the same cause, namely, their innate emotional susceptibility and want of power to resist external influences. It has been correctly remarked that hysteria seldom, if ever, shows itself in such women as would be called "masculine," whereas the men and boys who are attacked by it are those who would be said to be of a "feminine" disposition. One influence which does much to favour the development of the disease is the want of a proper education. If the girl is fanciful and capricious her parents yield to every whim. If she is morbidly self-conscious, she is allowed to indulge her vanity, and to nurse the belief that she is gifted and misunderstood. If she is studiously disposed, she is left to pore over books, and never gets proper exercise in the open air. If she craves for precocious excitement, she is taken to balls and theatres when still young, she is permitted to sit up late at night, to spend the morning hours in a soft bed, to live on a rich and stimulating diet, and to feed her imagination with romances. The influence of example may also induce a liability to hysteria; the daughter of a hysterical mother

is very likely to acquire the disease in her turn; so potent does this cause appear to be, that according to the best authors there is no need to suppose that hysteria is ever transmitted by descent. In comparison with the emotional excitability in a girl, her intellectual capacity has but little influence either in rendering her susceptible of hysteria or in guarding her against it. As regards bodily constitution, some hysterical women are robust, with fresh-coloured cheeks and a vigorous circulation, but by far the larger number are weakly and delicate.

The real relations between hysteria and affections of the generative organs are best elucidated by the aid of the analogy afforded by some of the paroxysmal neuroses. We have seen that in many persons the attacks of megrim are constantly excited by disorders of the visual apparatus, but that in others this is not the case. We have found that vertigo is in some individuals brought on by diseases of the ear, and in some by diseases of the digestive organs, but that in others it is traceable to neither. So also it appears that the neurosis, hysteria, cannot essentially depend upon any one set of external exciting causes. In some cases it perhaps develops itself spontaneously. Very often anæmia or chlorosis appears to play the most important part in its causation, and it has sometimes been observed as the direct result of deprivation of food. In other instances—probably in the immense majority—its manifestations are directly called forth by impressions which reach the nervous centres from without. But these are by no means always derived from the generative organs. Emotional and psychical influences—a sudden alarm, the shock caused by the death of a relative, or the unexpected loss of property—may bring on hysteria in a person who had before seemed perfectly healthy. A similar result may follow a gunshot wound (as has been observed by Weir Mitchell), or it may be brought about by a railway collision. In some cases a spirit of imitation suffices for its production; the disease has been known to spread like an epidemic from one woman to another among the inmates of a hospital or a workshop. In other instances, as was pointed out by the late Dr Anstie, indulgence in alcohol seems to be the occasion of hysteria.

Again, it often happens that several of the conditions which may bring forth manifestations of hysteria are present together. A person who is attacked after a sudden fright may before have had her health depressed by misery and exhaustion of mind and body, or may have been labouring under religious excitement, jealousy, wounded self-love, or remorse. The liability to hysterical symptoms of all kinds is always increased at the periods of menstruation, and there is reason to believe that it is also augmented in many women at the climacteric age, although this does not appear from statistical data.

But no doubt there are many cases in which disorder of the generative organs is really the main cause of the development of this neurosis, and it is practically wise to accept Addison's teaching that such disorder should be carefully searched for whenever it can possibly be supposed to exist. Fortunately we are able to investigate the state of the ovaries by pressure above the brim of the pelvis without offence to a patient's delicacy; but writers are by no means agreed as to the actual facts in regard to the frequency of ovarian irritation or other morbid conditions of the sexual apparatus in hysterical women. Jolly disputes the accuracy of Charcot's statements as to the proportion of cases in which hyperæsthesia of the ovary is present. He quotes Scanzoni and Amann as having found among their hysterical patients as many as 19 or 20 per cent. in whom the generative organs were

perfectly healthy, and, as he remarks, the fact that these observers were professed gynæcologists renders it certain that their figures in regard to this point are too low. Speaking from his own impressions he is disposed to state the proportion of hysterical patients free from these complaints at more than 50 per cent. ('Ziemssen's Handbuch,' xii, 2, p. 461).

Diagnosis.—That this is often difficult must be sufficiently apparent, not only from what has been said in the present chapter, but from the repeated references to hysteria which have occurred in our discussion of organic diseases of the nervous system. The point to insist on here is the importance of careful search in all doubtful cases for the positive indications of hysteria which have been described at pp. 823, 827. Anæsthesia limited to small portions of the cutaneous surface is probably present in many cases without the patient's knowledge, and its discovery may go far towards clearing up a doubtful case.

At the same time one must bear in mind that a woman may be hysterical and yet be affected with organic disease, either of the brain or of some other part of the body. This remark is true even of cases in which all the symptoms of hysteria are present in the most marked form, still more if we include its more aberrant or doubtful manifestations. Sydenham went so far as to say that the majority of women were hysterical, and since the psychical characteristics of this neurosis are after all only an exaggeration of those which belong to the whole female sex, it is obvious that no absolute boundary line can be drawn.

Again, it is important to note that there is no kind of proportion in the degree to which the different symptoms of hysteria are developed in the same patient. A woman who has hemianæsthesia, or paralysis, or some local pain may present scarcely any indications of emotional susceptibility, may be quite free from uncomfortable sensations in her throat, and may never have had the slightest approach to a hysterical attack. Yet the whole course of the case may show conclusively that the complaint is of this nature. Indeed, when one has to deal with the more marked affections, such as paralysis or contraction of a limb, which is clearly not due to organic disease, one may be justified in making a diagnosis of hysteria, even in the absence of all corroborative evidence.

So, again, one may safely attribute to the same cause all cases of hemianæsthesia if unaccompanied by motor paralysis. And even when the loss of sensation is limited to a single limb, one would probably be seldom wrong in coming to the same conclusion. Sir William Gull, however, has recorded in full detail a case in which he at first made a mistake ('Guy's Hospital Reports,' Third Ser., vol. iv). A nurse in the hospital, aged thirty eight, complained of numbness in the left arm. Below the elbow sensation was completely wanting; but when the skin of different parts above that joint was carefully tested with the point of a needle, there were all kinds of contradictions in her statements. She would one minute say that she could feel the needle at a certain spot and the next minute that she could not feel it. Such observations led to the conclusion that the case was one either of feigning or of hysteria. But after the anæsthesia had existed for two years, the limb became gradually paralysed. Two years later she died from the effects of an accident, and it was then found that the spinal membranes were thickened, especially in the cervical region, and that the posterior columns of the cord and the grey matter had undergone degeneration.

Treatment.—We must in the first place consider what degree of success

may fairly be aimed at in dealing with hysteria. We can hardly expect to modify to any great extent the peculiar morbid constitution which constitutes its essential feature, although moral influences may strengthen the power of self-control. A complete cure of the hysterical tendency is seldom to be looked for, at least until the patient has passed the climacteric period. The earlier the stage at which the disease began to manifest itself the more unfavourable is said to be the prognosis. It is very important that a child who displays emotional excitability which seems likely to develop into hysteria should be placed under the most favourable hygienic influences possible. Exercise in the open air, a plain nourishing diet, limitation of the hours of study, early rising, sea bathing, and the use of the shower bath, at once suggest themselves as prophylactic measures.

When a hysterical patient is anæmic or chlorotic, she should of course take one of the preparations of iron for a considerable time. If there be any uterine disorder it is generally advisable that the patient should employ some astringent injection for the vagina, such as the compound alum lotion which Addison recommended so highly, and which contains a drachm of sulphate of zinc and a drachm of alum in half a pint of water.

In dealing with special symptoms, again, one must never overlook the importance of moral treatment. We have seen (p. 825) how successfully this may sometimes be employed in the management of paraplegia—an affection which, more than almost any other manifestation of hysteria, is apt to persist for months and even years if left to itself. So, also, one can sometimes cure hysterical aphonia by urging the patient to speak and confidently assuring her that she is able. For this affection electricity, too, is very useful. Dr Reynolds recommends that sparks should be taken from the larynx; or that a shock should be administered from a Leyden jar; or that an interrupted current should be passed through the throat. In long-standing cases Dr Morell Mackenzie's instrument may be employed, by which galvanism is applied directly to the vocal cords. Each of these methods often leads to the instantaneous restoration of the voice. Dr Reynolds speaks of a strip of empl. lyttæ applied round the throat as having occasionally been successful when electricity had entirely failed.

Paralysis of the limbs may be treated with advantage by faradisation, and passive movements and frictions should also be diligently employed. But Dr Reynolds says that he has found no remedy so effectual as the application of a narrow piece of blistering plaster completely round the affected parts.

Hysterical anæsthesia is said to be sometimes benefited by the application of a rather powerful faradic current by means of a dry metallic brush. Stimulating liniments may also be rubbed into the affected parts.

The remarkable effect on hysterical anæsthesia of the application of gold coins, metallic bracelets, and magnets, and even of pieces of wood and other indifferent objects, attracted much attention a few years ago at the Salpêtrière Hospital (see the second Report by MM. Charcot, Luys, and Dumontpallier to the Société de Biologie, 'Brit. Med. Journ.,' 1878, vol. ii, p. 548). It is difficult, if not impossible, to unravel the underlying facts from the tangled mass of hysterical perversions, physical and moral; but any good effect which "metallotherapie" may have by its action on the imagination of the patient is more than exceeded by the attendant evils (cf. Dr A. H. Bennett's case, 'Brit. Med. Journ.,' 1878, ii, 563; and Dr Donkin's paper, ibid., p. 613).

At present metalloscopy and metallotherapy have begun to recede, whither

the once famous "metallic tractors of Perkins" and odic force of Reichenbach preceded them; and we hear more of "hypnotism," a revived mesmerism, and "suggestion" in this induced hypnotic state.*

For rigid contraction of the limbs, the application of the continuous galvanic current is sometimes useful. But probably benefit more often results from straightening the affected joints under chloroform and placing the limb upon a splint, than from any other plan of treatment. Sir Thomas Watson speaks of cases in which a stream of cold water directed upon the contracted part and continued in spite of the patient's complaints of pain, led at once to the relaxation of the spasm. He also mentions an instance in which Sir Charles Clarke cured by the same method a girl who had hysterical trismus and could neither speak nor eat. He began to pour pitchers of water upon her face, but before he had emptied the second pitcher she began to scream and complain, with her mouth open widely enough.

When hysteria manifests itself by producing a chronic and long-standing contraction of a limb, the administration of medicines seems to be altogether useless. But for most of the other effects of the disease drugs are certainly useful. Most writers speak of assafœtida and valerian as owing their virtues chiefly to their disgusting taste. But many hysterical women actually relish assafœtida. Moreover, pills containing valerianate of zinc ought on such a theory to be almost inert, whereas there is no doubt that, in doses of gr. j—ij, they are most effectual in removing aphonia, hemianæsthesia, and hysterical hemiplegia. In 1874 a woman was in the clinical ward of Guy's Hospital who had paralysis of her left arm and leg, the latter being rigidly extended; she took large doses of assafœtida for some days without benefit, but was afterwards quickly cured by the valerianate of zinc, so that she walked out of the hospital within five days from the time when she first began to take it. In other cases we have obtained equally good results with assafœtida, five grains of which were taken every three or four hours. Dr Reynolds has found bromide of potassium altogether ineffectual in any form of hysteria. Both he and Jolly recommend that opium or morphia should be given for the relief of pain and to procure sleep; and they rightly observe that for the former purpose the subcutaneous injection of the alkaloid is especially effective. But one must always keep before one's mind the evil results which may follow from the establishment of a morphia habit (see p. 429). The continued use of chloral as a hypnotic is to be objected to on precisely similar grounds. And perhaps more harm is done by alcohol than by either of them. The late Dr Anstie was well justified in the strong protest he uttered against the practice of advising a hysterical woman to take a glass of wine or a teaspoonful of brandy, or even a dose of sal volatile to keep up her spirits whenever they are depressed, or to enable her to bear a frequently recurring pain. One should never sanction the employment of alcohol in any form by such patients, except perhaps in very moderate quantities and at meal-times only.

When one is called to a patient who is actually in a hysterical attack, there are several methods by which one may succeed in cutting it short. One plan, suggested by Dr Hare, is to keep the patient from breathing for a certain time by forcibly closing her nose and mouth. The vigorous inspiration which she makes, as soon as she is allowed to do so, is said to be

* See the late Mr James Baird's excellent little book, 'Magic, Witchcraft, Animal Magnetism, Hypnotism, and Electro-Biology' (1852); and Professor Heidenhain's 'Animal Magnetism,' translated by Dr Wooldridge, with a preface by Mr Romanes (1880).

often followed by a relaxation of all spasm and a subsidence of the fit. Dr Reynolds says that he has found this mode of treatment notably useful when an attack is sufficiently prolonged to make it worth while to interfere with the natural course of events. Another method, to the value of which many can testify, consists in drawing the patient's head and shoulders over the edge of the bed and pouring cold water upon them from a large jug. Sometimes one has merely to make preparations for carrying out this procedure, by giving the necessary orders to the patient's friends in her hearing, in order to bring the fit to an end. An ancient hospital sister in Esther Ward never failed to cut short an outburst of hysteria among her patients by the same homely method.

Compression of one ovary will sometimes arrest a hysterical fit, even although it may be of an epileptiform kind; Charcot speaks of one patient who learnt to apply the pressure herself. Another procedure, often adopted by the late Mr Stocker, consists in pressing upon the arteries and other structures on each side of the neck, in the same way as has already been described under the treatment of epilepsy (p. 809).

One must always keep in remembrance the fact that, excepting in seizures of a strictly epileptiform character, the patient is perfectly alive to all that is going on, although she may seem to be unconscious. A calm and quiet demeanour, the avoidance of all expressions of sympathy or of uneasiness about the issue of the attack, may do much to shorten its duration; for if one is flurried or alarmed she is sure to notice it.

Hysteria in male subjects.—As above stated, this remarkable neurosis or combination of neuroses is, in the vast majority of cases, a disorder of the emotions, the will and the bodily functions of *women*—particularly incident to the periods of puberty, of early married life and of the menopause, but possible at any time after the infant has developed into the girl. Nevertheless undoubted cases of hysteria occur in the male sex, just as undoubted cases of hypochondriasis occur in women. It is extremely rare in full-grown men, but is not very uncommon in boys and youths from the age of eight or ten to puberty and, less frequently, from that period till two or three and twenty.

In Dr Wilks's lectures on 'Nervous Diseases' there are several cases recorded. In one the principal symptom was a so-called laryngismus, in another aphonia, in another convulsions which were supposed to be due to spinal meningitis, in another the "malleation" or hysterical hammering with the arms referred to above (p. 832). Sometimes the boy can be thrown into spasms like those of tetanus or of a frog poisoned with strychnia, as in a patient of Mr Holden's who was cured by removing a fatty tumour.

Dr Rühle, of Bonn, relates the case of a spoilt boy of fourteen, who had terrible convulsive attacks ending with vomiting, and was cured by "removal from home, cold shower baths, and the fear of the rod" ('German Clinical Lectures,' p. 449).

The editor was once consulted about a schoolboy, who had twice been laid up at home on account of hæmorrhage from the bowels. He was ruddy and well nourished, and no disease of any organ could be discovered. A specimen of "melæna" was at last procured, and proved on examination to contain no blood, but a silver salt from a solution that he had used in photography. He then became violent and threatened to kill himself; but was soon cured by the wholesome discipline of a public school.

A naval surgeon gave a graphic relation in the 'Lancet' a few years ago, of a cabin boy of sixteen, who completely lost his voice and appeared to suffer from attacks of choking and suffocation. He was carefully treated for several days, until a sudden and unexpected pinch made him swear loudly, and thus revealed the imposture.

While these sheets are passing through the press, we have in Philip Ward an apparently healthy young countryman, twenty years old, who beside dyspepsia and hypochondriasis, suffers from "fits" of a decided neurotic character with well-marked globus hystericus.

HYPOCHONDRIASIS.*—From hysteria we pass to hypochondriasis by what seems a natural transition, for these two neuroses are commonly regarded as closely allied to one another, and do in fact resemble one another in one prominent symptom—namely, their tendency to simulate organic diseases of various parts of the body. Indeed, some writers have gone so far as to declare that hypochondriasis, which chiefly occurs in men, is in the male sex the representative of hysteria in the female; and the uncertainty which still exists as to the relative frequency of hysteria in men and boys arises mainly from the fact that cases which would be at once set down as hysterical, if they occurred in women, are often wrongly attributed to hypochondriasis because the patients are of the other sex.

But hypochondriasis may be seen in women, especially about the time of the menopause, without any admixture of the proper character of hysteria. Moreover, there is an essential difference between the two diseases. We have seen that the mental state in hysteria is characterised by an exaltation of the emotional faculties, and an increased susceptibility to outward impressions, the will being unable to control the feelings. But in hypochondriasis the morbid sensations which the patient experiences are not dependent upon any external cause; they are created by the mind itself. And thus Romberg was not without justification when he designated it the very antithesis of hysteria.

Again, hypochondriasis is unattended with any of those nervous vagaries —convulsive attacks, paralytic affections, hyperæsthesiæ, anæsthesiæ— which are so characteristic of hysteria. Its predominant symptom is always of one kind; the patient believes, without cause, that he is the subject of serious bodily disease. It is true that the full development of the complaint is said to be in most cases preceded by a period in which there is merely a general perversion of his feelings. He becomes gloomy, reserved, and wrapped up in himself; or his mental state alternates between a moody silence and high spirits with great loquacity. Presently, however, he becomes conscious of a pain which seems to have its seat in some particular region of his body. Upon this he at once concentrates all his attention, and he is now a "hypochondriac."

The part to which the morbid sensations of hypochondriacal patients are most commonly referred is, as might be inferred from the name, the upper part of the abdomen. The disease is one which has retained the same appellation since the days of Hippocrates and Galen. But they conceived the actual seat of the disease to lie in the viscera which are situated immediately below the diaphragm; and a similar hypothesis was intended to be conveyed by the equivalent English expression, "the spleen."† Indeed it is

* Ὑποχονδρίακον πάθος—Passio hypochondriaca—Passio atrabiliosa—Melancholia (in part)—Humor atrabiliosus—Milzsucht—The spleen.

† The first to vindicate the position of hypochondriasis among the neuroses was the celebrated anatomist Thomas Willis.

the fact that the digestive organs are generally more or less disordered. Dyspepsia is often present; the bowels are constipated; large quantities of gas are apt to accumulate in the alimentary canal, giving rise to flatulent belchings and noisy rumblings in the abdomen. In all probability the starting-point of the morbid hypochondriacal feeling is really an impression proceeding from the viscera, for among the symptoms of disease of the stomach and of the liver, we shall find depression of spirits and irritability of temper. But what is peculiar to the complaint with which we are now concerned is the marked exaggeration of every local pain. Moreover, the region to which the pain is referred seldom, if ever, remains the same during the whole progress of the case. After having long dwelt upon a gnawing or burning pain at the epigastrium, which he felt sure must indicate cancer of the stomach or liver, the patient all at once loses this, and perhaps begins to suffer from symptoms which, if due to organic disease, would point to the throat as its seat. Or he is attacked with dyspnœa and palpitation of the heart, and feels convinced that he is labouring under serious cardiac disorder; or a slight cold leads to a cough, and he begins to collect the sputa, is certain that he is phthisical, and consults the physicians who have the greatest reputation for pulmonary affections; or he finds himself giddy, experiences a sensation of weight and pressure in the head, and forthwith thinks of nothing but of the apoplectic fit which he believes to be impending. But perhaps the most miserable of all hypochondriacs are those who refer their sufferings to the genital organs. To this class belong by far the larger number of those who consult one for spermatorrhœa, or for impotence. They complain that they have dragging pains in their testes, that these organs always hang too low, that their urine is turbid and must contain semen, that they have emissions at night, during defæcation, and at other times. One sees at once that their whole attention and thoughts are concentrated upon the sexual function; and, to make matters worse, they are sure to have studied with avidity a number of books and pamphlets, written for the very purpose of stimulating their apprehensions, and of inducing them to seek relief from the quacks who write them.

If further evidence were required to prove that the disease is a neurosis, it would be afforded by the close relation which can be traced between it and other nervous affections. This was especially insisted on by Anstie, who maintained that the hypochondriac almost invariably belongs to a family of which other members have been of unsound mind. Unlike hysteria, hypochondriasis is markedly *hereditary*. As to whether the disease tends to pass into insanity in the same individual there is much difference of opinion. Melancholia is the form which bears the closest superficial resemblance to it; but Romberg points out that this is characterised by a tendency to self-negation, whereas in hypochondriasis the whole attention is concentrated upon personal feelings and sensations, and he goes on to show how the difference is expressed in all the patient's relations with other persons. A person affected with melancholia treats his medical attendant as an enemy or as an ignorant pretender, and constantly tries to avoid him; but the hypochondriac looks up to him as his guardian and saviour. He is constantly on the search for new plans of treatment, and expects that each fresh one will relieve him of his complaints. As Romberg puts it, "The more physicians, the better he is satisfied; he likes to change them as often as he would change a poultice. Yet, in spite of all, he is not unhopeful, and is never weary of life. A tendency to commit suicide is no part of hypochon-

driasis ; and any doubtful case in which such an attempt is made may be safely set down as one of actual insanity. Probably the same may be said of every case in which the patient has distinct delusions, as (for example) that he is made of glass, or that the devil is playing a barrel organ in his belly, or that the bowels are about to escape through the abdominal parietes, or that he has swallowed spiders, and that they are multiplying within his body. Griesinger, though so great an authority on mental disorders, seems to have entirely mistaken the boundary line between hypochondriasis and melancholia ; the cases which he relates as examples of the former are almost all of them really instances of the latter disease. This perhaps explains the circumstance that he found hypochondriasis "extraordinarily frequent in young people," and occurring sometimes in the years of childhood ; whereas Gull and Anstie express the general opinion when they say that it is scarcely ever seen under the age of puberty, although it very rarely makes its first appearance in anyone who is more than fifty years old.

A point which must always be kept in mind is that a person labouring under well-marked hypochondriasis may also have organic disease—of the stomach, for example, or of the liver, or an aneurysm of the aorta. Many a patient has had his complaints made light of, until the signs of one or other of these diseases have become too manifest to be overlooked, or until he has died suddenly, from rupture of a large vessel, or from angina pectoris. Anstie further remarked that in certain cases the possibility of malarial poisoning or of chronic alcoholism must be thought of, before one sets down a patient's symptoms as the result of mere hypochondriasis.

The *treatment* of this disease is commonly difficult and unsatisfactory. An essential point is that one should not attempt to make light of the patient's sufferings, but should show oneself to be really interested and anxious to relieve them. Exercise, short of severe fatigue, should be taken daily, and mental occupation and amusement should be cultivated with the greatest care. Sea-bathing, the cold water cure, the warm baths of Gastein, may each be prescribed with advantage ; but it is difficult to say how much of the benefit is due to change of air and scene, and alteration in the habits of life. Any disorder of the digestive organs must of course be carefully investigated and corrected. The continual exhibition of laxatives are injurious ; it is better to depend on diet and exercise to procure a healthy state of the bowels. But an occasional blue pill and black draught is most valuable in many cases and may be repeated once or even twice a week. Valerian is said to be sometimes useful, and so is with some patients tincture of sumbul. Neither quinine, strychnia, nor phosphorus is generally of service, nor even iron, unless there be marked anæmia. Anstie rightly laid stress on the importance of not prescribing alcohol for hypochondriacal patients, even in the form of medicinal tinctures, lest by doing so one should encourage habits of over-indulgence in drink.

In weighing beforehand the probable success of treatment in a case of hypochondriasis, one has to take into consideration not so much the apparent severity of the symptoms as the length of time they have lasted and the circumstances under which they began. The younger the patient the better the prospect of cure ; particularly if the family history is good, so far as regards the occurrence of insanity or other nervous diseases in his near relations.

PSYCHOSES

OR

DISORDERS AFFECTING THE MIND

(*By G. H. SAVAGE, M.D.*)

Insanity due to (1) *disorder of functions of brain;* (2) *the expression by the nervous system of bodily disease;* (3) *the expression of disease of the brain—Classification—Causation.*

Acute delirious mania—Acute and other forms of ordinary mania—Melancholia with bodily complaints—Hypochondriasis with mental complaints—Mental melancholia—Stupor, active or passive—Delusional insanity—Hallucinations of the senses—States of mental weakness—by defect—by instability—Specially named varieties of insanity: puerperal, alcoholic, moral, syphilitic, gouty, plumbic, febrile, phthisical, asthmatic, cardiac, renal and diabetic—Epileptic insanity—Paralytic and tabid insanity.

Testamentary capacity—Certificates and other medico-legal points.

Idiocy and imbecility.

In approaching the study of insanity, it is first of all important to remember that we have not a definite disease, such as typhoid fever or phthisis, to investigate, but that the condition or state called insanity depends, not only upon the evident symptoms, but upon their bearing on the social environment of the individual. There is, in fact, no absolute standard of sanity, and the divisions of the condition called insanity are to a great extent arbitrary. Insanity will have to be here considered chiefly from its clinical or medical aspect, but yet its social and its legal aspects must not be entirely neglected. Though there is no standard of sanity, yet it so happens that disorder of the nervous system expresses itself more or less along definite lines, and we shall have to point out the pretty regular association of symptoms as they occur in the different groups of insanity.

We must first recognise that while some cases of insanity depend upon cerebral disease or degeneration, others depend upon diseases of the body, which are referred to the nervous system; so that there is in the latter cases a mental aspect to a bodily disease. Thirdly, we shall have to consider the most difficult of all cases—those in which there is disorder of function without any visible pathological change in the nervous system. So, then, we have (1) disease of brain with disorder of its function; (2) disease of some function or functions of the body with insane or nervous interpretations; and (3), disturbance or disorder among the functions or parts of mind.

While recognising *forms* of insanity, we must remember that these forms shade insensibly into one another, that no symptoms are absolutely characteristic of any one form of mental disorder. The *forms* are, then, arbitrary,

but more or less convenient, grouping of symptoms. The brain and nervous system have their special ways of expressing disease, just as the lungs, the stomach, or the kidneys have their special symptoms or ways of expressing theirs. We must look for the chief symptoms of insanity in either perversions of sensations, perversions of will, perversions of emotions, or loss of control or disarrangement of one or more of these parts of mind. The factors of mind for our present purpose will be considered to consist first of all, and perhaps most necessarily, of the inherited nervous system of the individual—the nervous basis—to which is added the experience gained by the various special senses—*perceptions*—and these bound together and arranged by *memory* form a structure upon which the *will*—balance of motions—and the *emotions* are supposed to play. And in each case of insanity it is well carefully to inquire first into the inheritance, next into the normal or abnormal working of the parts of mind as evidenced by the psychical and muscular expression. In considering the forms in which insanity appears, it will only here be necessary to make a few characteristic divisions, as the following:

Melancholic states, in which there is mental and bodily weakness and excessive self-consciousness.

Maniacal states, in which there is weakness with loss of control.

Dementia, in which there is weakness with more or less evident defect in mental function.

Beside the above, there are states due to the misleading by false sense impressions. These have been grouped under *Delusional insanity*.

The above are the more common groups into which symptoms of insanity form themselves. But these symptoms may occur in two distinctly opposite pathological states. They may occur as symptoms of disease of the brain, as seen in the degenerative process called "general paralysis of the insane," or they may occur in what we have seen to be the second and third groups of insanity. In fact, insane symptoms, whether they occur with brain disease, or bodily disease, or a disorder of mental function, express themselves very much in the same way. Just as one may meet with cough which is due to disease of the lungs, cough which is due to a spasmodic condition, as in asthma, and cough due to defects in cardiac circulation, so we may have delusion due to organic disease of the brain, to bodily disease, as bad supply of blood, or to disorder of function, as seen in false sensory impressions.

Insanity occurs among the civilized and uncivilized, but in its more marked and serious forms it is specially a disease of the more highly civilized, and it will be seen that the more fatal forms of brain degeneration seem to increase directly in proportion to the high pressure of present life. Insanity is not, however, rapidly increasing in England. The statistics of the Commissioners in Lunacy show that whereas in 1875 there were 24·75 males, 28·43 females per 10,000 of the population in England and Wales insane, there were in 1885 respectively 26·61 males, 30·80 females per 10,000 in England and Wales insane, so that the increase is only about two per 10,000; and when we consider the greater general longevity and the consequent accumulation of chronic cases in asylums, it is pretty clear that there is no such increase of insanity in England and Wales as to cause real alarm. In further evidence of this the Commissioners' reports show that the admissions of fresh cases in 1886 were really lower than the admissions of fresh cases ten years before. In fact in 1875, while 5·36 men and

5·18 women per 10,000 inhabitants were admitted as insane patients, ten years later 4·8 and 4·9 respectively only were admitted.

In considering the so-called *causes* of insanity, the student must be warned that it is extremely rare to find one definite cause producing any attack of mental disorder, and it is therefore better to use the term *conditions* rather than causes of mental disorder. Again, though it is perfectly true that certain vital conditions, such as race, stage of civilization, climate, and the like deserve to be considered as conditions predisposing to insanity, yet, in a short chapter such as the present, it is beyond our scope to consider more than some of the general conditions which give rise to mental disorder. These causes may be divided for convenience into those acting chiefly upon the nervous system, and those acting chiefly upon the body—the so-called *physical* and *moral* causes. These again can be divided into *predisposing* and *exciting ;* and though some causes act distinctly as moral causes, yet it will be found that in the majority of cases one cause may be both moral and physical, and may also act as a predisposing and as an exciting cause. Take, for example, alcoholic stimulants, which act primarily as a physical and predisposing cause ; but the associations of the drunkard are likely to lead him into moral troubles, which again react, and a fresh outbreak of drink and excitement may be the real exciting cause of the outbreak of insanity. The most common *moral* causes are mental anxiety, worry, overwork, money losses, religious excitement, love affairs, domestic trials, fright, and occasionally other emotions. Among the *physical* causes, intemperance, organic disease of the brain, injury to the head, venereal excesses, masturbation, epilepsy, pregnancy, parturition, and lactation are the more common causes assigned in England, and, in addition to these, one of the most important conditions of insanity—*heredity.* It is extremely difficult to be sure of the part played by inheritance, yet it is sufficiently certain that a large proportion of people are insane because of the insanity of their parents. The insanity which is passed on from parent to child has special peculiarities and will have to be referred to more in detail later on. There seems in such cases to be liability to disorder of function rather than to disease of the brain itself, and there also appears a greater predisposition to recurrence in cases with strong neurotic inheritance. Besides direct inheritance we have to remember that insanity may be produced in families where epilepsy, hysteria, hypochondriasis, some spasmodic nervous affections, and the like occur ; and it is probable that if to these conditions in one parent be added any special cause of organic degeneration in the other, the offspring will be rendered more unstable, that is, more liable to fall out of step with the other members of a civilized society.

Causes of insanity act directly, *i. e.* immediately, only in very rare instances. The mischief is much more commonly the result of frequently recurring disturbance, and therefore in considering any case of insanity with reference to causation it is important that changes in temper, disposition, and habit should be carefully investigated. It must be understood that causes in the majority of cases are not easy to be traced, and in many cases the supposed *causes,* such as sleeplessness and dyspepsia and even alcoholism, may really be the earlier *symptoms* of the disease.

It is necessary to refer to a few of the general conditions in more detail. In cities degenerative changes and general paralysis are most common, whereas the latter is almost unknown in peasant races, such as the Celtic population in Ireland, Wales, or Scotland. Education is a cause of insanity, if ill-suited

to the nature of the person, but over-education alone is very rarely so. More women than men become insane, but more men have general paralysis and more women recover from insanity. Puerperal and climacteric conditions affect women. Insanity of any form may attack patients of any age, but in childhood the tendency is to idiocy, in early youth to imbecility, in youth to weak and impulsive mental states; in middle life the greatest amount of acute insanity occurs, as well as most general paralysis, at the climacteric melancholia and delusional insanity are frequent, and as age increases the tendency is to dementia.

Solitude and sedentary occupations followed apart are very dangerous conditions; hence widowhood is of evil influence. Domestic worry chiefly affects women, and business worry and anxiety men. Love affairs and sudden shocks, whether painful or pleasant, affect mostly women and very young people. All causes producing excessive nervous exhaustion affect the young most; as do also the abuse of stimulants and chronic poisoning, such as that by lead.

Injuries are not frequent causes of active insanity, but lead to diseases of degeneration. Tumours and coarse lesions of the brain, syphilis excepted, are also rare causes. Religious and political excitement act mostly as exciting causes. Fevers and any causes of delirium may start insanity.

Bodily diseases causing pain or weakness may set up nervous disorder. Insanity may depend on bodily disease, or on physical disfigurement and its moral effects. Insanity may alternate with other nervous diseases, such as epilepsy or hysteria, or it may alternate or replace other nervous or bodily diseases, such as asthma, gout, rheumatism, eczema.

ACUTE MANIA.—By acute mania we mean a state of mental weakness with marked loss of control. Under this head we shall have to place a variety of disorders extending from delirium to simple hysteria, and it will be convenient to make two distinct groups of the maniacal cases.

There is a well-recognised and well-understood condition—delirium—and formerly it was insisted that there were essential differences between mania and delirium, but it is certain that there are connecting links. There are cases in which there is some delirium but much maniacal excitement, and others in which there is little maniacal excitement and more delirium. It is well, then, to remember that when speaking of patients suffering from mania, we may have to consider either those in whom there is great bodily and mental disturbance or those in whom there is great mental disturbance with little or no bodily disease.

Acute delirious mania is a term used to express the symptoms occurring in the cases of mania in which the delirious element is most marked. Typhomania, brain fever, and other terms have been used, but it seems that we still are in want of some general descriptive term which will include all the cases which deserve to be grouped together, because, though most of these delirious cases are more or less maniacal, yet there are examples in which all the other bodily symptoms are present, but instead of mania there is either melancholia or partial weak-mindedness, or at least confusion of thought. Acute delirious mania is a disease most commonly met with in young people of both sexes. It is common in the highly nervous, the accomplished and educated. It is generally ushered in by a slow process of change in temper, or by general feelings of uneasiness, restlessness, malaise, or hypochondriacal symptoms. It is common to meet with young women

who have had some disappointment in love, some shock, or who have suffered from some physical disorder, producing nervous weakness or anxiety; they complain of sleeplessness and headache, loss of appetite, amenorrhœa, and general inability to apply themselves to ordinary occupations. This may be the early stage of acute delirious mania. Acute delirious mania may also follow shock, fevers, such as scarlet fever, measles, and the like; poisoning, as from belladonna, from intemperance in alcohol or ether, and in my experience it may also follow the inhalation of chloroform. It is not confined to young people. The period of depression may be followed quite suddenly by one of excitement, exhibited by voluble talking, exaltation of ideas, or not uncommonly by a rather sudden and startling development of erotic excitement. Within a few days the delirium has reached its height: the patient then has the appearance of one suffering from typhoid fever, lying on her back with a tongue dry and brown, or thickly coated, with sordes on the lips, the eyes suffused, the cheeks either sallow or (during periods of excitement) brightly flushed, the lips moving without any audible result, the hands twitching or picking the bedclothes, the voice, if heard, harsh and raucous, the skin moist, with little or no increase of temperature; then rapid emaciation, tendency to bedsores, and excitement recurring irregularly, but most marked at night, with little or no reaction to the surroundings. There may be hallucinations of sight and hearing. There may be periods of tranquillity and apparent recovery followed by periods of excitement. During the period of excitement there seems to be no memory. Patients recovering have little or no recollection of what has happened during their illness. It is almost impossible to say how long the period of excitement will last, but from a few weeks to two or three months is the limit, and it must be remembered, that patients suffering from acute delirious mania are passing through a process in which exhaustion is the chief characteristic, and the excitement is invariably followed by a more or less prolonged period of depression, the depression not being that of melancholia, but rather that of inability to will or to desire, so that they are in a will-less, childish condition. From one third to one fourth of the young cases suffering from acute delirious mania die, and of the others probably a full third remain permanently weak-minded. Some rapidly gain flesh, but remain dull, apathetic, and unlike their former selves.

The chief points in *treatment* are to feed freely and abundantly. It is well to begin with a free purge by means of calomel or croton oil, and then to follow up with beef-tea, mutton broth, milk, brandy, and eggs. It is almost certain that artificial feeding will be required. Feeding by means of a nasal tube—or better, by putting a funnel in one nostril, and, while controlling the other, pouring the fluid food down the nostril—will be found of great service in feeding these cases. The feeding by a stomach-pump, by the rectum, or by the nose, must be repeated every three or four hours, and it is of the utmost importance that stimulants should be given without stint. In old days it was feared that with such apparent excitement of the brain stimulants would have an injurious effect, but all modern experience is that stimulants, instead of causing excitement to these patients, are more likely to produce rest. In many cases the excitement is so extreme that some form of restraint, medical or mechanical, must be used. Chloral hydrate, in doses of from fifteen to thirty grains at night, and repeated in ten-grain doses two or three times a day if necessary, may be tried, but it is better to do without any narcotic if possible. In

strong, young, active people the wet pack, *i. e.* the rolling the naked patient in a sheet wrung out of tepid water, and then rolling one or two blankets outside this sheet and placing the mummified patient on the bed for from one to three hours, will produce rest and quiet, but this treatment should be carefully watched by a skilled nurse, and during the process food and stimulant should be given. In some cases in the earlier stages warm baths with mustard in them will produce a stimulating effect upon the skin and secure a good night's rest. The great point to remember is that patients suffering from acute delirious mania are suffering from a disease which as a rule either rapidly kills or rapidly passes off, leaving the patient much exhausted, and as soon as the acute attack has passed it is of the utmost importance that they should have careful nursing, without much change in their surroundings. After the illness rest and quiet in the country or by the seaside is preferable, in the writer's opinion, to travel; and it should be remembered that in such cases intellectual work should not be undertaken for a twelvemonth from the onset of the disease.

Complete or only partial recovery may take place; the patient may be left more or less morally or intellectually crippled, and in some cases not only is there defect of mind, but, following the acute delirium, some more or less permanent wasting of the lower extremities may occur, and the writer has met with three or four cases of delirious mania in which contraction of the muscles of the calf has remained behind.

There is no very marked tendency to the recurrence of attacks of delirious mania. In fact delirious mania differs in that as it does in other respects from many forms of ordinary acute mania.

Ordinary Acute Mania.—In this condition, unlike the last, we have more organised expression of delusion and more marked loss of self-control with less bodily disturbance. The conditions under which maniacal excitement appears are endless, depending much upon the inheritance, education, race, and other peculiarities of the individual. We meet with every variety, from simple, uncontrolled lust to violent homicidal destructiveness, and no one description can fully cover the whole line. Mania may occur as only part of a morbid mental process; it may follow a melancholic stage, or epilepsy; it may be part of *folie circulaire* or recurrent attacks of insanity; it may be part of the symptoms in general paralysis of the insane or of dementia due to age or definite brain disease. But in all cases the symptoms are sufficiently alike to deserve our description.

The *causes of mania* are in no way special. Mania may follow from moral or physical causes, may follow injury to the brain, direct or indirect or may follow upon bodily disease or disorder. A shock may produce either mania, melancholia, or dementia.

Nearly all cases of mania begin with uneasy feelings about the epigastrium, occasionally with feelings either of emptiness or fulness of the head, rarely with headache or photophobia. Sleeplessness and fear of impending ruin are common, anorexia, constipation, restlessness, inability to apply themselves to work, irritability and emotional weakness. This period may last from a few weeks to several months, and may become extremely marked and profound. After the period of depression the patient may almost suddenly grow more restless, more excited, and markedly loquacious. He may say that he has been ill but that now he is quite well, and he may become extravagant, boisterous, and over-generous. He may

discover that he does not require to sleep so much or to eat so much as he did, and he may be full of ideas that he will be able to make a fortune by some invention. Any interference by his friends is resented, and in many cases such early interference precipitates the attack of mental excitement. Sleeplessness of a restless type is marked, appetite is variable, a craving for stimulants may occur. The bowels as a rule are confined, the skin is often sallow but of normal temperature, hallucinations of sight and hearing are not uncommon in the earlier stage, delusions as to property, wealth, position, and also as to the actions of friends, are common. Will as a rule is unstable, so that the patient is incapable of persistent effort. He is often emotional and irritable, but his memory is so far good that he remembers what is said or done, though things appear to him different from what they would in health. Sexual desire is frequently great, but there is probably no increase of sexual power. The general bodily condition is one of weakness, and the general mental condition is one of instability and mobility.

The two things that one has to guard against chiefly are the reduction of strength and any local or general injury to self or others, and the question of treatment depends almost entirely upon the question of danger to the patient or his friends. Mania may end fatally from sheer exhaustion; this occurs mostly in elderly patients or in people exhausted from some general bodily disease. A large number of maniacal cases get well, but, a certain number remain either permanently weak-minded or permanently unstable, so that they are constantly liable to recurring attacks of insanity. Of those who do not sink in the earlier stage, the period of excitement may last for weeks or months; probably four to five months is an average period for acute maniacal excitement to persist. During this period there will almost certainly be intervals during which the patient is more quiet and apparently improving, and it is noteworthy that after a night's sleep, produced either by narcotics or as a result of exhaustion, the patient may appear to be improving, but, we must be prepared for remissions and relapses in the course of ordinary acute mania. During the maniacal excitement destruction of clothes and filthiness of habits are very common and distressing. Each individual case exhibits peculiarities in its course, symptoms, and termination. One patient will suffer from some bodily ailment before the mental storm appears to pass; one will complain of headache, another of neuralgia, one of rheumatism, and another of restless sleepless misery connected with some trifling bodily ailment before the disappearance of the excitement. As a rule the symptoms disappear slowly, it being noticed that the patient is getting more self-control, has greater desire to see friends or relations, writes letters home, is anxious to see to his business, and very probably may be discontented with his situation. Discontent in persons suffering from mania is equivalent to the returning consciousness of a man who has been stunned; and just as in the latter case there is for the patient himself a painful consciousness, while for the onlooker there is the satisfaction of sensibility returning—so also discontent in the maniacal patient often means returning consciousness. In proportion as the patient has been excited will there be depression or mental exhaustion; and it is pretty certain that a prolonged attack of mania will be followed by a prolonged attack of mental exhaustion or depression, during which there may be more or less sleeplessness and loss of appetite, with general mental weakness; but in these cases change of surroundings and as speedily as possible a return home will be found the best treatment.

From a practical point of view, all powerful narcotics and depressants are merely means to keep a patient within control, and not means of really curing our insane patients. It will be necessary in some cases to try chloral, bromide of potassium and the like, and it undoubtedly in a few cases will be found that bromide of potassium alone or combined with chloral will lessen the excitement and give time for the patient to recover. But the most important consideration is that, whatever treatment is followed, we must be sure not to affect the appetite or we shall have cause to regret it. Abundant nourishment, with or without stimulants, associated with exercise in the fresh air and as much freedom as is possible, should be the treatment for such cases.

Before leaving the consideration of this group, we must mention the patients who have suffered from acute mania and who never permanently recover. Some, as already stated, remain ever after liable to recurrence of insanity, others remain weakened in some special way so that they are unable to fulfil their duties as before. Thus, one individual will be morally weak and is called either a kleptomaniac or a drunkard, another becomes emotionally weak and takes to ambitious schemes for reforming his fellows or to spending day and night in religious exercises, a third becomes volitionally weak and allows himself to be looked after and attended to by his friends without any desire to help himself.

It is only necessary to add that, if the antecedent stage of melancholy be long, the maniacal one is likely to be prolonged also.

After epilepsy very violent mania is frequent, and chloral given by the mouth or the rectum may check the mania if administered early after the fit. Cases in which the mania depends on senile changes are less favourable but not hopeless.

Maniacal excitement due to general paralysis generally passes off in a few weeks. Mania due to alcohol is very variable, but may last for weeks or months.

MELANCHOLIA.—By the term *melancholia* we mean a state of unreasonable mental depression; grief without a real cause, depending rather upon a physical than a moral determinant.

Melancholia may be but part of the disordered process or may be the whole of the morbid state. In most cases of acute mania and in most cases of general paralysis of the insane there is a stage of melancholia which ushers in the acute symptoms. Melancholy as a form of mental disorder occurs in all ages, though probably it is most commonly met with in and after middle age. It is most common in the dark complexioned. The symptoms of melancholia may depend on general depression of the vital powers, or it may depend upon some special diseased process, as, for instance, heart disease. The most characteristic symptom at the onset is extreme self-consciousness. This develops into grief which is variously expressed; irritability of temper, sleeplessness, loss of appetite and suicidal tendencies develop in order. Melancholia has been divided into many varieties. The chief methods of division depend upon the outward expression of the morbid grief. In some the symptoms are *active* and in others they are *passive*,—in the former the patient restlessly wringing his hands and openly complaining, while in the latter he is struck dumb by his misery. Melancholia is divided not only by its expression but by its special aspects: in one the expression of grief is in relationship to the bodily functions, while in the other the mental symptoms predominate. The former may be called *hypochondriacal*

melancholia, and the latter *true melancholia*. Every shade of melancholia may be seen in relationship to hypochondriasis, but in an asylum we see the more exaggerated cases which must be secluded in consequence of their desire for death and refusal to take food.

Hypochondriacal melancholy.—There are four distinct groups of cases which come under this head. First, those of patients whose one complaint is that they are dying and that nothing can save them, without their making a definite complaint of any special organ.

Next we have a large group of cases in which the symptoms are referred to the *head*. Thus young patients will tell you that their brains are hot or wasting or hollowed or melted, and women about the climacteric will complain of opening and shutting of their brains, of something coming away when they move their necks, or the like. Older patients will complain of adhesions between the brain and the skull, or of miraculous removal of brain and nerve power.

The third group includes those who believe that some terrible calamity has befallen their *digestive tract*. One patient believes that his throat is stopped: these symptoms are allied to exaggerated hysterical globus. Another is sure that his stomach is ruptured or imperforate, or that some connection exists between the stomach and the circulatory system. A few patients imagine that the rectum is permanently closed. All these cases have to be looked upon as exaggerations of hypochondriasis, cases in which every hypochondriacal symptom is greatly developed.

The last group of insane hypochondriacs contains those who believe that some trouble arises from their *reproductive organs*. Middle-aged men believe that they have been rendered impotent, that their testes have withered, or that by some evil habit or by some course of medicine they have become emasculated. Such cases are more commonly met with among men than among women. It is comparatively rare in asylums to find women with uterine and ovarian hypochondriasis. There are young sexual hypochondriacs whose disorder is partly due to masturbation, and these have varying disorders of the brain.

In all cases of hypochondriasis the one object of *treatment* must be the withdrawal of the attention as much as possible from the sensitive part by occupation, while the general health is being improved. Judicious change of air, change of scene, travelling and mechanical occupation, with withdrawal from old associations and companions, are of the utmost value. Such cases recover in a fair proportion, but if past middle age, or if the development of morbid ideas have been slow, and if there be no definite physical illness associated with the mental disorder, the prognosis is bad. Such patients rarely become weak-minded, but persistently hold to their hypochondriacal delusions, which may remain for many years, the rest of the intellectual life of the patient being normal.

Ordinary melancholy.—Ordinary melancholia consists in emotional depression, in which the explanation of the feelings is moral and not physical. It may be the result of shock, or grief, or bodily illness. In certain predisposed persons any cause which reduces the vital standard below a certain point seems to be able to produce melancholia. The first symptoms are, as a rule, mere uneasiness, restlessness with inability to take interest in surroundings, tendency to tears and emotional disturbance, dread of some

impending calamity, with undue sensitiveness to the conduct and the remarks of others. These symptoms are associated with loss of appetite, sleeplessness, and feeble circulation, as seen in winter by chilblains and cold extremities, together with loss of appetite, constipation, and amenorrhœa in women. These evidences of reduced power are all part of the process, and may rapidly pass into most pronounced mental depression. Up to this time there has been a feeling of misery, but there has probably been little or no explanation as to its cause. Later hallucinations of the senses or delusions may arise, and these depend to a great extent upon the education, age, and immediate surroundings of the individual, so that the young woman thinks that her virtue is called into question, while the old man thinks that he is going to the workhouse. It is common to hear patients in this condition refer to their past wickedness, and to say their souls are lost, that they are possessed by the devil, that they are "the Scarlet Woman," that they are metamorphosed into beasts, or are unnatural, and that they ought to be dead. At this time they will be sleepless, and probably will refuse to take food voluntarily. There will be complete inaction of the bowels and general apathy and listlessness; suicidal tendencies become marked, and it is important to remember that patients as a rule select a special form of suicide which they prefer, and will wait for an opportunity rather than seek death by any other means. Suicidal attempts are mostly to be feared when patients believe themselves to be impotent, when they believe themselves to be injuring their nearest relations, or when they dread being tortured "to make them confess," or when they believe themselves to be followed and dogged about. Patients who "hear voices" are also very liable to suicide. The melancholic process is, as a rule, longer than the maniacal one, and an average of from six to eight months is the period for most curable patients. The symptoms, after slowly increasing, may slowly recede, the whole prognosis depending upon the steadiness of the improvement both in mind and body. In some the cure is sudden after sleep or after some sudden improvement in general health, as improved digestion or the like. Perfect recovery may take place, or, what is more common, there may be a slight excess of excitement after the melancholia has passed away. There is a great tendency to relapse in melancholic patients, especially in those belonging to an insane stock. A certain number of patients die from melancholia, but as a rule the cause of death is secondary to the mental disease, there being some congestion of the lung or extreme exhaustion and emaciation depending upon bad nutrition or insufficient feeding. In cases of chronic melancholia wasting of the brain is well marked, and the pia mater is sodden and easily separable.

The *treatment* of melancholia naturally divides itself into preventing harm and doing good. The first essential is to prevent suicide, and this is only to be done by constant watching, by the utmost care in the selection of nurses, and by insisting on the patient having his rooms on the ground-floor. Rest in bed, especially during the colder months, is valuable, warmth and food, with stimulants, being very important. In some cases of active melancholia morphia may be useful. When patients are treated at home, it is necessary to give narcotics, the rule being not to give them continuously, to avoid giving them in increasing doses, and to vary them as much as possible.

The next most important thing is the method of feeding. Patients who refuse their food absolutely may be fed by the rectum, by the mouth, or by the nose. As a rule, patients who resist should not be fed by enemata. In feeding by the mouth, if sufficient care and time be taken

in nearly all cases food may be given by the spoon being introduced by the side of the teeth in small quantities. But if resistance be great it is better to pass a nose-tube or the stomach-pump. The nose-tube is very easily passed, and nurses can be trained in a few lessons to do it without danger; a large-sized soft catheter fixed on the end of a small glass funnel is all that is required, the tube being passed down one nostril, while the other nostril is compressed by the finger. Three to five pints of milk, six eggs, two pints of beef-tea or broth, and four ounces of brandy, may be given in twenty-four hours.

Among the forms of melancholia the most important are *simple melancholia*, that is, melancholy without delusions, *melancholy with stupor*, to which we shall refer again, and *active* or *resistive melancholia*, in which an active expression of grief occurs. There is no special form of melancholia deserving the terms "religious" or "suicidal."

Simple melancholia is often present in men of middle age and of active intellectual habits. Misery without cause, self-acccusation about trifles, sleeplessness and loss of appetite, inability to apply the mind or the attention, are the chief symptoms.

A month's rest under supervision, away from wife and friends, followed by restful and recreative travel, are all that is needed, but probably twelve months' rest from mental labour will be needed. This state may occur in much-examined youth, in much-worked or worried manhood, or may appear at the climacteric or decline of life. Suicide is common. There are other varieties of melancholia associated with bodily disease. Thus, undeveloped or suppressed gout may be represented by melancholia, and phthisis may show itself only as progressive weakness and refusal to take food, with suicidal impulses. Melancholia, if of an active type, often benefits if morphia be given, and in young cases shower baths are of service. In gouty and simple cases a course of Turkish baths will often work wonders.

STUPOR, INCLUDING ACUTE PRIMARY DEMENTIA AND MELANCHOLIA WITH STUPOR.—Under this one head two very distinct groups of cases are included, but in both of these the characteristic symptoms are the stupor, the silence, and the abstracted appearance of the patients. According to some writers there is no such thing as acute primary dementia; they hold that all these patients owe their mental attitude to some dread or terror. In fact they believe such patients to be in a kind of chronic panic, but it will in any case be necessary to distinguish between two groups occurring under this head, and for convenience we will call them the active and passive groups.

Active Stupor.

Common in adolescents, who have a terrified expression, are wasted and thin, with skin purple and cold, resist or are cataleptic, refuse food, are sleepless, wet, and dirty, have distinct delusions of dread, retain memory through the attack, refuse all food and require abundant forced feeding with stimulants; electricity and massage may be useful.

Passive Stupor.

The passive form occurs more frequently as the result of some cause of exhaustion in young or middle-aged people, who have a silly aspect, muscles are flabby, skin greasy, limbs relaxed; they eat what is given them, sleep well, are clean if watched; there is no evidence of the existence of delusions, little or no memory on recovery; and for treatment they require baths, exercise, and general tonics.

The above groups resemble one another in history and in progress to a great extent; most of them occur in young adolescents. It seems as if in

certain weak-minded families there is energy enough for boyhood or girlhood, but when manhood and womanhood comes on it fails. With such cases shock, grief, masturbation, disappointment, overwork, unhealthy occupations, fevers, intemperance, rapid childbearing, or the like, may suffice to produce the breakdown. When once the condition of stupor has been established, as a rule it persists for several months, though occasionally there are recurrences of stupor between intervals of sanity. Little or no good comes to these cases unless the general health can be markedly improved. The patient, if in active stupor, is probably more or less cataleptic, and has to be forcibly fed. He wastes, and is wet and dirty, is very liable to secondary inflammation, and often has chilblains. He takes no notice of his friends. The stage of stupor is of very variable duration and may end in death, in slow or sudden recovery, or in partial weakness of mind.

If in a state of mere passive stupor the stage may be very persistent, but commonly is associated with periods of temporary recovery or of periods of excitement, or with sudden outbreaks of impulsive violence. This passive state frequently results from or follows an acute stage of mania, or develops after some severe and exhausting bodily illness.

Feeding, warmth, and watching are of great service. One danger in such cases is death from some secondary complication; local inflammation or phthisis may develop and terminate rapidly. After recovery there is frequently a period of exaltation during which sexual desire and tendency to lust and intemperance may arise. The prognosis depends greatly upon the family history. If neuroses are common in the family, and if the patient is very young or badly developed, or if he have a badly shaped head, or any tendency to phthisis, the prognosis becomes absolutely bad. If a patient recovers from stupor it is very necessary that for some years he should be carefully watched, and should not return to any occupation which involves severe strain either of mind or body, so that it is much better after recovery from stupor to send the patient for one or more sea voyages, or, if possible, to let him follow some mechanical or outdoor life.

Delusional Insanity.—So far we have considered cases in which there have been perversions of the whole mental functions associated with more or less bodily disturbance, but under this head we have to consider cases in which the perversion of the senses is the chief symptom. The expression "out of one's senses" is recognised as meaning madness, but only some patients are definitely out of their senses. Such are the cases now to be described. It can readily be understood that as our intellect depends to a great extent upon the impressions we receive and store up from our senses, if our senses mislead us the intellectual result will differ greatly from that of the ordinary standard. The cases which I have to describe depend chiefly upon hallucinations which are sense impressions not depending upon any external or objective sensation. Thus a person who in the stillness of the night hears "*voices*" has hallucinations of hearing, and the person who, similarly situated, has *visions* or smells *poisons* or tastes *filth* is suffering from hallucinations of the senses of sight, smell, and taste respectively. Hallucinations of hearing are the most common, next in order we have hallucinations of sight, then in order those of touch, taste, and smell. Nearly all hallucinations may be reckoned as painful nervous impressions. The explanation of the sensation which is given depends to a great extent upon the education and occupation of the individual.

Patients suffering from *delusional insanity* are generally past middle life, single or widowed, often of insane family, leading solitary lives or fulfilling positions which are uncongenial to them in one way or another. Hallucinations have been frequently traced to solitary confinement in prison, and I have met with several cases in which they have developed in men living isolated lives in the wilder parts of India and other English dependencies. As a rule the earliest symptom complained of is nervousness and nervous weakness. They become excessively sensitive, and this leads to a condition in which everything seems to point to or to affect the individual; his mind is like the tender corn to the walker, and just as every movement seems to affect the latter, so every action of others seems to be directed against the former. He becomes suspicious, jealous, bad-tempered, more and more solitary, and this increasing solitude further develops the ideas of *suspicion*. He believes himself to be *persecuted* and *followed about*. He may think that his thoughts are read by others or appear aloud to himself and to others so that they read his secrets, he hears people coughing, jeering, or making allusions to him or to his family. He may hear the name of "devil," or worse, constantly whispered in his ears; he may then take the law into his own hands and assault someone whom he believes to be his persecutor. The ideas of interference may become more organised, so that he is convinced that bodies of men, such as the Jesuits or the Freemasons, are interested in his ruin. Under these circumstances the patient becomes exceedingly dangerous, and, unfortunately, it is very hard to break through the hallucinations and to appeal to him by means of any other ordinary sense impressions which might in time loosen the bands of the false sense impressions. If these symptoms have developed slowly for twelve months the prospect of recovery is slight. Patients who believe themselves to be followed or persecuted should be most carefully secluded or watched with the utmost care as they are both homicidal and suicidal. They are sometimes so exceedingly suspicious that they will deny their delusions so as to mislead those of whom they are afraid. Besides those who believe themselves to be persecuted there are others who believe that everything refers to them, and others who believe that the whole of their sense impressions are make-believes,—that the whole world is a kind of play which is being acted before them.

The general *treatment* of cases suffering from delusional insanity must depend very greatly upon the nature of the delusions, and the length of time which they have existed. Medical treatment is practically useless, but in some cases persuasion, associated with change of surroundings, works wonders. One young man believed that his relatives jeered at him from the roof, and that others made use of foul terms against him and them. This patient was excessively violent, yet by employing him, amusing him and bringing distinct irrefutable evidence that these people did not exist on the roof and did not accuse his friends of iniquity, we slowly brought him to be more amenable to discipline, and at the end of twelve months he was discharged so far recovered that he has since emigrated and is now in perfect health.

Chronic Weak-mindedness.—Loss of faculties, more or less general, may result in such extreme mental weakness that it is unsafe for the patient to be at large, as he would on the one hand be a tool for the wicked and on the other a prey to the vicious. Chronic weak-mindedness, as seen in

an asylum, may result from age, apoplexy, course of brain disease, or it may follow nervous exhaustion, such as general paralysis or tumour, in epilepsy or any other conditions of nervous or mental excitement.

Simple *weak-mindedness* is *gauged* by *loss of memory* and *loss of control*, and every variety may be seen, from the weak-minded imbecile to the lustful and dangerous, demented lunatic. The general course of ordinary weak-mindedness due to age and the like is as follows :—Progressive loss of control of the emotions, loss of memory and tendency to collect articles of no value on the one hand, while there is thoughtless and reckless extravagance on the other, a redevelopment of lust and a tendency to intemperance, which all lead to a wasteful expenditure of the reduced nervous capital. Often patients who have led reputable lives up to sixty may pass through every phase of moral degradation before it is discovered that their immorality was the result of arterial degeneration. Such cases require to be recognised and to be rather watched than sent to asylums, if the means are sufficient, because the probable termination of such cases is an apoplectic fit or permanent childishness.

As the subjects of chronic weak-mindedness we must include a large number of patients who, having suffered from acute attacks of mania or melancholia or stupor, instead of recovering their old mental balance, remain either *permanently crippled* or *permanently unstable*. The permanently crippled may exhibit their weakness by loss of one or more of the higher faculties, so that one becomes impulsive and homicidal, another ceases to respect truth and honesty and is called a kleptomaniac, another disregards decency and becomes altogether brutalized. In some a single faculty, such as memory, may seem to be affected out of all proportion to the others ; the most characteristic loss after all is that of the highest social relationships. Patients who have not perfectly recovered from acute attacks of insanity invariably lose their affection for near relations and friends, and pass into a condition of philosophical calm or social indifference. Beside the loss of faculty already described, there are cases of permanent instability. Every asylum contains patients who are subject to recurrent attacks of mania or melancholia, patients who during twenty, thirty, or forty years will periodically have recurrent attacks resembling in every particular the previous ones, with the probable exception that each attack is rather longer than the others, and leaves some more or less well-marked scar upon the intellect. Among these we have to recognise cases of chronic recurrent mania and of chronic recurrent melancholia.

One other group remains, and that is of those cases which, after an acute attack of insanity, have a few symptoms surviving, organised as it were, so that the patient becomes automatic, with certain peculiar actions or modes of thought fixed. Thus one patient has for sixteen years sobbed out hour by hour, "I don't know what to do," and yet there was no sorrow in the later years, the lamentations having become purely automatic, and the patient eating well, sleeping well, and becoming fat. In other cases with chronic chattering incoherence, there may be persistence of some hallucinations, so that the patient is living in two mental worlds, or perhaps it were better to say between them ; hearing faintly the real messages from the one, and more clearly the false impressions from the other. Many such cases, though violent at first, may be treated with sufficient safety at home, as is evidenced by the existence in so many country places of feeble-minded villagers, ready to run an errand or enter into boyish games. Such weak-minded persons may be trained to perform mechanical tasks sufficiently

well, so that the simple drudges of an asylum are often patients suffering from chronic feebleness of intellect.

So much for the permanently crippled. The permanently unstable are those who though fairly reasonable during most of their lives are constantly liable to fresh attacks. In some, the recurrences are at long intervals, and are comparatively harmless to the nervous system; in others the frequency or severity of the attacks causes progressive destruction of mind.

The symptoms may be maniacal or melancholic. Each attack as a rule resembles its predecessors in mode of development, symptoms, and mode of termination, but each attack may further impair the stability.

One patient may have an attack of acute mania and recover, a second attack occurs in four years, a third in three, a fourth in two years, and after that one or more attacks may occur yearly till the patient either passes into chronic mania or into hopeless weak-mindedness. In cases of recurrence, if maniacal symptoms are present, powerful drugs such as hyoscyamine in one tenth of a grain, given subcutaneously, may check the outbreak or the recurring habit, especially if the drug be given when the earliest sign of disturbance is noticed.

In recurrent cases of melancholia with suicidal ideas, with each recurrence suicide must be guarded against. Therefore such cases are always anxious ones if allowed to be at large.

With the unstable cases hereditary tendency is common. Such cases may live to an ordinary age. In some cases a weak-minded state gives place periodically to outbursts of violence.

Puerperal Insanity.—There is no special form of insanity deserving the name "puerperal," yet, as the puerperal conditions are not infrequently causes of insanity, the term Puerperal Insanity is convenient.

Insanity may be developed during pregnancy; it may be but an exaggeration of the longings of pregnancy, and on several occasions a distinct connection has been traced between some absurd or morbid appetite and a neurotic tendency.

Beside the mere longings of pregnancy and their insane development, one meets with true insanity occurring early in pregnancy. This is most common in women who have already suffered from some form of insanity before. Thus, a woman who has had one or more attacks of puerperal insanity is liable to a development of this disorder with a succeeding pregnancy. Insanity of pregnancy may occur in the earlier months, and may pass off spontaneously about the fourth month, or the insanity may develop during the later months of pregnancy, and in that case will probably not pass off till long after delivery. As a rule, the insanity of pregnancy is of a depressed type, the patient being suspicious, jealous, refusing food, dreading poison, and irritable. Sleepnessness, refusal to take food, and tendency to suicide or infanticide, are the most dangerous symptoms and those which have to be the most guarded against. The induction of premature labour is generally useless; and the best treatment is to improve the general health by change of scene, removal from home, or at least removal of children from home, withdrawal of husband's society, and careful watching.

The second group of cases of insanity with pregnancy contain those that have become insane after the fourth month. Such cases have frequently suffered much from exhaustion, such as frequent childbearing, repeated

lactations, and vomiting of pregnancy. They may be maniacal, but usually suffer from melancholia, with ideas of unworthiness, and are strongly suicidal and often infanticidal; some suffer more from simple nervous exhaustion and stupor. The treatment of these cases resembles that usual for such conditions—rest and food, and removal from home.

It is rare for the insanity to pass off permanently with delivery. In some cases during the pains of labour, and immediately after, there is a temporary improvement, but as a rule the sanity following labour in these cases is temporary.

The next class to be considered contains those who become insane soon after delivery. Puerperal insanity occurs in a very large proportion among women with distinct insane inheritance. The causes are generally prolonged, not sudden; repeated pregnancy, poverty, severe suffering with sickness during the pregnancy, or several of the above causes combined, and though there are few noteworthy symptoms till after delivery, yet we should be prepared to find, on careful investigation, that there have been threatenings and warnings which have been neglected.

Within two or three days of delivery occasionally there is a temporary or transitory maniacal attack allied to the febrile disturbance associated with the oncoming of milk, and, like that, the transient mania may be relieved by a free purge. It is noteworthy that in this transitory mania infanticide or suicide may occur.

Insanity occurring after labour is divided into that which befalls the puerperal woman at once, and that which develops within the first two months after delivery. The former contains the greater number of maniacal cases, and the latter the more melancholic. Puerperal insanity does not follow instrumental labours in any great excess. Quite natural and easy labours are frequently followed by puerperal insanity in predisposed subjects. Puerperal insanity has undoubtedly a great tendency to recur in the same individual, but it will not recur unless she become again pregnant. Puerperal insanity may be characterised by either maniacal or melancholic symptoms, or from the first there may be stupor. The onset of the disorder is generally as follows:—Sleepnessness for several nights, with irritability, loss of appetite, and querulousness, especially against the husband and child; then increased irritability or anxiety, with ideas of poisoning and refusal to take food; afterwards appear hallucinations of the senses, violent outbreaks occur, or profound melancholy may develop, in the one case the woman being destructive, and in the other suicidal. During this period the milk and lochia may be natural; on the other hand, one or both may be absent. From a practical point of view it is necessary to attend carefully to the milk, and to follow the old wives' simpler methods of "dispersing the milk" rather than making use of belladonna and other poisonous drugs. Friction with castor-oil or the application of hot salt are useful.

An attack of puerperal insanity usually lasts for several months, and requires most careful watching and care. The patients rapidly emaciate or neglect themselves, and develop some secondary disorder which often proves fatal. There is no more special danger in the insanity than there is in the puerperal condition, but the two combined require double care in their treatment. Abundant feeding is the rule, with removal from home, and the sparing use of chloral or bromide of potassium.

The period of excitement may be unduly prolonged, so that in some

cases patients are maniacal for eight or ten months before there is any sign of abatement of the disorder. One of the most characteristic signs of puerperal insanity in its later stages is the patient's discontent and jealousy of her husband, her tendency to mistake those who are about her, and afterwards a stupid, weak-minded stage which is extremely difficult to treat. Following an acute attack of puerperal insanity we must expect to have a prolonged period of depression; and as soon as the patient has ceased to be actively antagonistic to her husband and home, and especially as soon as she expresses a desire to return to her family, it is desirable to make some movements in that direction. It will be found at this period that the woman is getting stout, indolent, sleepy, and sometimes too contented with an asylum; amenorrhœa also exists, and frequently this amenorrhœa and general condition of apathy are only relieved by return to home and domestic cares.

In such prolonged cases a second pregnancy is particularly hazardous; and in all cases of puerperal insanity it is of the utmost importance to impress upon the husband the danger of another pregnancy occurring within at all events two years.

Seventy-five per cent. of puerperal cases recover, about 5 per cent. die, and 20 per cent. remain chronic. Of those who recover, the majority recover slowly; mental and bodily health improve together. Of those who die, some die from septicæmia, but more from exhaustion due to the weak bodily condition associated with the mental excitement. Of those who are uncured, a large proportion have had many previous attacks, but a certain number of patients do not recover from their first attacks of puerperal insanity, and it is therefore well to be guarded in the prognosis even from the first.

Beside puerperal insanity, we have a form of mental disorder associated with *lactation*. From two to three months after delivery patients become suspicious, sleepless, irritable, and inclined to wander from their home. In some cases there is amenorrhœa, but more often menorrhagia, due probably to incomplete involution of the uterus. These patients are sallow and anæmic, with bad appetite and constipated bowels; they frequently suffer from hallucinations of smell, taste, and hearing.

As a rule they require merely tonic treatment. They recover in the course of two to six months if removed to healthy surroundings, and away from home cares and worries. They require warmth, food, stimulants, fresh air, and, in the summer, baths at the seaside.

Alcoholic Insanity.—Although no one special form of insanity depends upon drink, yet there seems to be a definite relationship between alcoholic intemperance and the variety of insanity. It is therefore well to recognise the chief forms assumed by insanity depending upon drink.

Sudden outbursts of drink in nervous subjects will lead to delirium tremens, and delirium tremens, instead of passing off as it ordinarily does, assumes in such cases more or less the character of a chronic delirium. With some patients acute delirious mania follows an acute alcoholic debauch. In such cases the removal of stimulants will not suffice to cure directly, and the persistent use of opium is rather injurious than otherwise. Hallucinations of sight and hearing similar in all respects to those met with in delirium tremens occur, but are more persistent, refusal to take food is constant, emaciation takes place rapidly, and, unless the patients are fed abundantly

with milk and nutrient soups, a fatal issue may be expected. If the patient be young and strong, a purge by croton oil and an emetic of sulphate of zinc may be found the best treatment to start with. If the excitement seems likely to wear out the patient, chloral alone or with bromide of potassium may be given at night, but it is best not to give these drugs continuously or in too large doses.

Beside the acute mental disturbance produced by alcohol, we meet with every variety of delusional insanity, patients believing that their food is poisoned, that they are being watched, spied upon, and the like. Jealousy with vindictiveness is very often met with. Insane interpretations of morbid cutaneous and muscular feelings also occur; thus patients will complain of being galvanized, mesmerized, or "interfered with." The prognosis in those suffering from delusional insanity due to alcohol is not very hopeful, and in any case they should be kept under observation for many months before they are discharged as *well*. In some cases the morbid sensations persist and the morbid interpretations remain, and yet they cease to be active agents upon the patients' will, so that we meet with chronic cases of alcoholism, with chronic delusions, who are able to be at large without danger to themselves or friends. Chronic alcoholism probably tends in some cases to the production of general paralysis of the insane; it is much more certain that it tends to senile dementia, and in the cases of senile dementia connected with alcohol there is a great tendency to apoplexy, hemiplegia, and paralytic weakness of mind.

It is legal to send a patient suffering from alcoholism into an asylum if he cannot be controlled, or if he be dangerous to himself or others; yet we ought to be extremely cautious in signing certificates for persons suffering from chronic alcoholism with symptoms of insanity, for though seclusion will restore them to their former mental state, that is not a healthy or normal one, and the prospect of legal proceedings is in such cases considerable.

Moral Insanity.—It may seem unphilosophical to describe a form of insanity as belonging to the moral apart from the intellectual side of man, but the term will perhaps appear clearer to some readers if moral insanity be defined as a perversion of the nervous system by which the man or woman is rendered "anti-social." To give the best idea of what is meant—there are two distinct conditions under which moral insanity may exist: first, the children of insane or highly neurotic parents seem in many cases to come into the world unable to grow up into full intellectual and moral capacity; secondly, there are certain diseases of the brain or disorders of the mental functions which, having upset the nervous balance, leave a fresh moral standard far below what was the normal and healthy standard of the individual. Thus there are some patients who are unable to develop into their moral and social places, and there are others who having filled their proper position are as the result of disease reduced to the lowest moral rank.

First as to the young patients, the offspring of parents who have been drunken, epileptic, or insane, they not uncommonly are morally rather than intellectually insane. They are perverse and incapable of being trained into their duties to society. Thus they are frequently precocious and sexually vicious, they are cruel, mischievous, untruthful, thievish, and at times pyromaniacs. At the same time these persons may have one or two special gifts. Thus, they may be extremely rapid calculators, they may be able to reproduce

musical sounds readily, or they may have wonderful memories for isolated facts. As a rule such patients require isolation. As they grow older they become more dangerous from their sexual passions and from their destructive tendencies. They often pass into eccentric, weak-minded drudges in asylums.

Beside these we have to consider those who are morally weak as the result of acute attacks of insanity. After acute mania some young patients will be left kleptomaniacs, others will have lost their self-control and become amorous and erotic, others cease to be truthful and in one way or other show marked anti-social qualities. In these cases again there is little or no prospect of recovery,—they are like people who have been scarred by the smallpox, and nothing will ever efface the marks.

Not only after mania but after other forms of mental disorder, moral defects may become manifest; and the same may result from fevers. Temporary, and in a few cases permanent, moral change has been observed to follow an attack of typhoid or rheumatic fever.

Moral perversion may occur as an early symptom of general paralysis of the insane; it is common with alcoholism and is frequently seen in the earlier stages of senile dementia. In some cases the faults or vices of old men are really the result of early degeneration of the brain.

Moral insanity is scarcely capable of medical treatment. In the case of many young and middle-aged persons the best and indeed the only course is to let the delinquent receive the due punishment of his faults. The discipline of the jail has undoubtedly done good in some cases. Such young persons will ruin themselves and their friends, and if steps are taken to send them to asylums, they often retaliate and cause endless legal trouble.

Syphilis may give rise to various forms of insanity. The moral effect of the consciousness of transgression may set up hypochondriasis with syphyliphobia, and may thus lead to general paralysis of the insane. Syphilis may produce gummata and, with these, various degrees of mental weakness; or epilepsy, which may likewise lead to the same result. Syphilis may give rise to arterial changes which may lead to dementia, or to paralysis ending in dementia. In a considerable number of cases of general paralysis of the insane there is a history of syphilis.

It is not uncommon to find patients who have suffered from syphilis and who have recovered from some secondary symptom such as ptosis, external strabismus, or hemiplegia, pass into weak-mindedness or into conditions hard to be distinguished from general paralysis. In a certain number of weak-minded children and idiots, and in some elder patients, congenital syphilis has produced sensory or intellectual weakness. In any case of insanity in which syphilis has previously occurred, anti-syphilitic drugs should be used; but in most cases resembling general paralysis or with marked mental defect no good result will follow this treatment.

Gout may occur in neurotic families and may or may not be of importance. Before and after a gouty attack there are often marked mental changes in a patient, and in some cases of so-called suppressed gout most profound and suicidal melancholia may seem only to pass off when the gout disappears. Gout may seem to alternate with insanity as with asthma or eczema. Gouty degeneration of arteries is not uncommonly associated with signs of senile decay and dementia.

Lead-poisoning may produce maniacal excitement or it may end in dementia. The same poison may produce all the symptoms met with in

general paralysis, and may almost certainly give origin to that disease. It also leads to epilepsy and its results (cf. p. 673).

After *fevers* it is not uncommon to meet with more or less mental weakness, which does not much depend upon the severity of the fever. This is not uncommon after typhoid fever, and may range from simple loss of memory to loss of control and excitement, with emotional disturbance. In persons of highly nervous temperament, the delirium of any fever may set up mental disorder, which may appear as acute delirious mania or, more commonly, simple acute mania.

Phthisis is related to insanity in many ways. The insane are more than usually liable to phthisis. Insanity in one parent and phthisis in the other gives rise to a very unstable state of mind. Many patients who recover from severe attacks of mania or melancholia die a year or two afterwards of phthisis.

A form of insanity called phthisical insanity has been described. The patients are suspicious, and refuse their food because they believe it to be poisoned; they often have hallucinations of taste and smell, and of hearing; they rapidly waste, without having any cough or expectoration; the temperature is more or less like that of phthisis, and dulness is found if listened for under the clavicles. These patients require forcible feeding with stimulants and cod-liver oil. Hæmoptysis with temporary relief to the mental symptoms is not uncommon.

Spasmodic asthma often occurs in neurotic families, and it may occur in persons who have had attacks of insanity. In some cases the spasmodic attacks disappear and the patient becomes melancholic, only being restored to his right mind when the asthma returns.

Heart disease may act as a cause of insanity; mitral incompetence being most frequently associated with feelings of distress, anxiety, and melancholia, while aortic lesions, and at times mitral contraction, have been met with associated with mania and great excitement.

With *renal disease*, the tendency is toward mental depression.

Diabetes is not uncommon in neurotic families, nor is it very rare in insane persons; but generally with the onset of insanity the diabetes disappears.

Epilepsy and insanity are allied by origin. Epilepsy occurs in neurotic subjects, and epilepsy in infants causes idiocy or imbecility. Frequent recurrence of epileptic fits, whether in the major or minor form, leads to weak-mindedness. Hallucinations of the senses may precede the fits; and after them periods of unconsciousness may follow, during which highly complex acts may be performed, the patient being in a state allied to somnambulism (cf. *supra*, p. 820). Epileptic fits may be followed by fury of the most violent kind, during which brutal and bloody deeds may be done (p. 818).

Convulsions may probably be replaced by mental disorder—"l'épilepsie larvée." In this the patient after a sudden arrest of the work in hand goes through more or less highly organised acts unconsciously; these acts in each recurrence are exactly alike, and just as each epileptic fit is a repetition of its predecessors, so is each mental attack like its forerunners.

Jacksonian epilepsy is not so frequently followed by mental disorder as is the idiopathic epilepsy which occurs in neurotic subjects.

Paralytic insanity.—After apoplexy there is a great tendency to weak-mindedness. This is not always enough to deprive the patient completely of self-control, but it often leads to loss of memory and to a certain want of

self-control manifested in erotic lust, in emotional weakness, and in a tendency to be easily influenced by others. It commonly passes from the slighter to the graver forms of dementia, especially if fits recur.

Tumours of the brain generally tend more or less certainly to loss of some of the functions of the mind.

In *locomotor ataxy* we frequently meet with a neurotic history, and in many cases ataxy is an early symptom of general paralysis. But besides, with locomotor ataxy there may be outbreaks of mania, generally of a suspicious, jealous nature; or the symptoms of the disease may be interpreted thus by the patient: he may say that his legs are electrified and his sexual power removed by his enemies, or he may accuse people of twisting his guts or destroying his sight.

If the symptoms are part of general paralysis, the prognosis is bad; but if due to locomotor ataxy, the prognosis is much better as far as length of life is concerned.

Administrative Capacity.—One of the most important duties of the medical man is to be able to judge of the mental capacity of a patient who may have had some attack of insanity or other nervous disturbance. First, it must be remembered that some persons who have been insane recover completely, and may be able to transact business as well as ever; that the prospect of recovery and of mental capacity is lessened with the increasing number of attacks and with increasing years. Wills may be made by persons who are chronic lunatics, and yet the wills may be so reasonable that judge and jury will support them. It is important to remember that to upset testamentary capacity it must be shown that the patient had no knowledge of what he was doing, or that he had mistaken ideas about what he was effecting, or that his judgment was biassed by delusions of one kind or other, or, that being weak, he was unduly biassed. Defective memory is a thing to be specially noted. If it can be shown that a man when making a will did not remember the number of his children or whether they were living or dead, there would be ground for disputing that will. If, on the other hand, it can be shown that he was emotional and easily led by others, even though the memory was not very defective, yet it might be shown that he was too readily influenced at the time he made his will by those who were near him. If it can be shown that he had a causeless antipathy —the result of a delusion—to his direct heirs, it is only necessary to prove the nature of these delusions and their existence about the time the will was made. Apoplexy probably gives rise to weak-mindedness more frequently than any other disease of the brain, and the mental weakness produced by apoplexy certainly gives rise to the greatest number of trials in the probate courts. A man may have one or more attacks of apoplexy and yet may remain of "disposing" mind; but it is well to remember that after fits of apoplexy memory is very frequently affected, emotional disturbance is readily raised, and other signs of weakness and change of character are generally present.

There may be complete testamentary capacity without capacity for speech, in fact aphasia and weak-mindedness are not necessarily related; but it may be found difficult to get a jury to understand that a person unable to give the name to the simplest object may yet be able to dispose of his property. This difficulty will be greatly increased if agraphia also is present.

THE CONFINEMENT AND RESTRAINT OF INSANE PATIENTS.—One of the most difficult points for a medical man to decide is as to whether a patient should or should not be sent to an asylum; and having decided on sending a patient away from home, the next difficulty arises as to where the patient should be placed.

For the reception of persons of insane mind there are houses receiving one patient only; this is called *single care.* These houses are without any license and no certificate is necessary for many patients so placed. But it must be understood that, even though a patient be placed in "single care," it will be necessary to have the ordinary medical and other certificates in due and proper form, if he is so insane as to need his actions to be controlled.

Beside houses which receive only one patient, there are others, *licensed houses*, for the reception of two or more, and these merge insensibly into the private *asylums* which are to be found in most counties of England. Next there are registered *hospitals* into which patients are received at various rates, the principle of the hospital being that while some patients are received free or for small sums, others pay more than their actual cost and thus help to pay for the treatment of the poorer ones. There are also the large borough and county *asylums*, expressly intended for the reception of those who are not sufficiently wealthy to pay for private asylums, and who, from one cause or another, are ineligible for hospitals. Lastly, a certain number of quiet harmless lunatics are confined in the *workhouses* and workhouse infirmaries.

If a person of unsound mind is to be removed to the borough or county asylum, it is necessary that notice should be given to the relieving officer of the parish, who in due course reports to the medical officer of the same parish or some other medical man; and the patient being brought before a magistrate, and the medical man having certified that he is of unsound mind and unfit to be at large, the magistrate signs an order for the transference of the patient to the county asylum. If a person of unsound mind be found wandering about the streets, he may be given in charge of the police, and being taken before the magistrate, a doctor being called by the magistrate, may give evidence which satisfies the justice of the peace that the person is of unsound mind and requires detention; and then he may, on the magistrate's order and the medical man's certificate, be sent to the asylum. There are special provisions for cases in which no magistrate is available, or when the magistrates decline to act; thus two medical men signing a certificate can practically enforce the removal of a patient to a county asylum. No patient can be kept or retained in a medical man's house for profit except under full legal certificates, unless he be a near relative, it being manifest that no one's liberty of action must be restrained to the advantage of another without legal authority.

Before receiving a patient into an asylum, hospital, or private house, it is necessary that four documents should be completed. First, the ORDER for the reception, which should be made out and signed by a relative of the patient within one calendar month of his reception into the hospital. (2) A STATEMENT as to the name, age, and other facts concerning the patient and his attack of insanity, which should also be signed by a near relative. (3 and 4) TWO MEDICAL CERTIFICATES, which must be signed within seven clear days of the reception of the patient into the asylum, hospital, or private house. These certificates must be signed by medical men who have independently and separately examined the patient; they must not be in any connection with the person or asylum receiving the patient; nor must they have any connection

N.B.—Under all circumstances, if possible, the ORDER and STATEMENT below to be filled up by the Patient's Relatives. If no Relatives, by the nearest Friend.

ORDER FOR THE RECEPTION OF A PRIVATE PATIENT.

Schedule A, No. 1, Sects. 4, 8.

Lunatics 1. (16 & 17 Vic. c. 96.) PRIVATE PATIENT.

I, the undersigned, hereby request you to receive (*Name of Patient in full*) _____ whom I last saw at _____ on the (A) _____ day of _____ 188____ a (B) _____ as Patient in your Hospital.

(A) *Within* ONE MONTH *previous to the date of the order.*

(B) "Lunatic" *or* "an idiot," *or* "a person of unsound mind."

Subjoined is a Statement respecting the said (*Name of Patient in full*) _____

Signed, *Name*, (C) _____
Occupation (if any), _____
Place of abode, _____
Degree of Relationship (if any) or other circumstances of connection with the Patient. _____

(C) "Order" must be signed, if possible, by NEAREST RELATIVE.

Dated this _____ day of _____ 188____
To _____
Superintendent of _____

STATEMENT.

If any Particulars in this Statement be not known, the fact to be so stated.

DATES MUST BE INSERTED.

Name of Patient, with Christian Name at length . . . . _____
Sex and Age . . . . _____
Married, Single, or Widowed . _____
Condition of Life, and previous occupation (if any) . . _____
Religious Persuasion, as far as known . . . . _____
Previous Place of Abode . _____
Whether First Attack . . _____
Age (if known) on First Attack . _____
When and *Where* previously under Care and Treatment . _____
Duration of existing Attack . _____
Supposed cause . . . . _____
Whether subject to Epilepsy . _____
Whether Suicidal . . . . _____
Whether dangerous to others . _____
Whether found lunatic by Inquisition, and Date of Commission or Order for Inquisition . _____
Special Circumstances (if any) preventing the Patient being examined, before Admission, separately by Two Medical Practitioners . . . . _____
Name and Address of Relative to whom Notice of Death to be sent . . . . . _____

Signed, *Name*, (E) _____
Occupation (if any) _____
Place of Abode _____
Degree of Relationship (if any), or other circumstances of connection with the Patient _____

(E) *The "Statement" must be signed, but "where the person signing the statement is not the person who signs the order, the following particulars concerning the person signing the statement are to be added."*

8 & 9 VICT., CAP. 100, SEC. XLV.

N.B.—Medical Certificates of Patient's Examination, and the Signatures, are required by the above Statute to be dated within Seven clear Days of the Patient's Reception.—In stating the Residence, the number of the House must be specified, when there is any, and if not, the name of the occupier.

The Medical Men signing the Certificates must not be in Partnership, nor one an Assistant of the other; and they must use the terms specified in the Statute, for which see the notes of reference below.

By Order of the Commissioners in Lunacy.

1.—It is absolutely necessary that the Medical Men should write their Certificates legibly, so as to afford the opportunity of an exact copy being made.

2.—"All alterations in the original Certificates, *unless by the certifying Medical Men*, invalidate them; and *the initials of the latter* must be placed to every change or addition made."

3.—"If a Registered Medical Man describes himself as 'A duly qualified *Registered* Practitioner,' it is not necessary that he should specify his medical qualifications in full, in addition."

MEDICAL CERTIFICATE.

Schedule A, No. 2, Sects. 4, 5, 8, 10, 11, 12, 13.

I, the undersigned_____
being a (A) duly qualified Registered Practitioner_____
and being in actual practice as a (B)_____
hereby certify, that I, on the_____day of_____188____
at (C) *Here insert the street, and Number of the House (if any), or Name in full of Occupant*_____
in the County of_____ separately from any other Medical Practitioner, personally examined_____of (D) *State full address and Occupation (if any)*_____and that the said_____is a (E)_____ and a proper Person to be taken charge of and detained under care and treatment, and that I have formed this opinion upon the following grounds, viz.:—

1. Facts indicating Insanity observed by myself (F) *Some definite* FACT *or* FACTS *must be specified.*

2. Other facts (if any) indicating Insanity communicated to me by others (G) State the *Name in full* of the person giving the information.

Signed, *Name* _____
Place of Abode _____

Dated this_____day of_____One Thousand Eight Hundred and_____

(A) *Here set forth the qualification entitling the person certifying to practise as a physician, surgeon, or apothecary, ex. gra.:*—"A duly qualified Registered Practitioner, *which is sufficient.*"

(B) "PHYSICIAN," "SURGEON," or "APOTHECARY," *as the case may be.* "General Practitioner" will not do.

(C) *Here insert the street, and* NUMBER *of the house (if any), or other like particulars. N.B.—If no street nor number to house, state* NAME IN FULL OF OCCUPANT.

(D) *A. B. of* _____ *insert* FULL ADDRESS *and* OCCUPATION *(if any).*

(E) "Lunatic" *or* "an idiot," *or* "a person of unsound mind."

(F) *Here insert the facts. Some definite* FACT *or* FACTS *must be specified.* Please to write the facts legibly, and on the lines if possible.

(G) *Here state the information and* NAME IN FULL *from whom received*

with the person signing the "order;" nor must they be in partnership with one another. But there is no objection to a relative signing a medical certificate if he have not signed the order. The medical certificate need not be written on official paper, if it is in official terms. The medical certificate must state not only the facts indicating insanity but also the facts requiring the detention of the person of insane mind, for a certificate not only implies that a patient is of unsound mind, but also that he is unfit to be at large. Certificates, though informal or even incomplete, will yet suffice to hold the patient if the dates and the signatures be correct. Other details may be amended within a fortnight of the reception of the patient into the house or asylum.

In signing certificates it is well to remember that the most important points are the facts observed by one's self, and these facts bear much greater weight if they can be shown in brief to be associated with a development of nervous disturbance. Thus sleeplessness, restlessness, refusal to take food, threats of suicide, and a tendency to wander from home, would quite suffice without the additional fact which may have been communicated by A— B—, the nurse, that the patient tried to throw herself from the window and said she was possessed by the devil. Whenever the certificate is strengthened by the addition of facts communicated by others, the name in full of such person must not be omitted. Certificates in England only hold while the patient is in England, and are broken by escape to the Continent, to Scotland, or to Ireland; but if a patient escape from an asylum and remain in England, the same certificates hold good for fourteen days, during which period he may be recaptured.

Licenses for the reception of more than one lunatic into a private house are granted in the neighbourhood of London by the Commissioners, and in other parts of England by the magistrates in quarter session. Patients under certificates are in direct relationship to the Commissioners, who require a complete copy of the certificates upon which they have been received as well as a statement within a week of the reception of the patient as to his bodily and mental condition. They also require that a medical visitation book of a prescribed form should be kept and entries made from time to time of the visits paid by some independent medical man to the patient. The frequency of these visits will be decided upon by the Commissioners themselves. All letters written by the patients to the Commissioners in Lunacy must be forwarded unopened, all other letters of patients must either be forwarded to their address, or, being initialled, must be kept for the inspection of the Commissioners on their visit or of some other constituted authority.

Patients can be transferred from a private asylum into a private house, or from a hospital to a private house, or the reverse; permission having been sought and granted by the Commissioners in Lunacy. When a patient is sent from one establishment to another, the order of the Commissioners, with a copy of the original order, statement, and certificates must be forwarded with him. Patients cannot be transferred from a private asylum or private care to a county or borough asylum, or the reverse.

Though a patient is of unsound mind and under certificates, his friends and relatives have no right to transact any business in his name unless a power of attorney have been previously granted by the patient; so that it is advisable in all cases where recurrences of insanity occur, or where the patient is sufficiently sensible to understand the threatening illness, to get a power of attorney before he loses self-control. This may save great trouble and expense afterwards.

If it be necessary to transact important business connected with a person of unsound mind, the only legal method is to place the affairs in Chancery, there being a special department of the Court for the purpose. Of Chancery cases there are two groups. The first includes only small properties, that is under £2000 capital or £100 a year income, and for these the procedure is simple and rapid. It is as follows :—Two affidavits have to be drawn up in a form to be presently referred to, stating in detail the facts of the patient's insanity and his inability to transact business. These affidavits having been laid before the judge in Chancery, with certain other facts as to his affairs and his relations, by the family solicitor, an order may be given by the Court of Chancery for the administration of this small estate; and two people, respectively called the committee of the person and the committee of the estate, will be apppointed.

The following is an epitome of the second and longer process, which is costly and difficult to set aside, and therefore not to be undertaken without grave consideration: for the Court of Chancery will almost certainly realise the property of the patient, and a man's property may be realised at a very great loss while he is of unsound mind. It is therefore best to avoid placing any acute and presumably curable case under the Court of Chancery. When, however, it is necessary to put the affairs of a patient under the care of the Court, the family solicitor generally asks the medical man in charge of the case, or the asylum superintendent, to make a draft affidavit, and he also probably gets some independent medical expert in lunacy to draw up a second affidavit. These affidavits differ entirely from the ordinary medical certificate, there being no restriction as to consultation or separate and independent examination of the patient, and it is best to fill the affidavit up in the following way :— First, state your name, your medical qualifications, your qualifications to judge of the sanity of the patient, more especially your opportunities of having seen him; then give your judgment as to the form of disease from which he is suffering; give in the next two or three paragraphs a statement as to his general behaviour, his intellectual capacity, the delusions from which he suffers, the tendencies to suicide or destructiveness which he may have, and finally, give your judgment as to the improbability of his speedy recovery; or, if you believe him to be fatally or incurably insane, give your judgment that he is so and is unfit to take care, first of himself, second of his affairs. These two affidavits having been presented, in due course notice will be given to the patient that an inquiry or so-called inquisition or commission in lunacy will be held. The patient may then instruct counsel himself or he may demand to be tried by jury and to be defended by counsel and to be present himself. Or he may demand that he may be tried before a Master in Lunacy before a special jury. In one case at least a judge of the Court of Chancery took the position of the Master in an inquiry. In some cases where the patient is extremely suspicious, there may be considerable difficulty in persuading a jury that he is of unsound mind; and in nearly all cases where there is likely to be opposition of this kind, where in fact the patient is still so reasonable as to be able to defend himself, it is better to take any other means that are possible. If the patient be found insane by inquisition, the Court of Chancery in due time appoints one person to look after the affairs of the patient, who is called the committee of the estate, and another to look after the person of the lunatic, who is called the committee of the person.

A Chancery lunatic remains so, though he travel or though he escape to the Continent; he can be transferred from one asylum to another or from an asylum to a private house without any order of transfer from the Commissioners, all that is required being a signed order from the Court of Chancery the committee of the person.

If a patient recover after he has been made a Chancery patient, it is necessary, before he can have his freedom and control of his property, that the whole of the proceedings should be set aside by a similar process. Two medical men (preferably those two who have signed the first affidavits for the reception of the patient) should draw up two affidavits, this time stating their belief and the grounds for their belief that the patient is restored to his mind and is now fit to manage himself and his affairs.

The county and borough asylums are intended only for pauper lunatics, but really a large number of patients have to pass from private care to these county asylums because their friends are unable to pay the charges, which range from one guinea per week to almost any sum. Therefore chronic patients have to be provided for by the parish, the friends being called upon to pay for their maintenance in proportion to their ability and to the cost in the asylum. A certain number of county asylums at present receive paying private patients for sums under one pound a week, and it is probable that in any future lunacy legislation county and borough asylums will be empowered to receive such paying patients. Patients may, under certain conditions, place themselves in registered hospitals or private asylums as voluntary boarders.

Idiocy and Imbecility.—These two are but grades of the same want of intellectual development, "idiocy" ranging from almost complete absence of intelligence, whereas "imbecility" may approach very nearly to mere eccentricity and oddness.

Idiocy depends in the majority of cases upon structural defect in the brain. A certain number of idiots are the offspring of insane parents; certain others owe their condition to injuries received in early childhood; while certain forms of physical and mental disorder occurring in weakly or nervous children may arrest mental development at a higher or lower point, leaving as the result an idiot or an imbecile. The brain is so delicately constructed that a slight damage done to it during its period of development interferes with its permanent and normal development.

In looking at the pathology of idiocy, we have to recognise the fact that some children are born with defective brains, so that in some there are very little more than the ganglia at the base: a brain which suffices for the nutrition of the body but is unequal to the development of the mental functions. In other cases important parts of the brain are absent, its commissural fibres or the connecting fibres being deficient; and in the same category are to be considered the changes produced in the brain by defect of one or more of the senses. The child who comes into the world without sight and without hearing stands a very great chance of being an idiot through the consequent starvation of the brain. In idiots with defective brains we meet with every shade, from the anencephalous idiot, whose brain weighs but a few ounces, to the hydrocephalic idiot, whose brain is deficient though his head is enormous. In certain idiots there is rather a general deficiency than non-development of any one part of brain. All parts are there, but they are not sufficient. The convolutions are found

to be broad and few, the grey matter is either not fully developed or is defective in quantity, or there is an enormous overgrowth of the connective tissue, so that in some idiots there are not only enormous heads but weighty brains (cf. p. 648). Beside all these, there are some children who in aspect are healthy, but who are idiotic or imbecile in consequence of some ill-understood defect in the power of the brain to work. If one may use the term, there are some "functional" or "neurotic idiots" who have fairly-sized brains with fairly-developed convolutions, and yet with inability to make use of the brain they have. Some of these belong to the nervous stock generally, and others seem to have had their capacity for intellectual development arrested by some acute disorder of infancy. It is a question whether intermarriage will produce such cases.

The case of the so-called cretinous idiots will be considered in a separate chapter (*infra*, p. 876), and it is therefore unnecessary to describe the theories of the development of idiocy from arrest of growth in the base of the skull.

Most idiots are short-lived, many are defective not only in mind but in body. Many of them have malformations of limbs or a peculiar stunted aspect, with awkward, ungainly walk. Most of them are restless and irritable. Although generally slow of comprehension and dull as far as education is concerned, yet there are a certain number of what have been called "idiots" in whom some special faculty of the mind seems to grow at the expense of all the rest. Just as one may meet with a morally imbecile or morally insane person with certain intellectual activities fully developed, so in the learned idiot one may meet musical, mathematical, or mnemonic ability of a very high order.

Idiots have been divided according to their *capacity for speech*, the very lowest being unable to speak, the next group being able to ejaculate or make use of simple words, a higher grade being able to comprehend short sentences; while the lowest imbecile, somewhat in advance of the highest idiot, would have still greater power, and a higher imbecile would be able to learn and repeat many things, slowly perhaps, but yet surely. Other varieties have been based on the *facial peculiarities;* some idiots having extraordinarily receding foreheads have been compared to fish, and others have the aspect of a Chinaman, while some seem to resemble other races or even some of the lower animals; and when one takes up a book in which the physiognomy of man is compared with the physiognomy of brutes, one is struck with the examples which have evidently been taken in many cases from idiots and imbeciles.*

For our convenience here it will be well to take the groups of idiots as arranged by Dr Ireland. He makes ten groups—(1) Genetous idiocy; (2) microcephalic idiocy; (3) eclampsic idiocy; (4) epileptic idiocy; (5) hydrocephalic idiocy; (6) paralytic idiocy; (7) cretinism; (8) traumatic idiocy; (9) inflammatory idiocy; (10) idiocy of deprivation.

(*a*) *Congenital idiocy.*—The first class contains those cases which start in fœtal life; there is general weakness as a rule, mental and bodily, and the prospect is altogether unfavourable. The same holds good to a great extent with the microcephalic idiot, whose head, according to Dr Ireland, should measure less than seventeen inches; there being a distinct and absolute

* 'De Humana Physiognomia,' Joannis Baptistæ Portæ, Neapolitani libri iv, ed. altera, Francofurti, 1618. Note particularly the figures of human faces resembling the ox and the pig (pp. 82 and 90).

want of brain the prospect here is bad. In the third class of eclampsic idiots are placed those children who have ceased to develop in consequence of convulsions which have occurred during early childhood. Probably, however, a very large proportion of these come of a nervous stock, and are thus already unstable when some such exciting cause as teething suffices to start convulsions which so modify the brain in its development that it never assumes full vigour. Some of these cases may be trained to simple mechanical work.

(*b*) The next is a nearly related group containing the *epileptics*. Fully one third of the idiots in the Metropolitan Idiot Asylum at Darenth are children whose idiocy is at all events accompanied by epilepsy. As a rule the epilepsy weakens both mind and body, and in many cases seems to be associated with great moral perversity, the epileptic idiot being hard to manage, and in very few cases suitable for home treatment. A certain number of epileptic idiots outgrow their epilepsy, and though mentally defective may be developed to a moderate intellectual standard. Such patients should be tried with bromide of potassium, but cautiously; food and fresh air with discipline and association with healthier children are the best forms of treatment.

(*c*) With *hydrocephalus*, idiocy is not uncommon, and if the hydrocephalus be great it is almost certain that there will be intellectual defect; but the majority of cases with considerable hydrocephalic and mental weakness die, so that the number seen in a large idiot asylum is not great. A good many of these cases by careful treatment improve, and from idiots may be trained into the class of useful imbeciles, to a certain extent trustworthy, but having organic deficiency of brain which prevents them becoming more than useful drudges. Beside the hydrocephalic idiot, we have to recognise a group of *paralytic* idiots, in whom some local defect of brain occurring in early life has given rise to paralysis of one or more of the extremities, and has left a mental as well as a physical scar upon the brain. In these cases again we can expect but little improvement from treatment; the weakened limbs may be galvanised, friction and passive movements with baths may be used, and some power may be gained in them, just as some slight power may be gained in the faculties.

(*d*) The next group contains the *traumatic* idiot. A number of idiots are found to be males that are firstborn, and a certain number of others are found to have had injuries to their head during parturition, and it is therefore well to remember that the delicate brain, too severely pressed upon by a narrow pelvis, or interfered with unnecessarily or severely by instruments, may be so permanently injured that idiocy results. Injuries before birth, at birth, or soon after birth occurring to children, will frequently prevent development; and there is no doubt that falls and similar injuries in early infant life give rise to a considerable proportion of idiots. Many of these are rather imbecile than idiotic; for the brain, which ought under natural circumstances to have fully developed, has been so interfered with that it only develops partially, and with it the mental faculties also are impaired but not wanting.

(*e*) *Inflammatory* idiocy is very closely allied to the last group, but under this head have been placed the idiots with hypertrophy of the brain. Here there is excess of connective tissue which interferes with the proper development, or at all events the proper function of the grey matter. Beside these, there are also the cases in which some disease of the nose

or ears has set up inflammatory changes about the base or sides of the brain and interfered with its general development in a more or less mechanical way. As might be expected, the grades of idiocy from this cause vary very considerably.

(*f*) The only other important group is that associated with *deprivation of the senses.* It is quite certain that among the lower orders a considerable amount of weak-mindedness was formerly, and to a certain extent is still, due to the want of special education for the deaf and dumb. Deaf and dumb children if not specially educated will from the deprivation of external impressions be but partially developed, and in some cases this deficiency will be more marked than in others. In some, in fact, who would be weak-minded at the best, the additional restriction of their intellect by the want of communication with the outer world leaves them imbecile or idiotic.

The same thing is still more true of those who happen to have loss or impairment of more than one sense: to them the portals of knowledge are closed. Nevertheless if these patients are taken early and trained specially, a large amount of instruction may be given them and great amelioration of their condition will occur.

Idiots, if in the lower grades, should almost always be sent away from home. If there are other children, the influence of the weak-minded child upon them is distinctly injurious.

CRETINISM,

DELIRIUM TREMENS, SUNSTROKE, AND METALLIC TREMORS

CRETINISM AND ALLIED CONDITIONS.—*The idiocy of cretins as an endemic disease—Its relation to goitre—Sporadic cretinism in England—The cretinoid condition in adults (myxœdema)—The condition which follows removal of a bronchocele (cachexia strumipriva) in man—and in animals after removal of the thyroid.*

ALCOHOLIC POISONING. — *Chronic — Its symptoms and treatment — Acute—Delirium a potu—Symptoms—Prognosis and treatment.*

HEAT-STROKE.—*Origin—Cardiac and cerebro-spinal forms—Mortality—Sequelæ—Diagnosis—Treatment.*

MERCURIAL TREMORS.—*Course and symptoms—Diagnosis and treatment.*

CRETINISM AND ITS ALLIES.*—There remains to be described a form of idiocy which is peculiar in being constantly associated with remarkable peculiarities of cranial and bodily configuration: subjects of it are called "cretins." That the peculiarities in question are constantly present is not, indeed, admitted by everyone. Even Virchow says that he has found the skulls of notorious cretins, or of individuals from cretinous localities, presenting all kinds of abnormalities. But may not these have been skulls of non-cretinous idiots, who happened to be born in cretinous localities? However this may be, it is certain tha in the immense majority of cretins there is a special type of cranial develop ment. The mental symptoms do not appear to differ from those of ordinary idiocy. As in that condition, there are all degrees of defect of intelligence.

Endemic cretinism.—The most marked form of this curious disease occurs endemically in the Alps. It is in their bodily configuration that cretins differ from ordinary idiots. They are seldom more than four feet and a half in height and often below three feet. They have large heads, especially in the direction from ear to ear. The features are broad and thick; the eyes are wide apart; the nose is very flat at the root, and spreads out enormously towards the alæ; the mouth is very large, widely open, with thick lips, and allows the saliva to escape. The forehead and cheeks are wrinkled and the skin is course and rough, so that they have always an appearance of old age. A cretin twenty years old is exactly like a cretin of forty. The hair comes low on the forehead and is coarse and bristly. The chest is narrow, the belly large, and the limbs crooked. The hands are broad, with short fingers.

The observations of Virchow with regard to the dependence of malformations of the cranium upon a too early closure of the sutures find an

* Endemic cretinism is an exotic disease, and we therefore have no English word for it. The word is adopted by the French (as *crétin*) and Germans (*Kreidling*) from Switzerland. Its origin is uncertain; some would derive it from *chrétien*, others from a Romänsch word, *cretira*, of similar meaning, and others from *creta*, in allusion either to the chalky complexions of those who suffer from the disease (Littré) or to the calcareous formation of the districts where it prevails. A Royal Commission on Cretinism which reported to the Sardinian Government in 1848, divided those affected with it into three classes, for which they proposed respectively the names of "crétins," "semi-crétins," and "crétineux."

important application in the case of cretinism. That great pathologist has in fact shown that all the peculiarities in the configuration of the skull and face in this disease are the result of premature ossification of the basilar process. In a cretinous fœtus he found complete synostosis of the basi-occipital and basi-sphenoid bones, a continuous layer of diploë passing from one to the other, so that no trace of the original separation could be discovered. The basi- and pre-sphenoid bones were also fused together; but this is less important, because their union usually takes place soon after birth, whereas the basilar process and the sphenoid bone should remain separate until at least the fifteenth year. The completion of this "tribasilar synostosis" (as Virchow calls it) appears to be followed by the cessation of all further growth of the basis cranii in an antero-posterior direction. It also gives rise to a marked deformity in the shape of the internal base, the most constant features of which seem to be that the sella turcica is very narrow, and that the parts in front of the posterior clinoid processes form a greater angle than normal with the clivus or basilar process. Virchow found that the slope of the clivus was unduly steep in one fœtus. But at an early age a steep clivus is normal; and Nièpce has stated that in the cretins whose bodies he examined this part was horizontal; an observation which receives confirmation from a dissection of my own, to be presently referred to. It would therefore seem that the premature closure of the spheno-occipital suture, although it arrests growth, does not prevent the occurrence of some at least of the changes in the form of the base of the cranium which should take place in the years of childhood.

Laterally, the cretinous skull doubtless expands in its growth to an abnormal extent, so as to compensate for the deficiency in its antero-posterior diameter. This gives to the features of the cretin their peculiar breadth. The sunken appearance of the root of the nose is probably due to deficient forward growth of the vomer.

But if we admit that the configuration of the head and face in cretinism is directly attributable to premature synostosis of the basal sutures, it still remains a question whether this throws any light upon the ætiology of the disease as a whole. Some of the peculiarities in the build of the rest of the body may, indeed, possibly be due to a similar interruption in the growth of other bones. Thus Griesinger suggests that the shortening of the fingers and other parts of the limbs may depend on a too early ossification of the epiphyses of the several long bones, and hints that from this point of view cretinism may be regarded as the antithesis of rickets.

The importance of such speculations, however, is trifling in comparison with another question which must now be discussed, namely, that of the very close relation which exists between cretinism and *endemic goitre*. In this country as well as in others, goitre prevails in districts in which endemic cretinism is not met with. Indeed, the only place in England where the latter disease has been observed, is the village of Chiselborough in Somersetshire; and Dr Petherton, who in 1847 pointed out its presence there, told the author that it has since almost died out. On the other hand, there is probably no district in the world where endemic cretinism occurs without goitre being still more common. Moreover, it has been noticed that when a family migrates into a place where both diseases are met with, goitre is the first to appear; it is only in the second or third generation that cretins present themselves. These facts suggest the conclusion that both diseases depend upon a common cause, but that goitre results where it has

been in operation for a shorter time or with less intensity, cretinism when it has been in operation for a longer time or with greater intensity. Now, goitre or bronchocele is not a mere hypertrophy of the thyroid; it is a disease cystic, vascular, fibrous, of that remarkable organ, hence it is not surprising that a cretin, instead of possessing an enormous bronchocele, sometimes has no thyroid at all. Many of the worst cretins have no goitre. According to the Report of the Sardinian Commission, goitre is absent in one third of the cases of cretinism.

Sporadic cretinism.—Now, it is a most remarkable circumstance that in England a form of cretinism sometimes occurs which also bears a close relation to affections of the thyroid body, but which is not peculiar to one part of the country rather than to others. In papers in the 'Medico-Chirurgical Transactions' (1871) and the 'Pathological Transactions' (1874) the author described it as Sporadic Cretinism. In this form of the disease there seems never to be a large goitre, but sometimes a slight swelling can be detected; in the case of a boy who came to Guy's Hospital from Halden, in Kent, a sister had goitre. In the great majority of cases, however, the thyroid body, so far from being enlarged, is entirely absent, no trace of it being discoverable on dissection. This fact was first pointed out by Mr Curling in 1850. In almost every other respect sporadic cretinism is identical with the endemic form of the disease. Some of the figures which illustrate the above communications on the subject (figs. 1 and 3, pl. iii, 'Med.-Chir. Trans.,' vol. liv, and fig. 2, pl. xii, 'Path. Trans.,' vol. xxv) show how close is the resemblance between the two diseases in their general characters. Among the patients affected with sporadic cretinism, one, at the age of sixteen years and a half, was only two feet eight inches high; another, when twenty years old, was only two feet four inches high. They have the same broad, short hands and short fingers, the same dry hair, the same rough scurfy skin. Their heads are large and broad; their noses are flat at the root, so that the distance between the eyes is increased; and their mouths are large and gaping, with thick lips. In the only case in which an autopsy was made (through the kindness of Dr Grabham, of Earlswood) the base of the skull was much altered in shape; the posterior clinoid processes were at a much higher level than the anterior, and the sella turcica was exceedingly narrow. The clivus was horizontal, and its position seemed to be part of a general elevation of the occipital bone in relation with the other bones, for the cerebellar fossæ were exceedingly shallow. The line of union between the sphenoid and the occipital bones could no longer be traced, but as the patient was twenty-one years old at the time of death this of course had no significance.

The intelligence of those who are affected with sporadic cretinism is very imperfect, and many of them present an extreme degree of idiocy, and are even deprived of the power of speech. Their disposition is generally mild and tranquil. In this respect they seem to differ from the subjects of the endemic disease; for Griesinger says that the latter are unsociable and repugnant to each other. Probably, however, much depends on the conditions under which such patients are placed, and the amount of care and interest bestowed upon them.

One of the most curious features of sporadic cretinism has still to be mentioned. It consists in the presence on each side of the neck in the "posterior triangle," outside the sterno-mastoid muscle, of a soft, lobulated, and moveable tumour. This on dissection is found to consist entirely of

adipose tissue, which, however, may differ a little from the rest of the subcutaneous fat in appearance, being of a pinker colour. A characteristic case of sporadic cretinism is figured by Dr Fletcher Beach in the 'Trans. Intern. Med. Congr.,' 1881, vol. iii, p. 627, in which there was no thyroid and two large supraclavicular masses of fat. In one of the author's patients, these supraclavicular swellings were much larger than hen's eggs. Their size seems to be influenced to some extent by the state of the general health. In a case of Dr Beach's ('Path. Trans.,' xxv) they were well marked when the patient first came under observation, but disappeared entirely before death, which was caused by exhaustion after protracted diarrhœa. It therefore seems possible that the poverty and unfavourable hygienic influences under which the subjects of endemic cretinism labour may prevent the formation of supraclavicular tumours in that form of the disease. It is at any rate certain that their presence has not yet been noted, unless a reference to them is included in a remark of Virchow's to the effect that there is sometimes a large development of subcutaneous fat in cretins, with leucophlegmatic swelling of the external soft parts. Is it possible that less skilled observers have confounded them with the goitre?

However this may be, it would be premature to conclude that an absolute distinction between sporadic and endemic cretinism is afforded by the presence of those fatty tumours. Indeed, they are sometimes found slightly developed in adults who otherwise appear to be in good health.*

If the two forms of cretinism are in any way related to one another, we have a right to expect that the sporadic cases should throw light upon the obscure pathology of the endemic disease. Sporadic cretinism does not, so far as is known, depend on any external cause. Dr Langdon Down has suggested that it is due to alcoholic intoxication on the part of one of the parents at the time of procreation. But it has more than once been seen in several children of the same parents. Thus in a family of twelve, three children were cretins of an extreme type, while the rest were healthy, and one was an accomplished oarsman. They were all born in London, and their parents were in comfortable circumstances. The disease probably began to develop itself in them from the time of birth. Another of the author's cases of sporadic cretinism was that of a girl, who was stated by her relations to have been perfectly healthy until she was eight years old, when she fell ill with what was supposed to be a second attack of measles, and kept her bed for a fortnight. After her recovery her physical development underwent a remarkable change. Her features were previously well formed; they now acquired the cretinous configuration. Her hair, once black and abundant, became light-coloured, dry, crisp, and scanty. She ceased to grow; at the age of sixteen years and three quarters she was only four feet one inch in height. Her hands and feet were not larger than those of a child six or seven years old. She had a fatty tumour, the size of a hen's egg, above each clavicle; and no trace of the thyroid could be discovered in front of the trachea. Sporadic cretinism seldom develops itself so late as this, and endemic cretinism is probably always congenital. This case is very difficult of explanation. But taken with the fact that the thyroid is congenitally absent in so many cretins, it certainly suggests that the febrile illness at the age of eight years led in some way to atrophy of that organ, and to this supervention of the cretinous state.

* They were very large and well marked in a man weighing fifteen stone who consulted me recently (August 1887) for obesity and other symptoms of alcoholic excesses.—Ed.

Deficiency of the thyroid does not seem to be recorded except in cretins. There is therefore a close relation between the two conditions. But what the relation is, and on what common cause they both depend is most difficult to decide. With respect to goitre, it most frequently occurs in the valleys of a limestone formation; but neither drinking snow water, or water impregnated with lime salts, magnesia, or of ferric sulphide, nor want of light or air, nor living on any kind of food, can be its cause, for it is found in the absence of one or all these suggested causes. Cases are common not only in the Alps and Pyrenees but in Southern Germany, in the Peak district, where it is known as Derbyshire neck, and in mountainous regions in Central Asia, India, and other parts of the world.

Cretinism, as above stated, is far more restricted in range, but beside the sporadic cases just described, it occurs endemically in the goitrous regions of Switzerland, Saxony, the Engadine, and the Tyrol. It is found among the inhabitants of valleys, because the valleys are the parts inhabited; among those who drink snow water, because snow water is drunk among the mountains—for there are no cretins in the Arctic regions—and it is found among a goitrous population, because some unknown cause produces both diseases.

Two views may be taken of the relation of cretinism and goitre. One was put forward by the author of this work in 1871, namely, that they are both antagonistic effects of the same cause, and that goitre is protective against cretinism; that when the cause begins to act, or acts with but little intensity, the sole effect is goitre, but that if it acts with great intensity, or upon successive generations, it at length produces cretinism as well as goitre. The not infrequent absence of goitre in cretins—when not dependent upon congenital deficiency of the thyroid—might then be attributable to some local morbid change in that organ, by which it is prevented from enlarging as usual under the stimulus of the unknown morbid cause. It is conceivable that when goitre has existed in a family for two or three generations, the structure of the thyroid may undergo deterioration in some of the succeeding generation. The likelihood of the occurrence of such a local degeneration is perhaps augmented by the consideration that all families in which advanced cretinism is prevalent tend to undergo complete extinction within a very few years. According to the Sardinian Commission it is rare for any family residing in the Valpellina to reach a fifth generation. If it were not for immigration into this valley, it would become altogether depopulated. Such a deterioration of structure in the thyroid would of course favour still more the development of cretinism, according to the theory suggested. Large goitres are frequently present in the non-cretinous brothers and sisters of those cretins who themselves have no goitres or only small ones.

The other explanation of the relation between goitre and cretinism is that they are not antagonistic, but consecutive effects of the same unknown cause; that endemic goitre is not a true hypertrophy, but a degeneration of the thyroid, and that although a local disease, yet when inherited, or when supervening early, or when, most of all, the thyroid is congenitally absent, it produces more widely spread and serious disturbance so as to affect the nutrition of the whole body. We shall presently see that this view is supported by the results of experimental and surgical removal of the thyroid. But before mentioning this artificial cretinism it will be well to describe an allied condition not endemic but sporadic, and not congenital but often late in development.

Cretinoid Condition in Adults.*—Sir William Gull first described in 1873 ('Trans. Clin. Soc.,' vol. vii) a very remarkable condition which he terms *cretinoid*, and which occurs in adults—most frequently in adult women. It is characterised by a change in the features, which become broad and flattened; the eyes appear unduly wide apart, the alæ nasi become thick, the lips large, the connective tissue below the eyes loose and baggy, and that under the jaws and in the neck heavy, thick, and thrown into folds. The tongue is so large as to interfere with wearing false teeth, and to embarrass articulation. The hands are broad and spade-like. The texture of the skin becomes smooth and the hairs thin and scanty, while individually they are thicker and coarser than before. The general hue is pale, sometimes of a dirty-white, recalling that of the cachexia caused by malaria, by syphilis, or by lead, sometimes of a clear lemon-yellow like slight icterus; but the cheeks are most commonly rosy, not only from dilated veins, but with a diffused purple which looks like rouge. At the same time the disposition of the patient undergoes an alteration, activity of mind giving place to a gentle placid indifference.

One very remarkable case of this kind occurred in a young lady who was at one time not unpleasing in appearance, a patient of Dr Wilks. Her features underwent a most extraordinary change, and she became so hideous that the boys shouted at her when she showed herself in the streets. Her face became elongated; and her nose, lips, and mouth were enormous. There was not the slightest infiltration or hardening of the skin or subcutaneous tissue. She also had amaurosis, but this was perhaps an independent affection.*

A striking one was lately in Mary Ward (1885). She had the coarse scanty hair, the sallow complexion, with spots of bright red on the cheeks, looking as if she painted, the subconjunctival œdema, the broad, clumsy hands, thick blubber lips, slowness in answering, and tranquil temper. The contrast with a photograph taken ten years before was most remarkable. Though the "bladders" under the eyes looked ready to burst, acupuncture failed to bring out a drop of serum. Another woman was in another division of the same ward, under Dr Taylor, and the resemblance of the two was remarkable. In fact, the physiognomy when fully developed is unmistakable.

A few years ago (1884) there was a man in Philip Ward who was recognised as being in an early stage of the same condition. His deliberate answers, heavy aspect, and placid ox-like demeanour were already characteristic. Afterwards œdema, spade-like hands and the other features above described developed into a typical picture; so that, when subsequently in St Thomas's Hospital, he was shown by Dr Stone at an examination (1887), and every candidate recognised the disease at once.

The uniformity of aspect of these affections, whether occurring in women or more rarely in men, is not the least remarkable of its characters. Many cases have been now recorded in England since Sir William Gull first drew attention to the subject; by Dr Ord ('Med.-Chir. Trans.,' 1878, 'Clin. Trans.,' xiii, 1880), Dr Dyce Duckworth ('Clin. Trans.,' xiii, 1880), Dr Cavafy

* "A cretinoid state supervening in adult life in women (Gull)."—*Synonyms.*—Myxœdema (Ord), Cachéxie pachydermique (Charcot), Cachexia strumipriva (Kocher), Myxœdème primitive et opératoire (Reverdin).

† Dr Wilks has kindly informed the editor, in answer to inquiry as to the sequel of this case, that the patient was twenty-eight years old, had suffered from neuralgia, and gradually became blind. Her features were swollen, and her hands could not be fitted with ordinary gloves. She died, after six years, "comatose," but at a distance from town, so that there was no autopsy. The urine did not contain albumen.

(*ibid.*, xv, 1882), Dr John Harley ('Med.-Chir. Trans.,' 1884), Dr Drewitt ('Clin. Trans.,' xvii, 1884), Dr Nixon ('Dublin Quart. Journ.,' Jan., 1887). Cases have also been recognised in France and in Germany.*

The pathology and causes of the disease are quite unknown. It differs from the sporadic cretinism above described in the absence of deformity, with late development and slight affection of the mind. But it seems probable that it depends on the same essential cause and differs chiefly from the fact that this operates when the stature, the ossification of the bones, and the mental faculties are already fully developed. The late Dr Mahomed at one time believed that these patients were only examples of chronic Bright's disease; in many cases there is certainly albuminuria, and in some the other signs of granular degeneration of the kidneys, but in others equally marked the urine is perfectly normal. The apparent œdema is not ordinary anasarca, for serum does not exude on puncturing the skin. In a patient who died at St Thomas's Hospital Dr C. Charles found excess of mucin in the œdematous tissues after death, whence the name "myxœdema" has been proposed by Dr Ord for this remarkable cretinoid condition in adults and generally accepted. The condition which suggested it is probably not constant, for no excess of mucin has been found when sought for in other cases.

Artificial cretinism.—Although myxœdema had scarcely been recognised on the Continent, a remarkable condition which has been called "cachexia strumipriva"† was observed as the result of extirpation of bronchocele by Swiss surgeons, Dr Reverdin, of Geneva, and Dr Kocher, of Berne. After some months the patients who have survived the operation begin to show mental hebetude, pallor, œdema, and some other of the characters of an adult cretin ('Arch. f. kl. Chir.,' 1883, p. 254).

In the following year Schiff, Wagner, and Sanquirico successfully removed the thyroid in dogs and found that the operation was followed by cerebral disturbance, tremors, and convulsions, at first clonic but then tetanic, ending in death by coma.

Mr Victor Horsley has repeated these experiments on monkeys ("The Brown Lectures," reported in the 'Brit. Med Journ.,' Jan., 1885). He finds that (usually within a week) after the operation, fibrillary tremors appear in the limbs, which, like those of paralysis agitans, cease on voluntary movement. The monkey becomes anæmic, with increase of leucocytes as well as diminution of red discs. It sits moping and imbecile. The eyelids and abdomen swell. The temperature falls below normal, all tremors disappear, and the animal dies comatose in five to seven weeks. Two remarkable conditions appear to justify the application of the word myxœdema. One is great swelling of the submaxillary and parotid glands, so that the latter become as it were transformed into muciparous glands, and the other is the great increase of mucin in the connective tissues, especially the tendons and superficial fascia, and its appearance in traces in the blood. These facts rest upon analyses which were carefully carried out by Dr Halliburton.

Certainly the similarity of these results with those observed in human beings after thyrodectomy, and of both with the cretinoid condition of Sir William Gull and with sporadic and endemic cretinism itself, are very remarkable, and support the view above taken of the relations between cretinism and goitre.

* By Riess, Erb, Senator, and Landau ('Berliner klin. Wochenschrift,' 1886, 1887).

† Struma, as the equivalent of scrofula, meant originally a swollen neck, and was long applied indifferently to goitre and to swollen cervical lymph-glands. In Germany it has been restricted to the former meaning, so that struma means a bronchocele and strumipriva refers to its removal.

Alcoholism.—Until recently the action of alcohol upon the nervous centres was commonly supposed to consist mainly in the production of an acute disease which, from two of its principal symptoms, was called delirium tremens.* But it is now known that a long-continued state of ill-health is of far more frequent occurrence, and for this the name of chronic alcoholism has come into general use.

Chronic alcoholic poisoning.—Among the *symptoms* the most important is muscular tremor. This is more commonly noticed in the hands, which are unsteady and shake; but Anstie found that in a majority of cases the lower limbs were in reality affected before the upper. The patient is often able, at first, to control the movements by an effort of the will. They are generally more troublesome in the morning than at any other part of the day, and they may render him unable to do any work requiring nicety of manual adjustment, until he has taken a dram, or eaten some food, by which for a time they are removed. Even before marked tremor occurs, the action of alcohol is sometimes manifested by a peculiar restlessness; the limbs are apt to start involuntarily and cannot be kept quiet except by an effort of attention. At the same time there is an irritable condition of the mind; the patient, though he may feel drowsy when he goes to bed, cannot sleep, but keeps turning from side to side.

At a somewhat later stage, other cerebral symptoms develop themselves. Anstie speaks of a buzzing or rushing sound in the ears as very common; and with it there is often a dull diffused headache. Muscæ volitantes are often complained of; flashes of light may seem to pass before the eyes, especially at night, just when the patient is dropping off to sleep; and momentary attacks of vertigo often occur.

The intellectual and moral powers afterwards become impaired. All certainty of purpose is lost, and there is a mental inquietude which makes it impossible for the patient to settle down to any ordinary occupation or to complete the tasks he may begin. He often has a vague feeling of dread for which he cannot account; or he may become the subject of some delusion, such as that an enemy is lying in wait to injure him. Anstie mentions as another symptom that the patient often has a vivid apprehension that he is in danger of falling down a precipice, even when walking on firm ground in broad daylight, and he says that this is of unfavourable augury.

Another occasional effect of chronic alcoholism consists in pains in the limbs, especially around the wrists and ankles, as well as in the shoulders and down the spine. They are somewhat paroxysmal in their character, returning each day at about the same hour, most commonly towards night; and they are greatly aggravated by fatigue, whether of mind or body.

Impairment of sensation is also apt to occur, especially in the upper limbs; and the power of muscular coordination may be lost, so that the state of the patient is like that of a man affected with locomotor ataxy. Epileptiform convulsions sometimes show themselves; they are of the gravest augury. The mental state often passes into mania or melancholia, and ultimately complete dementia may develop itself.

Indications of gastric disorder are present at an early period. A common complaint is of nausea in the early morning, or even of actual vomiting, which may recur day after day as regularly as during early pregnancy. Morning diarrhœa is another frequent symptom. There is generally a failure of appetite, particularly at breakfast time. The tongue is foul, with a thick

* Delirium potatorum—Intoxication alcoolique—Säuferwahnsinn.

yellow fur; or it may be red and glazed. The breath commonly presents a peculiar fœtor, which is not that of any alcoholic drink, and which can hardly be described, although when once smelt it is unmistakable. The eyes are red and watery; the conjunctivæ are often jaundiced; the features look flabby and expressionless; the nose, cheeks, and forehead are often reddened, with crimson points and lines ("stigmata"), corresponding with minute veins which have undergone dilatation. Sometimes the same parts of the face present a papular or pustular eruption, such as is commonly known by the name of "acne rosacea," or the nose may be enormously enlarged, with pendulous outgrowths from its alæ. However, it must not be understood that the appearances last mentioned are seen only in those who have indulged in alcoholic excesses; they are sometimes seen in persons who have been perfectly temperate; acne rosacea in particular, in women whose menstrual functions are disordered.

The limbs of a person suffering from chronic alcoholism are commonly wasted, especially the legs. The abdomen on the other hand is generally enlarged or pendulous, and its parietes, as well as the contained viscera, may be loaded with adipose tissue. In those whose chief beverage has been beer, even the extremities may be covered with fat.

With regard to the chronic action of alcohol upon the nervous centres Anstie remarks that, contrary to what is the case with the digestive organs, the effects depend almost solely upon the quantity imbibed and not at all upon the form in which it is taken. There are, however, immense differences in the liability of different individuals to suffer from this poison. Some can drink spirits freely for years and seem to be none the worse for it, others break down in health under comparatively small amounts of drink. Women in particular show little power of resisting its evil effects; perhaps because those who are intemperate commonly pass all their time indoors.

Among the *antecedents* of intemperance must be mentioned, in the first place, those occupations in which persons are brought continually into contact with intoxicating liquors. A large proportion of the patients who present symptoms such as have been described are men employed in breweries or distilleries, public-house keepers and their wives, and travellers for wine and spirit merchants. Cabdrivers and hawkers, and others who are exposed to rough weather, are also very apt to be intemperate; while for some persons, as cooks and shoemakers, it is admitted as an excuse that their occupation is monotonous, deprives them of proper exercise, and keeps them confined in close, ill-ventilated rooms. Depressing mental influences drive others to drink; poverty and despair make them eager for the oblivion of intoxication. Again, there are cases in which stimulants were at first taken for the relief of pain, but in which the habit of indulgence grows upon the patient until it becomes a disease. Anstie has rightly laid great stress on the responsibility of allowing women to take wine or brandy in order to render them less susceptible to neuralgia or to the sufferings which are so apt to attend on the menstrual period or on lactation.

Lastly, there is no doubt that proclivity to intemperance is capable of hereditary transmission. This proclivity is by some writers regarded as itself a neurosis, and they give a particular form of it, the name suggested by Roesch, of "Oinomania," more correctly "Œnomania." The patient is seized every few months with a craving for drink, and for days together he behaves like a madman, taking long journeys without any purpose, or perhaps behaving indecently. But when the affection passes off—at the

end of a month or so—he regains his usual health. He then lives soberly and chastely, and manages his affairs with activity and intelligence.

The *morbid changes* that are found in the nervous centres of drunkards are essentially atrophic. The convolutions are rounded and wasted, and the cerebro-spinal fluid between them is in excess. The arachnoid is opaque and thickened. Even the bones of the cranium are sometimes found denser than normal and without diploë.

The *diagnosis* of chronic alcoholism may present every degree of difficulty or it may be perfectly easy. Commencing general paralysis, locomotor ataxy, hysteria, and the nervous malaise which may result from dyspepsia, are perhaps the conditions most likely to be confounded with it. Unfortunately one cannot place any confidence in the statements of the patient himself, however guardedly our questions may be put, for persons who indulge in secret drinking are almost always untruthful. Anstie recommends that one should rather abruptly but not uncourteously hazard the observation that the diet must be strictly unstimulating; this, he says, will often cause the patient to betray the truth by his manner, or his air of surprised unconcern may prove that the suspicion is groundless.

In the *treatment* of chronic alcoholism the most important point is that the patient should abstain entirely from all intoxicating drinks. It is in such cases above all others that large hydropathic hotels are of service, where regular habits and exercise in the open air are enforced, while an unstimulating diet is provided and all facilities for procuring drink are withdrawn.

The administration of certain medicines may also be of considerable service. Anstie recommends especially quinine, which (if there should be sickness) may be given in a state of effervescence. Dr Marcet's favourite remedy was the oxide of zinc; he says that in doses of from two grains upwards thrice daily it has a powerful effect in inducing sleep. Bromide of potassium often does great good in cases of this kind. According to Anstie half drachm doses of ether with the same quantity of tincture of sumbul may do much to restore tranquillity to the nervous system and so to make sleep possible. As a more direct hypnotic he recommends from a quarter to half a grain of the extract of Indian hemp. One should always employ the hypodermic injection of morphia (from one tenth to a quarter of a grain) rather than give any preparation of opium by the mouth, and this only when the urine is free from albumen and the patient has been purged. Even in advanced stages Anstie obtained striking benefit from the administration of cod-liver oil; and he says that when there is much tremor, strychnia is often useful in a dose of $\frac{1}{48}$ to $\frac{1}{32}$ of a grain three times daily.

As to *prognosis*, the symptoms of chronic alcoholism may last for years; and even the hardest drinkers sometimes reach old age. They are indeed apt to become prematurely old, their hair turning grey and their arteries becoming rigid. Gout, chronic disease of the kidneys or liver, or fatty degeneration of the heart may be developed, and cerebral apoplexy may cut them suddenly off. Such diseases as pneumonia or fever are ill borne, and are very likely to prove rapidly fatal.

Acute alcoholism.—In many cases the nervous symptoms of alcoholism take an acute form, and one that may quickly endanger life. They then constitute a disease which has for many years been known under the name of delirium tremens or "delirium potatorum." The earliest account of it, it appears, was published in 1813 by Dr Sutton, of Greenwich, but the first

writer to describe its course in detail, when not disturbed by the administration of medicine, was an American physician, Dr John Ware of Boston; and his work, based on the observation of nearly a hundred cases, is perhaps superior to any that has since appeared.

The patient has for two or three nights been more disturbed by unpleasant dreams than usual, and has by day been more restless and tremulous. At length he becomes altogether unable to sleep, but as he lies awake his dreams still haunt him. Next morning he may again be rational, but his mind almost always wanders occasionally. Towards evening the delirium returns, and the second night is worse than the first. During the second day there is again a slight amelioration as compared with the night, but the mental disorder is now fully established and persistent.

The delirium in this disease is in many respects peculiar. The patient is not fierce nor violent, nor, on the other hand, is he depressed in spirits and dull in manner. He is loquacious and restlessly anxious to follow his accustomed vocation, but he sets about his business in a blundering manner, and his mind quickly wanders away to something else. If spoken to, he is sufficiently intelligent to answer, and for a minute or two he may converse rationally, but before long he starts off on some fresh topic. His friends probably endeavour to keep him in bed, but he is always wanting to get up and dress himself. Yet, if firmly opposed, he forgets his intention, at least for the time. He commonly has hallucinations of vision. Very often he fancies that rats and mice, or snakes, or cockroaches, are running or crawling over his bed, and he tells those about him that he sees them. Or he may address persons who are really absent, but whom he supposes to be in the room. He looks suspiciously behind the curtains or under his pillow, or stretches himself out of bed to see if someone is not concealed beneath it.

His hands are in constant motion. He picks at the bedclothes, or grasps at imaginary objects. If one asks him to put his tongue out it is very tremulous, and is quickly withdrawn; it is commonly moist, and more or less thickly coated with a white fur. The pulse is quick, soft, and feeble. The skin is moist and often in a state of profuse perspiration. There is an entire absence of appetite and even of desire for drink. It was pointed out by Dr Bence Jones that the amount of phosphates in the urine is very greatly diminished.

Event.—The duration of these symptoms is not absolutely constant, but it is far more regular than is generally supposed. The disease almost always goes on for two and a half days without showing any tendency to subside, and then between the sixtieth and the seventy-second hours it comes to an end. Towards this period the patient is very apt to show signs of exhaustion. His pulse becomes more and more rapid and feeble. His face, at first flushed, is now pale and haggard; his pupils are widely dilated; his tongue sometimes, but rarely, becomes dry and brown.

It is, however, precisely at this time that a favourable change is to be hoped for. Towards the end of the third night the patient commonly falls asleep. At first he is still uneasy and restless, his breathing is irregular, and after an hour or two he may wake up for a little while. He soon goes soundly to sleep again; his breathing is now slow and deep; a profuse sweat breaks out over his body. After six or eight hours he awakes and is relieved. In the next twenty-four or forty-eight hours he sleeps almost continuously, and after this his restoration to health appears complete.

In some cases, particularly in those who had been generally in good health, and in whom the attack has been the direct result of a debauch, it

terminates earlier—perhaps at the end of twenty-four hours. In other cases it lasts beyond the specified time. Dr Ware speaks of having once known it to extend to nearly six entire days. This is most apt to occur in those who have for a long time been habitually intemperate.

But the end of the disease is by no means always favourable. Sometimes a sudden attack of convulsions occurs, by which the patient is carried off; sometimes he becomes comatose; and sometimes he falls back in his chair and dies unexpectedly in a state of syncope.

Delirium tremens has a marked tendency to attack the same patient over and over again, unless he makes an entire change in his habits.

Ætiology.—So far as is known, delirium tremens never occurs except as the result of alcoholic intemperance; but it is often exceedingly difficult to discover how intoxicating liquors are obtained. Women, in particular, will suborn their servants or attendants, or procure tincture of lavender or sweet spirits of nitre from the chemist, or drink eau de Cologne.

At one time it was generally believed that instead of the immediate exciting cause of delirium tremens being the alcoholic stimulus itself, the disease was most apt to occur in persons who, from whatever cause, had been suddenly deprived of the drink to which they had been accustomed. Now, there can be no question that before the attack the patient has sometimes left off drinking for a few days; and, again, that it often shows itself in those who have been kept for a time without stimulants, in consequence of their having broken a limb or received some other injury. But many patients are attacked by delirium tremens as the direct result of a bout of hard drinking. And the experience of those who have the management of prisons and other institutions where abstinence is enforced, has demonstrated that this does not in itself bring on an attack, even in the most intemperate. The facts which have been supposed to establish the contrary opinion are otherwise explicable. First, a dislike for stimulants is sometimes an early symptom, so that the reason why the patient leaves off drinking may be that he is already beginning to suffer from the disease. Secondly, a sudden shock to the system is exceedingly apt to act as a direct exciting cause of delirium tremens in those who are predisposed to it by intemperance. It is this which renders the disease so common in the accident wards of hospitals. Indeed, it often comes on within the first few hours after the patient receives an injury, when there could have been no time for the withdrawal of stimulants to take effect. It is also frequently observed as a complication of acute pneumonia, erysipelas, or some other febrile disease.

Diagnosis.—As stated above, delirium tremens has been formally recognised by physicians only during the present century. Dr Sutton, however, mentions that Dr William Saunders, a former lecturer on medicine at Guy's Hospital, had for forty years described it as distinct from phrenitis. Dr Sutton had become acquainted with it when practising in East Kent, where spirits brought in by smugglers could be had in great abundance at a cheap rate. Certain of the practitioners in that district, he says, had learnt to treat such cases with opium; and this practice was attended with very marked success in comparison with that of others (including at first Dr Sutton himself), who regarded the disease as an inflammation of the brain, to be combated by venæsection, blisters, purging, &c. Even Sir Thomas Watson, in his classical work on medicine, approaches the subject entirely from this point of view. He speaks of once being summoned to a man who was supposed to be mad or to have brain fever, and of the necessity that one

should be alive to the distinctive symptoms of delirium tremens. But at the present day the opposite mistake is the more likely to be committed, a case being set down as one of this disease when it is really one of acute mania or acute melancholia, diseases which (it must not be forgotten) may also be the result of alcoholic intemperance.

Dr Wilks speaks of having seen general paralysis of the insane mistaken for delirium tremens on two different occasions by the same physician. In the surgical wards of a hospital there is often great difficulty in distinguishing the latter disease from the effects of injury to the head. Again, fever or acute pneumonia may be overlooked, and a case wrongly set down as one of delirium tremens, particularly if the patient should have been intemperate. But in such instances it is perhaps really present as a complication. Moreover, we must remember that pneumonia may be altogether latent in drunkards. Dr Wilks mentions rheumatic fever, when attended with cerebral symptoms, as particularly like delirium tremens in its superficial characters.

Treatment.—The observers who first distinguished delirium tremens from acute encephalitis obtained what seemed to them wonderfully successful results by treating it with opium, and until recently most writers have advocated this practice. Sir Thomas Watson, for example, recommends that three grains of solid opium should be administered as soon as the bowels have been cleared out by a moderate purgative ; and that if at the end of two or three hours the patient should show no inclination to sleep, one grain should be given every hour afterwards until the result is attained. Or, he adds, corresponding quantities of laudanum, of Battley's liquor opii sedativus or of morphia may be prescribed.

But this advice was based upon the supposition that the disease, instead of having a tendency to subside spontaneously, would run on and perhaps prove fatal unless the patient were made to sleep. Sir Thomas Watson expressly remarks that delirium tremens is not likely to be "healed with a *placebo* or by waiting upon nature." And it is clear that the physicians who laid such great stress upon the value of opium measured their success by comparison with cases of encephalitis healed by venæsection, leeches, blisters, &c. Wilks, Anstie, and most modern writers endorse Dr Ware's statements as to the natural course of the disease.

It is still, however, a question whether the course of the disease may not be abbreviated by hypnotic medicines. Anstie thought that this might be effected by chloral hydrate. He gave thirty grains for the first dose and repeated it in an hour if the patient did not sleep. He found that the patient almost always got two or three hours of sound repose, and sometimes much more. He was of opinion that in delirium tremens there is a tolerance for chloral, so that as much as ninety grains or even two drachms may be safely given in divided doses within twenty-four hours, at least for a day or two. But Dr Wilks is disposed to share Dr Ware's opinion that the duration of the disease cannot be shortened by giving medicines to send the patient to sleep. He thinks that a certain time is required for the subsidence of the commotion, and that a severe attack must last at least three days.

If this view is correct it would seem to follow that at the commencement of delirium tremens neither opium nor morphia should be given at all, or at least that their administration should not be repeated so as to cause contraction of the pupils. For, when hypnotic remedies are actively pushed, one is apt to find oneself after two or three days in a difficult position. The patient,

instead of sleeping, may be as excited as ever. His pupils are perhaps reduced to the size of pins' points, and he has had as much opium as would kill two or three healthy persons. Under such circumstances—which are precisely those in which a consulting opinion is most likely to be sought—there can be no question that the proper course is to wait. The further administration of hypnotics is dangerous. Dr Wilks says that he has many times seen persons suffering from delirium tremens sent to their last sleep by opium, and I fear that the same result followed the subcutaneous injection of morphia in one case that I saw some years ago. The fatal symptoms produced by these drugs in patients suffering from delirium tremens seem to differ from those to which they give rise in healthy persons. At least the patient just referred to became collapsed rather than comatose, and so in similar instances. Indeed, as already mentioned, the disease sometimes terminates by sudden collapse, even when no hypnotics have been given. Dr Bristowe believes that opium may still be used even in full doses in delirium tremens with good results if given early and watched.

Another question, which has sometimes been raised when a patient has died after the administration of a large dose of opium, is whether one can infer that this could not have been the cause of death from the fact that a certain interval of time had elapsed. Thus, in a case that occurred to Sir Thomas Watson, that physician decided that the medicine could not have been concerned in bringing about the fatal result, because nine hours passed after the last dose (one of three grains) was given before the patient became comatose. This conclusion would be perfectly valid if the case were one of suspected poisoning in a healthy man, but it is scarcely equally safe in delirium tremens. Dr Christison long ago pointed out that in persons actually intoxicated with alcohol the effects of opium are much retarded.

But whatever doubts may fairly be entertained as to the part played by opium in bringing about a fatal result in cases of this kind, there can be none as to the importance of treating delirium tremens so that no such question can be raised. We have seen that most patients do well even if no opium at all is given, and therefore the fact that many recover after having taken heroic doses is no justification for administering them.

This, however, does not settle the problem whether or not it is advisable to give safe and moderate doses of opium in the early period of delirium tremens, at a time when there is no reason to suppose that the case will be one of so grave a kind. To determine that point, one requires to know whether the medicine, if it should fail to give sleep, can do any harm; and, again, whether it is ever needed at a later period, so that one may be then debarred from using it by having employed it at first.

But before we can answer these questions we must know what is the class of subjects in whom delirium tremens is most apt to prove fatal. Now, as regards *prognosis*, Anstie speaks of old age as especially unfavourable, and one has been always accustomed to think that for young subjects the disease ought to be unattended with danger in a first attack, and even in two or three subsequent ones; but Dr Aitken quotes some statistical facts published by Dr Macpherson, and based upon observations made in the General Hospital at Calcutta, and in them it is curious that the highest average mortality was in persons between twenty-five and thirty-five years old, being from 23 to 24 per cent. of those attacked, whereas the mean mortality at all ages was about 15 per cent. Among fifty-nine cases of fatal delirium tremens which have occurred within the last few years at Guy's

Hospital, there were three in persons between twenty and twenty-nine years of age, eight in those between thirty and thirty-nine, ten in those between forty and forty-nine, eight in those between fifty and sixty. What ratios, however, existed between these numbers and those of patients attacked by the disease during the corresponding periods is not ascertained.

The state of the *kidneys* affects the prognosis in delirium tremens very materially, uræmic coma being very apt to occur as a complication. The urine must be repeatedly examined for albumen and for casts; and the quantity passed each day must be noted. If the secretion should begin to fail, Anstie recommends hot fomentations and dry cupping to the loins, hot footbaths, and the administration of half-ounce doses of infusion of digitalis every three hours. Digitalis was recommended as a remedy for delirium tremens itself by the late Mr Jones, of Jersey, who used to give enormous doses of the tincture, and clearly showed that digitalis is one of the poisonous agents for which there is a special tolerance in this disease.

Of even greater importance in reference to the prognosis of delirium tremens is the state of the *heart*. Anstie pointed out that besides feeling the pulse, one should carefully watch the first sound of the heart with the stethoscope. And he laid still more stress upon the use of the sphygmograph. An "irregularly undulating" character of the pulse-wave—such as occurs in the typhoid condition—was found by him to be of the most unfavourable augury. In his article in the 'System of Medicine' a tracing is given which was taken from a man, aged forty, who, after being delirious for nearly a week, fell into a sound sleep of six or seven hours' duration, and when he woke appeared to be so much better that a confident opinion was expressed that he would recover. Anstie's sphygmographic observation, however, led him to augur the worst possible issue, and about twenty-four hours later the case terminated fatally.

The symptoms which indicate approaching failure of the heart's action are chiefly faintness and lividity of the countenance. Free stimulation appears to be the only resource when they manifest themselves. If the stomach will retain ether this may be given in half-drachm doses. But, if not, Anstie recommended port wine, especially such as is old and contains volatile ethereal compounds in abundance. He speaks of giving an ounce and a half of such wine every hour. He also says that hot mustard plasters to the chest may be useful.

Before giving either opiates or stimulants it is most important to see that the bowels are freely open.

General management.—The liability for the heart to fail in delirium tremens renders it necessary that the patient should be well supported by nourishment from the very commencement of the disease. He may have milk if he will take it; but, if not, he must be compelled to swallow strong beef-tea, beef-tea jelly, or soup. He may even have small pieces of underdone chop or steak, if he can be induced to eat them.

The administration of alcohol in moderate doses was formerly recommended in cases of delirium tremens as a routine practice; it was thought to aid in inducing sleep. But all those who have studied the natural course of the disease are now agreed that there is no proof of its acting in this way; and they point out that there are great objections to its use, since the patient's future depends entirely upon the effect which his illness may have in leading him to give up stimulants for the rest of his life.

Another matter of the highest importance is that the room should be kept cool and dark and quiet. The patient's friends, who commonly collect

around him, must be sent away, one or two active men being alone left to watch him. The gaslights are to be turned down, and in the daytime a dark cloth is to be hung before the window, unless the patient should be terrified by the hallucinations which visit him in the dark. If he should be very troublesome in wanting to get out of bed, the question must be entertained whether he should not be tied down. If an attendant can by persuasion, or by employing a little force from time to time, induce him to lie quietly in his bed, that is no doubt to be preferred. But it is far better that he should be kept in a recumbent position by a sheet folded across his chest and tucked well in, than that he should be violently held down and restrained for any length of time by the hands of those about him.

HEAT-STROKE.*—That exposure to intense heat is sometimes followed by alarming or fatal cerebral symptoms is now well known. The affection is not uncommon in the hotter parts of India, and it now and then occurs during the summer in England. It is also frequent during the heats in New York and sometimes in Australia. The common term " Sunstroke " is unfortunate in suggesting that the direct rays of the sun are required to give rise to it; whereas the truth is that it often comes on at night, when the temperature is very high, and when a number of persons are crowded together, as among soldiers in barracks, or sailors and others on board ship. Sir Joseph Fayrer mentions it as occurring to the stokers in the engine rooms of the Red Sea steamers. *Heat-stroke* is therefore a better name.

As might be expected, this disease often attacks several men simultaneously or in rapid succession. Dr Maclean speaks of having seen a great many soldiers of the 98th Regiment struck down, of whom about fifteen died on the spot, in taking possession of a steep hill in China in 1842. But those who suffer from heat-stroke appear to be never more than a minority of those who are exposed at the same time to the sun's rays. This of course depends upon the fact that the disease is due to the failure of those natural processes which should enable the human body to resist the action of excessive heat. Dr Maclean remarks that the closely-shaven heads of the Chinese bear the hottest sun without ill-effects, although it is true that these strange people generally make vigorous use of their fans to keep up a free current of air about their faces. Sportsmen in India expose themselves to very high temperatures with an impunity that would be surprising, were it not for the care which they take to wear light clothes, to protect the head and the spine, and to abstain from stimulants. Thus it is of the highest importance that soldiers and others who have to bear fatigue in hot climates should be suitably dressed, and should have no accoutrements which can interfere with the play of the lungs. More than one observer has noticed that large and fat men are especially apt to be attacked. Dr Maclean thinks that exhaustion from prolonged exertion is a predisposing cause of heat-stroke. Other factors which are believed to be often concerned in its development in those who are not directly exposed to the sun are overcrowding and defective ventilation. It would seem to be more likely to occur when the air is loaded with moisture, since this must interfere with evaporation from the skin; but there was extreme dryness at the time of an outbreak which Mr Longmore saw at Barrackpore. Europeans are supposed to be more likely to suffer from the disease when they

* *Synonyms.*—Solis ictus—Insolatio—Sunstroke—Heat-apoplexy—Coup de Soleil—La calenture (in part)—Hitzschlag, Sonnenstich.

have been only a short time in India, but there is good evidence that natives are by no means free from liability to it.

Symptoms.—The phenomena of heat-stroke vary in different cases.

(1) There is a "cardiac" form of it. In this death may be almost instantaneous by syncope, the man falling down suddenly insensible and making only a few hurried gasping respirations. Dr Morehead also describes milder cases, in which a sense of prostration and faintness are experienced, with vertigo, dimness of vision, dilated pupils, and drowsiness. The patient can be roused by pinching him, speaking to him, or sprinkling his face with cold water. There is constriction of the chest, with sighing respiration, a sense of weight or sinking at the epigastrium, nausea, and sometimes vomiting. The face and lips are pale. The skin is generally cold and clammy, with the exception of the head, which is somewhat hot. The pulse is feeble and it is generally slow. Under judicious management such cases often recover; but sometimes the pulse sinks, the breathing becomes more sighing and irregular, and death occurs, being perhaps preceded by convulsions. "Cardiac" cases seem to be met with only among those who are attacked in consequence of direct exposure to the sun's rays. When recovery takes place it is complete, leaving no sequelæ.

(2) There is a "cerebro-spinal" form, of which coma is the principal feature. This often comes on gradually. It may be preceded by nausea and loathing of food, giddiness, congestion of the eyes, extreme debility, and (as more than one observer has noticed) an unusual frequency of micturition. "I cannot hold my water" is said to have been in a large number of cases the first thing complained of. Dr Maclean speaks of the patients beginning with a wild shout of laughter, or with a delirious attempt to escape from an imaginary enemy; headache, he says, is seldom present. When heat-stroke occurs in a man sleeping in barracks, what draws the attention of his comrades to him is often his stertorous breathing. He is then found already insensible, with contracted pupils, deeply congested conjunctivæ, and a quick sharp pulse. As far back as 1860 Dr Morehead pointed out that the skin continued pungently hot to the close of these cases, and even for some time after death. More recently, since the introduction of the thermometer into practice, it has been shown that the condition is really one of hyperpyrexia; and, in consequence, the name of "thermic fever" has been proposed for them. Three instances of this variety of the disease occurred in 1876 at Bristol, and were fully recorded in the 'Lancet' by Dr Shingleton Smith and Dr E. L. Fox. One of them was in a man, aged forty-five, who had been turning a winch on board a steamship, exposed to the full glare of the sun, from 8 a.m. The temperature in the shade was from 92·3° to 96°. At one o'clock he felt unwell, and began to talk incoherently and to throw his arms and legs about. He had been drinking water freely. He was admitted into the hospital half an hour later, and he was then comatose, with pin-point pupils; the temperature was 107°; the pulse 160, weak, and intermitting; the breathing laboured; the face not flushed; the skin sweating. At twelve minutes past two the thermometer registered 109°. Under energetic treatment the temperature gradually fell to 100·4; the pupils became normal and afterwards dilated; but the pulse failed more and more until it could not be counted, and at 7.40 he died. On the very same day, another man, aged thirty-five, was taken into the same hospital in a state of only partial consciousness, with general muscular tremor, soon followed

by convulsions and opisthotonos. His temperature was then 110·2°; his pulse was too rapid and too feeble to be counted. He died twenty minutes later, the thermometer at that time registering 111°. Two days afterwards, a third case was admitted at 2.30 p.m. in the person of a man, aged fifty-five, who had been driving a hearse, when he fell backwards, and the reins dropped from his hands. He was comatose; there was tonic spasm of the muscles of the legs; the pupils were contracted, but sensible to light. The temperature at 2.40 was 106·4°; at 2.50 it was 107°. The pulse was 141; the breathing was stertorous and at the rate of 27 in the minute. Under treatment the temperature quickly fell; at 3.25 it was 102°, at 3.40 it was 100°. In this instance recovery took place. These three cases appear to have presented all the chief symptoms of "thermic fever," as it is described by those who are familiar with sunstroke in India.

(3) Dr Morehead speak of a "mixed form" of heat-stroke, in which the symptoms are a variable combination of those of the other two forms. Sir Joseph Fayrer distinguishes syncopal, asphyxial, and hyperpyrexial varieties.

Event.—When heat-stroke ends in the patient's death, this generally occurs within nine hours, but sometimes not until towards the end of the second day. The average mortality of the disease is estimated at from 45 to 50 per cent. Fatal relapses, after recovery from incomplete coma, are not infrequent; cases which seem to be doing well require to be carefully watched with the thermometer until the skin becomes moist and cool. The only *post-mortem* appearances are those of congestion of the viscera generally. The blood is said always to remain fluid. In twenty-five cases of death, among forty-eight of heat-stroke, among the troops at Assouan in 1886, Surgeon Douglas Hunter reports the *post-mortem* appearances thus: intense lividity of the surface and ecchymosis of the conjunctiva; venous engorgement; muscles dark; only one case of meningitis. Meningitis is perhaps occasionally set up by the sun's heat (pp. 701–2); but those cases seem to be distinguished by there being an interval between the time of exposure and the onset of symptoms.

In 1856 there was in Guy's Hospital a sailor who four years previously, when crossing the equator, had been attacked with the disease in company with another man who died. He himself came to in a few hours, but he could not speak for a month afterwards, and for a time he lost the use of his right arm and leg. From all these symptoms, however, he had perfectly recovered; and he was now suffering from renal dropsy, which at length proved fatal. At the autopsy Dr Wilks found the arachnoid opaque and marked with white spots, the ependyma granular, and an excess of fluid in the brain. It is to be noted, however, that the vessels at the base were much diseased, so that the other morbid appearances may have been unconnected with heat-stroke.

It is well known that sequelæ of a very serious kind are not infrequent. Those mentioned by Dr Maclean are persisting headache, either fixed or shifting; a chorea-like affection of the muscles, generally those of the forearms and hands; epilepsy, particularly in those who have inherited a tendency to that disease, or have had fits in youth; and mental weakness.

Diagnosis.—Heat-stroke is no doubt easy to recognise in most cases; but this often is because the patient is known to have been exposed to a high temperature, rather than because the symptoms are in themselves distinctive. Maclean places reliance on certain characters of the pulse and of the respiration, and on the state of the skin and of the pupils in apoplexy, as serving to exclude that disease from consideration. Probably there need be no doubt as to the real nature of those cases in which there is hyperpyrexia from the

very commencement. But this is not always present; and probably a person attacked on a very hot day in India with cerebral hæmorrhage, or embolism, would be exceedingly likely to have his case set down as one of "sunstroke," even by good observers, especially if there were no obvious paralytic symptoms. It often happens that patients tell one that their complaints all result from a "sunstroke" which they had in England during the previous summer; but such statements must be received with caution.

Apart, however, from the classical coup de soleil or thermic fever of India and the tropics generally, there is a very much milder malady which is probably due to exposure to the sun and is most often seen in children in England. A boy, after playing in the sun, will come in complaining of feeling sick and faint; he may perhaps vomit, looks very pale and has a bad headache. Cool drinks and rest in a dark room generally set him right in a few hours.

In the *treatment* of coup de soleil the most important thing is the bold and immediate employment of cold. Ice should be applied to the head; a stream of iced water may be passed through coils of elastic or leaden tubing (which are now made for the purpose) in contact with the back and the chest; cold water must be poured over the head and neck and chest for a few minutes at a time. If the patient can swallow he should be allowed to drink freely. In the one successful case at Bristol (which, however, was less severe than the others) Dr Fox, besides applying cold, injected a grain of quinine under the skin in each of five different places; and at the end of half an hour the patient took ten grains of quinine by mouth. Indian practitioners are agreed that venæsection is likely to be injurious rather than useful. Dr Hunter recommends a large enema and a dose of calomel.

When, however, circulation fails, hot-water bottles must be applied instead of, or in succession to, the ice. And brandy or liquor ammoniæ (℥viij to ℨiss of water) may then be injected subcutaneously. Dr Maclean says that the application of a blister to the nape of the neck, or to the shaven head, may be of service. Artificial respiration should be resorted to in the asphyxial form of sunstroke. When convulsions set in, the inhalation of chloroform is recommended.

For *sequelæ* of heat-stroke, occurring in a person living in a hot country, removal to a temperate climate appears to be the best treatment. Dr Maclean states that at Netley there are always some cases of this kind, and that they are often very obstinate. He has seen long-continued counter-irritation to the nape of the neck, and a course of iodide of potassium relieve permanently some patients troubled with severe fixed pain in the head; but in other instances these measures have altogether failed. He gives a favourable prognosis to those who come home with epilepsy under such circumstances, as he has usually found the fits subside.

MERCURIAL TREMOR.*—Beside LEAD, of which the effects on the nervous system have been described above (p. 505), there is another poison, Mercury, which, by continued action, is capable of giving rise to certain peculiar and well-marked effects, included under the name Mercurial Tremor. The work-

* *Synonyms.*—Mercurial Shaking Palsy—Tremblement des Doreurs (Mérat), 1812—Mercurialzittern.

The following is De Haen's account of the disease among the miners of Idria more than a century ago, as cited by the late Dr Sanders, of Edinburgh:—"Deaurator, quinque et viginti annorum, horrendo artuum omnium maxime superiorum vexatus, ita ut nec comedere, bibereve solus, neque loquens amplius intelligere potuerit. Nutriendus, vestiendus, et (infantis instar) alvum urinamque positurus, adjuvandus erat: dolorum cæterum immunis."

people exposed to this affection have called it "the trembles." It is now so rare in England that a brief description will suffice.

The first thing which a man notices when he is beginning to suffer from mercurial tremor is that he is no longer sure of his arms and hands. So soon as he attempts to take hold of anything they shake. After a time every effort leads to jerking movements. When his arm is bent it is not by a continuous motion, but by fits and starts. His hand cannot be directed with precision to any object, but is thrown to one or the other side of it; when he has even grasped a thing he is often unable to let it go. Presently the lower limbs are affected; he may have to be led, and when he walks his limbs may tremble and dance about, so that (as Sir Thomas Watson says) they look as if hung upon wires. His tongue may become tremulous, his speech may be hurried, jerking (or *staccato*) and at length unintelligible; this last symptom constitutes what has been termed *psellismus mercurialis*. When the tremor has once been excited by exertion or emotion the man himself is unable for a time to stop it, but if he remains quiet for a little while it passes off and does not return until he has to make some fresh effort. Getting some other person to grasp the affected limbs sometimes controls their movements.

The commencement of mercurial tremor is generally gradual, but sometimes it is sudden, and a fit of passion has seemed to act as an immediate exciting cause of it. It is said to be more apt to occur in the cold and damp weather of winter than at other seasons.

As might be expected, salivation often accompanies the more severe forms of mercurial tremor. The teeth become loose, the gums are ulcerated, the breath is fœtid. The patient grows weak, anæmic, and thin. His teeth are said to become black, and his skin has been described as acquiring a remarkable brown hue. After a time he may begin to suffer from headache and loss of memory, he may become delirious and at length comatose, and in this way the disease may terminate fatally.

The disease in fact tends to advance so long as the patient remains exposed to the influence of the poison, but when he is withdrawn from it the tremor commonly snbsides in two or three months. Sometimes, however, the recovery is incomplete, the upper limbs being those which are most apt to remain unsteady. Relapses are frequent, and each successive attack is commonly more severe than the previous ones.

The workers in quicksilver mines—in which the ore is exposed to heat—are said to suffer severely from mercurial tremor. Formerly it was common enough in this country among "water-gilders," who employ for gilding an amalgam of mercury and gold, from which the volatile metal is expelled by the action of a charcoal fire. But water-gilding is now almost superseded by electro-plating. Another occupation in which mercury is used extensively is that of "silvering" mirrors; this, however, comparatively seldom gives rise to the tremor, probably because the quantity of metal volatilised is but small, since heat is not employed. But mercury does give off vapour to some extent even at 68° or 70°, so that it is not impossible for the disease to show itself in those who have to deal with the metal at ordinary temperatures. In 1864 a man, suffering from an extreme form of mercurial tremor, was admitted into Guy's Hospital under Dr Rees. He had merely been engaged in packing up skins which had been previously washed with an acid solution of mercury. He said that he had at one time been salivated, but some doubt would perhaps have remained as to the cause

of his symptoms, which terminated fatally, had not Dr Swayne Taylor succeeded in obtaining mercury from the kidneys, and (in smaller quantities) from the brain and liver ('Guy's Hospital Reports,' Third Series, vol. x, p. 176). This man had worked at his occupation for three years before he began to suffer to any considerable extent. It is generally said that a time varying from two to twenty-five years is required for the production of mercurial tremor. Sir Thomas Watson relates the case of a man who had been a water-gilder for seventeen years before he was attacked.

There is reason to believe that the disease is sometimes due, not to the inhalation of mercury, but to its absorption by the skin. Dr Mapother states that the anatomy porter of the Irish College of Surgeons, who at one time rubbed enormous quantities of mercury into the bodies of persons affected with syphilis, was for thirty years subject to mercurial stammering. Even the internal administration of this remedy has been known to cause tremor in those who have taken it for a very long time.

The *diagnosis* of mercurial tremor is very easy except in cases such as that which proved fatal in 1864 in Guy's Hospital, in which the patient might be unaware that he was exposed to the action of the metal. In its earlier stages it is liable to be mistaken for paralysis agitans; in its later periods for chorea. Anstie ('Lancet,' 1872, i, p. 734) mentions the case of a man who was supposed to have died of delirium tremens, until it was afterwards discovered that in his trade he had been inhaling the fumes of the acid nitrate of mercury.

Treatment.—Much has been done to prevent the occurrence of this disease in those whose occupations would otherwise expose them to it. The workshops are freely ventilated and provided with flues or chimneys. In some places it has been found practicable to interpose a glass screen between the upper part of the workman's body and the fire by which the mercurial vapour is set free. Personal cleanliness, frequent ablutions, and avoiding to take food in the room where the work is carried on, are regarded as very important, and it is said that abstemiousness in the use of alcoholic stimulants exerts a beneficial influence, though there may be a doubt whether the effects of intemperance have been isolated from those of other careless habits, which would be apt to prevail in conjunction with it.

Like other forms of tremor, that which is caused by mercury is often diminished for the time by a glass of wine or of some other stimulant. Sir Thomas Watson's patient said that when the disease was first coming on he found himself unable to get upstairs to his work until he had taken half a quartern of gin.

Among medicines iodide of potassium appears to be the most useful. It was suggested by Melsens on the same theory as for chronic lead-poisoning, and mercury has been said to have been detected in the urine while it was being taken. Sudorific remedies have been recommended, but Sir Thomas Watson says that he found tonics, and particularly iron, more useful than any other drugs in the case which he relates. Anstie found cod-liver oil very useful in several cases. Sir William Gull some years ago recorded an instance in which electricity appeared to be beneficial, sparks being taken from the spine. It was thus also that De Haen's patient (whose case was quoted above in a note) was cured.

DISEASES OF THE HEART

AND BLOOD-VESSELS

FUNCTIONAL AFFECTIONS OF THE HEART

Changes in the frequency of the pulse—Irregular and intermittent pulse—Alternate and twin pulse—Palpitation—Cardialgia—Irritable Heart—Treatment of the above conditions—Tension, length, and volume of the pulse—Syncope—Distinction from epilepsy and from sunstroke—Treatment.

ANGINA PECTORIS—*Onset—Symptoms—Event—Pathology and Diagnosis—Pseudo-angina—Anatomy—Ætiology—Prognosis—Treatment.*

IN dealing with the affections of the heart, we will take first those disorders which depend upon no discoverable organic change, and afterwards those in which more or less marked lesions are present. Under the former head (which is almost coextensive with that of *neuroses* of the heart) may be included various disorders of the pulse, palpitation and syncope, cardialgia and angina.

CHANGES IN THE FREQUENCY OF THE HEART'S BEATS.—The rate of the heart's contractions is commonly estimated by counting the number of beats of the left ventricle per minute, as indicated by the pulsations of the radial artery at the wrist. And provided that they are strong enough to be readily felt by the finger, are separated by appreciable intervals, and are equal (or at least not very unequal) in character, this answers perfectly well. But sometimes the radial pulse gives no correct information as to the frequency of the heart's action, and the stethoscope alone enables one to count its contractions satisfactorily. This is the case when they are very frequent and feeble, and especially when they differ widely among themselves in force, so that some of them fail to transmit a wave of blood so far as the smaller arteries.

With regard to the rate of the pulse it is almost impossible to avoid an ambiguity of expression, which yet is capable of sometimes giving rise to serious misunderstandings. Almost everyone speaks of the pulse as being "slow," or as being "quick" or "rapid," when what is meant is that it is infrequent or frequent. In strictness, a *slow* pulse (*pulsus tardus*) is one in which each individual pulsation of the heart takes more than usual time for its completion; a *quick* or *rapid* pulse (*pulsus celer*) is one in which the ventricular contraction is short and soon completed. In the present chapter, at any rate, we will maintain this distinction, and employ the terms frequent and infrequent whenever the number of beats in the minute is referred to.

As is well known, the frequency of the pulse is in health liable to wide variations in different circumstances. It is greater in the young than in the old, in women than in men, under exertion than during rest, in the upright posture than in sitting or lying, after a meal than when fasting. It is also liable to be increased by exertion, by emotion or by excitement. There are many persons who cannot place themselves under a medical examination, especially for life insurance, without the heart's beats at once rising to 120, 130, or 140 in the minute. One must always be alive to this source of error, which is, however, often to be avoided by counting the pulse a second time after an interval. It is said that the pulse may permanently stand at 100 in a healthy individual; but it is doubtful whether a pulse habitually above 80 ought not to be regarded as evidence of some morbid condition or tendency; and this was the opinion of Dr Latham ('Collected Works,' New Syd. Soc., vol. ii, p. 526).

(1) The diseases in which the frequency of the pulse is *increased* are numberless. They include not only all pyrexial affections, but a large proportion of non-febrile affections of whatever nature, beside the great majority of primary affections of the heart.

There are, moreover, some cases in which a very frequent action of the heart appears to constitute a disease by itself. Three such instances are recorded in the 'Brit. Med. Journ.' for 1867 by Dr Cotton, Sir Thomas Watson, and Dr Edmunds respectively. The patients were all males of middle age. In two of the cases there were several attacks at varying intervals, each lasting from a few hours to two or three weeks. The rate of the pulse was from 200 to 230, yet it was perfectly regular. The termination of the attacks seems always to have been absolutely sudden; in Sir Thomas Watson's patient, on one occasion, the beats of the heart, directly after having been counted at 216, fell to 72, exactly one third of the former number. Dr Walshe states that, in the cases of this kind, his patients, who were women, were by no means all of them hysterical or nervous, and some were distinguished by force of character. The causes which he recognised were—pedestrian excursions, the ascent of mountains, acute pain with effort to control its manifestation, and prolonged mental distress; but in not a few instances no cause could be discovered. In Dr Cotton's case the attacks were preceded by gastric disorder. The affection is not always devoid of danger. A sensation of faintness, dyspnœa, and even œdema of the lower limbs has been present in more than one instance; and Sir Thomas Watson's patient died during his fourth seizure, the heart on *post-mortem* examination being found large, as if it had been distended, while its muscular walls were very thin and soft.

Dr Bristowe had a remarkable case of rapid pulse which is quoted by Dr Broadbent in his Croonian Lectures ('Brit. Med. Journ.,' 1887, vol. i, p. 659). A young man, under twenty, had a pulse of 200 or 240 beats in the minute; and the beats were ineffectual, for he suffered from dropsy and hæmoptysis. He improved with treatment, although the pulse was liable to run up under excitement to 300. He died suddenly while playing the piano. *Post mortem* the heart was found dilated and the valves normal.

In 1870 Dr Wilks brought under the notice of the Clinical Society certain cases in which an extreme frequency of the pulse, associated with alarming dyspnœa and with palpitation, was due to nephritis, which itself was generally the result of scarlatina, although in some instances the indications of that disease had been almost unnoticed until the urine was found albuminous. In

almost every instance recovery took place within a few days, notwithstanding that the symptoms appeared to be of the most alarming character.

In the *treatment* Dr Wilks recommends purging, cupping, and salines with antimony, rather than the administration of stimulants. In his primary cases of rapid pulse in women, Dr Walshe found digitalis of no service, but nervine tonics were decidedly useful.

(2) An abnormally *infrequent* pulse is natural to some persons, in whom the heart never beats oftener than fifty or even than forty times in the minute; this does not appear to affect the prospect either of longevity or of recovering from illness. Among affections which may render the pulse infrequent are aortic stenosis, some cases of fatty degeneration of the heart, and jaundice; a *pulsus rarus* is also observed during convalescence from various acute diseases. It does not in itself indicate danger.

ARRHYTHMIA.—*Intermittence and irregularity of pulse.*—One of the more common kinds of perversion of the cardiac rhythm is that in which the heart from time to time "intermits" or leaves out one of its beats, or even two or three successive beats, its action in all other respects being perhaps perfectly regular. This, which might appear to be an alarming occurrence, is rather to be regarded as "the slightest form of derangement of the action" of the organ, to use the words of Dr Balfour. Sometimes the intermissions take place at intervals of only a few pulsations, sometimes not oftener than once in two or three minutes; sometimes they are themselves regular, sometimes altogether irregular in the frequency of their recurrence. The patient may be quite unconscious of any disturbance of the cardiac function, or he may (according to Dr Walshe) experience an instantaneous and transitory feeling of faintness. More often, what draws his attention to the fact that his heart now and then misses a beat, is that the beat which follows every intermission is unusually thumping. One may then say that besides the intermittence he suffers from palpitation. Even apart from subjective sensations it may sometimes be noticed that there is an unusual force and fulness of the pulse in the radial artery after each pause in the heart's action. And in some cases the beats of the heart are perpetually varying among themselves not only in their character but also in the rate of their repetition. Another feature may be that the organ occasionally makes a series of short ineffectual contractions in rapid succession, causing what the patient describes as a fluttering sensation within the chest. As Dr Balfour remarks, this may occur only at very long intervals, perhaps not oftener than once in several months. A curious point, confirmed by the author's personal experience, and by the statement of at least one of his patients, is, that when a person is accustomed to having his attention drawn to simple intermission of the pulse by slight feelings of palpitation in the left side of the chest, similar feelings may be occasionally experienced without any intermission taking place. Is it possible, under such circumstances, that there was a momentary contraction of fibres of an intercostal muscle, in obedience to some association of the superficial and deeper structures analogous to that pointed out by Van der Kolk and by Hilton?

Even the combination of extreme rregularity with frequent intermittence of the heart's action is sometimes a much less serious matter than might have been expected, especially in persons no longer young, who are able to lead quiet lives, and are neither called upon for active exertion nor compelled to endure the pressure of emotion. Such persons often continue to live for

years, and pass their days happily and usefully to others. This kind of cardiac disorder, however, may be a symptom of dangerous organic disease; even when no murmur can be detected with the stethoscope, there is always the possibility that some degenerative change in the walls of the left ventricle may be present.

As a rule, irregularity of the pulse should be regarded as of more importance than mere intermittence. There is, however, one kind of irregularity which signifies nothing, though it has been made the subject of grave consideration when observed in a candidate for life insurance: viz. a temporary acceleration of the pulse, for perhaps ten or twenty beats at a time, with subsequent slackenings, occurring in nervous persons, while one has one's fingers on the wrist, as the result of transient waves of excitement or uneasiness under medical examination.

But intermittence of the heart's action should never be made light of until the state of the organ itself and that of the vessels have been thoroughly investigated. If there be any undue arterial tension in particular, it should be carefully noted; in one well-marked instance intermittence of the pulse, observed from time to time during three or four years, was at the end of that period followed by indications of organic disease of the aorta and of the aortic valves. It is almost always a serious matter if the heart's beats begin to intermit after slight exertion, such as walking a little faster than usual, or hurrying for fifty or a hundred yards to be in time for a train.

Otherwise, there is no doubt that occasional or even habitual intermittence of the pulse is in many persons compatible with a good state of health, and with a fair prospect of longevity.* Dr Richardson relates that he once made an autopsy in the case of an aged man, in whom for many years the pulse had always intermitted once in eight beats; he found the heart perfectly sound and the coronary arteries normal.

Dr Walshe remarks that some people actually feel more comfortable when the rhythm of the heart is irregular than when it becomes (as it sometimes will) perfectly regular. It is also said that the tendency to intermittence passes into abeyance for the time during any illness attended with pyrexia. In one case Dr Richardson ('Trans. St And. Med. Grad. Ass.,' 1869) found the pulse intermittent in an infant on the day of birth, and this condition lasted for five years, after which it gradually disappeared; in another case he found it present in a boy five years old, who afterwards became entirely free from it. In adults, as a rule, it is due to some one of the complex conditions which are included under the term dyspepsia. Dr Balfour, indeed, is disposed to think that it is rarely dependent upon any form of indigestion except that which is associated with a gouty tendency. But in this few will agree with him. A circumstance observed with regard to this kind of pulse seems to be very suggestive as to its mode of production. As is well known, intermittence of pulse, when the result of dyspepsia, is very apt to come on after the patient lies down in bed. Now, the writer has noticed in his own person—and patients have assured him that they have noticed the same thing—that when the pulse is intermittent overnight, it often is so on the following morning also, although it becomes regular after one has risen from the recumbent posture. But the process of digestion, if it was going on at bedtime, must have ceased during the night. The probable explanation seems to be that what causes the heart's action to intermit is the presence

* "Such trivial causes will occasion them (intermittent pulses) that they are not worth regarding in any illness, unless joined with other bad signs of more moment" (Heberden).

in the stomach of solid pieces of food imperfectly masticated. It is well known that lumps of potato and other substances may remain in the gastric cavity for days together; and nothing seems to be more likely than that when one lies down such masses should fall from the greater curvature of the stomach towards the cardia, and should consequently irritate the terminal branches of the vagi spread over that part of the mucous membrane.

In some persons, intermittence of the pulse is sure to follow indulgence in tea, especially in green tea; and in others tobacco-smoking has a like effect. Again, Dr Walshe cites a case in which a shot, lodged in a bronchial tube, set up asthmatic seizures and rendered the pulse extremely irregular, both symptoms diasappearing when it was expectorated. In many persons intermittence of the heart's action appears to be traceable to overwork or to worry rather than to any other cause. Lastly, it sometimes results from the shock of some emotion, such as sudden terror or grief. Dr Richardson and Dr Balfour allude to cases in which it was set up by a railway accident or by a shipwreck; it would seem that then the intermissions gradually take place at longer intervals until they cease altogether. Dr Richardson relates the cases of two patients in whom intermittence of the pulse preceded an attack of mania, in one of them on several different occasions.

ALLORRHYTHMIA.—In some cases disturbance of the heart's rhythm shows itself, not in ordinary irregularity nor in intermission, but in the regular succession of beats of different force, or in the regular coupling together of beats in pairs. The former variety is named by Traube the *Pulsus alternans*, the latter the *Pulsus bigeminus*. By Sommerbrodt ('Deutsches Arch.,' xix) they are included together under the common name of "allorrhythmia." In English we might use the terms "grouped pulse" to denote a more or less constant succession of similar irregular contractions, and "doubled" or "twin" pulse for the more or less regular succession of a strong and a weak contraction.

An early example of the *pulsus alternans* was recorded by the author in the 'Guy's Hospital Reports,' for 1871, vol. xvi, p. 330. The patient was a woman of thirty, who came into the hospital with mitral stenosis (a condition not infrequently attended by *pulsus bigeminus*), denoted by a well-marked presystolic murmur. The usual rate of her heart's action was about 70. But sometimes it would rise to 92, and then only every other beat produced a pulse at the wrist, which accordingly was counted at 46; there was, however, reason to believe that the beats which failed to reach the radial arteries were attended with reflux into the systemic veins, inasmuch as a pulse could be felt at the root of the neck, apparently in the jugulars. It is worthy of notice that 70 is almost exactly the arithmetical mean between 46 and 92, for this fact perhaps justifies the conclusion that the efficiency of the alternate beats which were not felt in the arteries was just half that of the regular beats. At one time the allorrhythmic state of the pulse could in my patient be stopped at will, by making her walk so as to quicken the heart's action; the rhythm was then normal, but afterwards, when the heart began to slow again, it fell into the peculiar alternate rhythm. Digitalis, too, seemed at one time to be concerned in producing it, as has been suspected also in some cases of the *pulsus bigeminus* recorded by German observers. An allorrhythmic state of the heart's action, however, is not peculiar to cardiac affections; it has been observed in cases of cerebral hæmorrhage or softening (hitherto only when the right side of the brain was affected ?), and also in one case during convalescence from acute peritonitis.

How it is produced is not yet clear. Traube supposed the *pulsus bigeminus* to be a sign of the near approach of death, but on this point he was entirely wrong. This is clear from the cases that have been recorded by different observers; the patient above mentioned lived ten or eleven years after the publication of her case. Sommerbrodt seems to be near the mark when he compares this state of the heart with the modification of breathing which is associated with the names of Cheyne and Stokes. Just as the latter depends upon rhythmical changes in the degree to which the respiratory centre is stimulated by the circulating blood, so the latter may perhaps result from a like influence of the blood supplied to the cardiac ganglia. It is to be noted that when the pulse is at times allorrhythmic, it is apt at other times to be arrhythmic, *i. e.* irregular and intermittent. Allorrhythmia has been frequently observed as a sequel of epileptic attacks, and Tripier, of Lyons, has collected a large number of these cases.

Palpitation of the Heart.—In ordinary circumstances and in a state of health, one is unconscious of the heart's action—if one becomes aware of its pulsations, palpitation is said to be present. This need not necessarily be a morbid affection; it may occur to any person after great exertion, or under strong emotion. But, apart from such causes, it may be due to various kinds of disturbance of the heart; and it sometimes appears to be the chief or even the only thing that a patient complains of, so as to constitute a disease by itself. In some cases in which the cardiac pulsations seem to the patient to be extremely violent, the physician may find, on placing his hand over the left side of the chest, that they are in reality perfectly quiet and natural in character. Much more often the subjective sensation corresponds with the fact that they are greatly increased in force, so as to shake the chest, or the entire body, or even the couch on which the patient lies. This distressing palpitation may be accompanied by violent throbbing of the carotids, a sensation as though "the heart were jumping into the throat" or "the eyes bursting from the sockets," flashes of light before the eyes, dizziness, faintness, or an indescribable sense of discomfort in the region of the heart, rarely amounting to actual pain. The attempt to lie on the left side often greatly aggravates the symptoms. There is often extreme anguish with a fear of impending death. On examination the apex-beat is seen to be in its natural position, but it occupies too extensive an area. Dr Walshe describes the impulse as feeling like a *blow*, with even somewhat of a heaving character, if the heart is well nourished; like a *slap*, if the organ is feeble. The area of percussion dullness is usually normal in extent, but in prolonged paroxysms there may be some increase of it to the right of the sternum. The sounds are loud and clear, with a metallic ringing character; the first sound can sometimes be heard at a distance of some inches from the chest. Dr Walshe speaks of a basic systolic murmur as of frequent occurrence in patients who are in the slightest degree anæmic. He is also disposed to suspect that a transitory condition of mitral regurgitation may be present, causing a systolic murmur at the apex; but one ought to scrutinise very carefully the relations of any such murmur to the respiratory rhythm, for cardiac palpitation seems extremely likely to give rise to a peculiar form of murmur which will be described elsewhere as resulting from disturbance of the edge of the left lung by the heart's movement. The pulse is not necessarily increased in frequency; Dr Walshe says that in vigorous and plethoric subjects its rate may be normal or even below normal.

Palpitation of the heart is commonly present in various forms of organic cardiac disease, and the physical signs are then of course modified by those which belong to the particular affection that may be present. It may also accompany other forms of functional or neurotic disturbance of the organ; in exophthalmic goitre it is one of the principal symptoms. In these circumstances it may be more or less persistent. On the other hand, when it occurs by itself, it is usually paroxysmal. The duration of an attack may then be from a few minutes to several hours. Dr Walshe remarks that it may terminate with an abundant flow of watery urine, and that as it subsides the patient often falls asleep. Palpitation about puberty is often due to the heart not developing in proportion to the bodily growth.*

It is especially during early adult life that palpitation is apt to occur as a substantive disease. Nervous hysterical women are very liable to it; but the most severe cases of all are seen in excitable youths. It may be the result of exhaustion from over-study, from sexual excesses or masturbation, or from the abuse of alcoholic stimulants, of tobacco, or of strong tea. But in some cases none of these causes can be made out. It is then often due to overloading of the stomach with imperfectly masticated food, especially shortly before bedtime. It is probable that, like intermittence of the pulse, palpitation may have such an origin, even when there is no sense of fulness or discomfort at the epigastrium, nor any other of the common symptoms of dyspepsia. It is especially when the affection recurs night after night when the patient is in bed that this cause should be suspected. In other cases it comes on during exertion, as when the patient begins to walk, even slowly and on the level. Dr Balfour says that one characteristic of the nervous or functional nature of the palpitation in such circumstances is that it disappears if he exerts himself a little more, as by taking a short run. In other instances palpitation may be noticed to subside under any kind of pleasurable emotion.

Cardiac pain.—Pain is a frequent, though far from a constant, symptom of many affections of the heart, both functional and organic; on the whole, perhaps it is more marked in the former than in the latter. Its most usual seat is a spot a little outside the left nipple, beneath the fourth intercostal space, or the fifth rib; but it may be situated nearer the sternum, or to the right of it. Dr Walshe says that it is evidently deep seated, and not in the cutaneous nerves; but the correctness of this distinction is doubtful (*cf.* p. 419). Cardialgia is generally more or less paroxysmal in character and is often described as sharp or lancinating, sometimes as burning, tearing, or cutting. There may also be a constant dull heavy pain, and in some cases this exists by itself. It is often increased by exertion or fatigue, and it generally undergoes aggravation when palpitation occurs. It often radiates widely over the side of the chest, up into the neck, into the axilla and down the left arm, even sometimes down the right arm. When it thus affects the arm it is apt to be associated with a sensation of numbness or tingling; it sometimes ceases abruptly at the inner side of the elbow, sometimes extends down the forearm to the inner side of the hand. In some cases its principal seat is behind, near the angle of the left scapula. Dr Walshe says that tenderness of the surface is absent; pressure upon the principal spot rather relieves than increases the pain. Da Costa, however,

* See Bowditch on "The Growth of Children," and Beneké's observations on the volume of the heart at different ages, quoted by Dr Pitt ('Brit. Med. Journ.,' Nov. 27th, 1886).

in describing some cases to which I shall refer presently, says that the cardiac region was hyperæsthetic, especially after attacks of palpitation.

A modification of cardiac pain described by Dr Walshe is one which comes on when the patient bends forward, as in pulling on his boots, and is relieved by stretching out the chest wall and pressing on the surface. Dr Walshe has observed it more often in elderly than in young persons. He thinks that it may depend upon "twisting of the præcordial costal cartilages, which have lost the flexibility of youth," but it seems more likely attributable to upward pressure upon the heart through the diaphragm. Possibly this kind of pain ought to be regarded as one of the slighter forms of angina pectoris; at any rate, it is not seldom present in that disease.

Irritable heart.—The close connection between the various forms of functional or neurotic disturbance of the heart is well illustrated in a very interesting paper by Da Costa ('Am. Jour. of Med. Sc.,' 1871) on what he terms "irritable heart." This paper is further of great importance as containing what is almost complete proof of the gradual passage of such an affection into a condition of organic slowly progressive disease, by the heart undergoing hypertrophy. It is based upon no fewer than three hundred cases of soldiers in the army of the United States during the Civil War. The general history of these men is that having been called from civil pursuits into active service without previous training, they became liable to attacks of palpitation, to more or less severe pain in the chest, of a sharp and stabbing, or of a dull aching character, and to dyspnœa on exertion, so that they became unable to keep up with their comrades and were distressed by the weight of their accoutrements. On examination the pulse was found to be greatly increased in frequency; it was much influenced by position, so that there was sometimes a difference of thirty beats or more between its rate during standing and that during lying down; in some instances it was intermittent or irregular. The men themselves often looked strong and healthy, though their hands were apt to be bluish and mottled, and to be easily made pale by pressure. The cause of the affection was sometimes hard service in the field, particularly excessive marching. But in many cases it was directly brought on by an attack of diarrhœa, not sufficient to interfere with duty; or by fever, necessitating a few days' stay in hospital. The patients were generally young men, from sixteen to twenty-five.

The course of this affection was usually slow. But after some months of treatment it often ended in complete recovery, so that active exercise of all kinds could be borne without discomfort. In other cases the cure was imperfect, there being still a liability to cardiac symptoms on exertion. Hypertrophy of the heart was believed to have developed itself in twenty-eight cases out of two hundred.

Da Costa's observations with regard to the *treatment* of his cases are of great interest and importance. Rest was found to be very useful. Making the patient lie down for several hours daily often led to strikingly good results; and two men who were kept in bed—one by an attack of dysentery, the other by a broken leg—improved very rapidly.

General treatment.—Among ordinary patients, in whom we have seen that functional disorder of the heart is often due to overloading of the stomach, regulation of the diet is a very essential part of the treatment. Great moderation in the use of stimulants, or even total abstinence, should

be insisted on, in most cases. Care must be taken that neither tobacco nor tea is used to excess; all forms of excitement, including sexual indulgence, must be kept under control. When there is anæmia, tonics such as iron, zinc, or quinine may be prescribed with advantage.

Among drugs intended to affect the heart directly, digitalis is by far the most useful. This was Da Costa's experience, and I think that it is in entire accordance with that of other observers. There were, however, a few cases in which it had no effect either in modifying the frequency of the pulse, or in correcting its irregularity. In cases of simple palpitation, such as are described at p. 902, one might have doubted, on theoretical grounds, whether digitalis would do good. Dr Walshe, however, says that clinical experience is in its favour. Other medicines which may be of service are the bromide of potassium or of ammonium, hyoscyamus, cannabis indica, ammonia, and almost any form of ether, but particularly the spirit. ætheris comp. In hysterical females, assafœtida, musk, or valerian are useful. Schrötter recommends the application to the cardiac region of cloths wrung out in cold water, or even of an icebag; but Dr Walshe says that this is a dangerous practice, especially if the rhythm of the organ be in the smallest degree affected.

For palpitation accompanied by great irregularity and intermittence of the pulse, it is agreed by all writers that alcohol is one of the best remedies. Dr Walshe says that from a teaspoonful to half a wineglassful of brandy should be given, according to circumstances. It is, however, very important to take care that dram-drinking shall not become a habit. Digitalis is often of great use in steadying the heart's action, and Da Costa found that belladonna or atropine was especially likely to do good in cases in which the pulse intermitted.

Aconite seems to be of little or no service in cases of mere functional palpitation, but Da Costa obtained striking results from it when the heart was beginning to undergo hypertrophy. It often exerted a marked influence upon the force of the cardiac beats, without diminishing their frequency, whereas exactly the opposite effect was produced by digitalis. Consequently, in suitable cases the two drugs were given together with great advantage. Veratrum viride seemed to be intermediate in its action between them.

A point on which Da Costa laid great stress in the management of his patients, and which is doubtless of no less importance in like cases occurring in civil practice, was the maintenance of great care during convalescence. Before allowing the men to return to their regiments, he tested them by running and by other exercises, so as to see how the heart bore itself under strain. He gives reports of some cases which came under notice again after an interval of several years, and in which no relapse had occurred.

Tension, Volume, and Length of the Pulse.—An important character of the radial pulse is the resistance which it offers to compression by the fingers. This was well recognised by the older physicians, who used the names *pulsus durus* and *pulsus mollis* to denote degrees of tension, and called a small incompressible pulse "wiry," a small and compressible one "thready." These terms were still used after increased attention was given to the symptoms of disturbed circulation on the introduction of cardiac auscultation; but they were not better understood, until the researches upon arterial blood pressure by Ludwig and the subsequent invention of the sphygmograph by Marey gave more definite meaning to the terms previously in use.

Even now we have no trustworthy method of measuring the blood pressure in an unopened artery. Neither the pressure necessary to obtain a sphygmographic tracing nor even the characters of the tracing, beyond a certain point, will tell us this.

The tension felt by the educated finger placed on the radial artery depends first on the force of the left ventricle behind, secondly on the resistance in the capillaries and contracted arterioles in front; and this feeling of pressure or tension will be modified by the softness and elasticity or the hardness, rigidity, and thickness of the walls of the radial artery.

The highest tension is commonly found in the most chronic form of Bright's disease when there is increased resistance in front and a hypertrophied ventricle behind. But high tension is also found in many cases of early and acute nephritis such as occurs after scarlatina, and in a less degree in other acute inflammations, especially serous and parenchymatous. It is also met with during pregnancy, in some cases of chlorosis, and in full-blooded, free-living patients who find constant relief in blue pill, colchicum, and a purge.

A low tension with a relaxed artery is characteristic of pyrexia generally, particularly of the specific fevers, and of acute rheumatism.

The pulse is large or "full" with high tension in some cases of pneumonia and meningitis, and in early cases of acute Bright's disease. A more common condition is the short, full pulse with low tension, which when fully developed becomes the collapsing or "water-hammer" pulse.

A small pulse with high tension, shown by the length of the systolic expansion and the slight degree of the diastolic fall, is the opposite of the last variety, and constitutes the "persistent" and hard pulse of chronic renal disease.

A small, weak, and compressible pulse, which is also irregular, is one of the characteristic symptoms of mitral regurgitation.

The same hardness which is felt by the finger in the contracted and thickened artery of Bright's disease, may be produced by calcareous (or even ossific) degeneration of the middle coat of the radial artery, or by the more common atheromatous degeneration of large arteries. The visible pulsation and emptiness during diastole which in young subjects with elastic arteries is indicative of aortic imperfection, denotes in older persons a want of elasticity of the arterial coats, due to degenerative changes.

The pulse of the radial artery may be always weak, and its lumen easily compressible, without there being any lesion of the heart or any deviation from health. Hence "a poor pulse" is often a deceptive guide in a patient seen for the first time, or in a candidate for life assurance. A short, "slapping" pulse—*pulsus celer*, is associated with low tension and is often felt after depletion by hæmorrhage or purgation.

If the two radial pulses differ markedly in size or in force of beats, there is either some abnormal distribution of the arteries, or obstruction from a tumour pressing from without, or atheroma or some other lesion within. In most cases the tumour or internal lesion is aneurysm.

Syncope.—In the first chapter of this work the symptoms were described which accompany death by syncope or by failure of the heart's action; but it is necessary to return to the subject in this place, if only for the reason that in some cases the cardiac functions are for a short time completely arrested, without any danger of a fatal issue.

The symptoms of fainting need not be recapitulated in detail (see p. 16);

indeed, they are commonly known. But it must be pointed out that they vary greatly in degree in different cases. Sometimes, after having suffered for some minutes from giddiness and nausea and "faintness," the patient just for an instant loses himself more or less completely and then gradually recovers. Sometimes he remains unconscious for a considerable time. Dr Walshe disbelieves in the possibility of recovery after the sounds of the heart have ceased to be discoverable during so long a period as five minutes. But it is of course not uncommon for "fainting fits" dependent upon an enfeebled action of the organ, to last for an hour or longer, and yet to end favourably. The subsidence of an attack is ushered in by "gasping or rather sighing respirations at long intervals, and by gradual return of pulse, consciousness, and colour. Sometimes vomiting or discharge of flatus, convulsions, or profuse perspiration takes place at the time of returning consciousness." Dr Walshe, from whom these details are taken, thinks that the phenomena accompanying the re-establishment of life are generally painful and distressing, whereas in many cases the passage to unconsciousness is pleasurable.

There are other instances in which the stoppage of the pulse and the interruption of the mental faculties are absolutely sudden, and in which the resumption of the heart's action and the recovery of consciousness are no less instantaneous, while there are no subjective sensations whatever. But although seizures of this kind are spoken of as "fainting fits" by unprofessional persons, they are, probably, always of cerebral origin, and in fact belong to the minor form of epilepsy.

One great distinction between the attacks which are of an epileptic nature and those which may properly be referred to fainting, is that the former generally, if not always, occur without any definite exciting cause. On the other hand, true syncope is, as a rule, clearly traceable to some disturbing cause, though it may be apparently of a trifling character; the heated air of a crowded room, the sight of blood (even from a cut finger), the strong odour of flowers, the introduction of a catheter, may each cause fainting in certain persons. It is especially apt to occur in young adults, in women rather than in men, but it may frequently be seen in healthy and vigorous youths.

Among the causes of the graver forms of syncope, such as are apt to prove directly fatal, are hæmorrhage and various organic diseases of the heart and aorta, pulmonary embolism, and occasionally the too rapid withdrawal of ascitic or pleuritic fluid by tapping.

The "cardiac" variety of sunstroke must also be mentioned. In this the sufferer gives no sign of illness until he falls, gasps, and perhaps at once expires before anything can be done to help him. Dr Maclean says that this is the form most often seen in soldiers exerting themselves in the heat of the sun when dressed and accoutred (cf. p. 892).

In the *treatment* of a faint, the first thing is to place the patient in the open air or near a window and to make him lie down with the head as low as the shoulders; the clothes must be loosened about the throat and chest, and the crowd of sympathising friends must be sent away. A bottle of ammonia may be held to the nostrils, or if this be not at hand a bunch of feathers may be burnt and the fumes inhaled. Cold water may be poured upon the face, and if the stomach be overloaded, an emetic of mustard should be administered; for this, as Anstie says, "has a powerfully rousing influence upon the heart." Sal volatile, brandy, ether, or a tumbler of cold water may be given by the mouth; or, in severe cases, when the patient

continues unconscious, fifteen or twenty minims of either ether or brandy may be injected hypodermically.

Other measures, especially applicable when death seems to be impending, have been enumerated before (p. 17).

ANGINA PECTORIS.*—Under this name is known a very severe form of cardiac pain, occurring in sudden short paroxysms, and accompanied by a sense of impending death, which is not seldom actually realised. It was first described in 1768 by Heberden, who had observed several cases.† In its well-marked and typical form angina pectoris is rare. Cases are not infrequent, which more or less closely resemble it in the character of the pain, although in these there is not the same danger to the patient's life. Whether or not these should be classed under the same heading is a question that can be satisfactorily discussed only after the pathology of the affection has been considered. The following description refers to the classical examples of the disease.

Onset and exciting causes.—Angina pectoris is almost always absolutely sudden. The first attack usually occurs while the patient is walking, especially on rising ground, or with a strong wind against him, or shortly after a meal (Dr Walshe says, most often after the earliest meal in the day). But sometimes (as in the case of Arnold, of Rugby, recorded by the late Dr Latham) a person who has never suffered from it before is awakened by the first attack from his sleep. Subsequent seizures are apt to be brought on by comparatively slight causes. Thus the patient may gradually find that one form of exertion after another is unsafe for him. Or emotional excitement may give rise to it, as in the well-known case of John Hunter, who died within the walls of St George's Hospital in consequence of a dispute at a meeting there, of which he had previously foreboded that the result would be fatal. Dr Walshe has even seen a paroxysm brought on by emotion of a pleasurable character. In many instances the seizures, which at first took place only during the daytime, afterwards begin to recur even in the night. Dr Latham alludes to one patient who was attacked as soon as he lay down. The act of stooping, as to pull on the boots, or even in washing, is a frequent cause of the attacks of angina. In some cases they are brought on by such slight efforts as coughing, defæcation, or the hasty swallowing of cold water.

Symptoms.—The chief seat of the pain in angina pectoris is usually behind the lower part of the sternum, rather to the left than the right side; sometimes behind the middle or the upper part of that bone. When the patient attempts to describe it, he speaks of it as gnawing, tearing, or lancinating in character. But in many cases it seems to be altogether indescribable—a torture so intense that he feels that any increase of it, or even its continuance, must bring his life to an end. The pain often spreads

* *Synonyms.*—Pectoris dolor—Breast-pang—Syncope anginosa (Parry)—Asthma dolorificum (E. Darwin)—Cardiodynia—Neuralgia cordis (Laennec)—Hyperæsthesia plexus cardiaci (Romberg)—Angine de poitrine—Brustbräune.

† "The seat of it and sense of strangling and anxiety with which it is attended make it not improperly called angina pectoris." 'Commentaries on the History and Cure of Disease,' 1782, chapter lxx. The first description was in the 'Medical Transactions' of the College of Physicians for 1768. Some French writers have set up a claim of priority for one of their countrymen, Rougnon, on account of a letter written by him to Lorry a few months earlier, in which is related the death of a cavalry officer, M. Charles, by what was probably the same disease. But Dr Gairdner points out ('Reynolds' System,' iv, p. 537) that in so far as the account of a single case can be held to have anticipated Heberden's observations, the merit really belongs to Morgagni, who recorded a similar instance in a Venetian woman early in the eighteenth century.

round, generally through the left side of the chest, to the spine. It is sometimes accompanied by a sense of constriction, as though the sternum were forcibly drawn backwards: Dr Gairdner cites the case of a medical man in whom there was a subjective sensation as though the front of the chest were "bulged out in a convex prominence, terminating suddenly at the lower end of the sternum in a sharp and deep depression." Very often the pain radiates upwards into the neck or towards the occiput, down the left arm to the elbow or into the fingers, occasionally down the right arm, into the lower limbs or into the testes. In these distant parts it may be accompanied by feelings of tingling or of numbness. In some cases, as in one observed by Dr Walshe, it takes a course the reverse of what is usual, beginning at the left wrist and extending upwards to the heart. In the præcordial region there is often tenderness to pressure; but sometimes friction gives relief.

A patient attacked by this affection is instantly arrested in whatever he may be doing; if walking he is motionless until the seizure passes off; if standing, he dares not sit down. It is, however, a curious circumstance that some persons, after having been pulled up by the pain three or four times at the beginning of a walk, will afterwards go on with ease for several miles. The feeling of constriction in the chest may cause him to speak of experiencing a "want of breath" or a "sense of suffocation;" but all observers are agreed that there is never any dyspnœa in the proper sense of that term, and that no lividity of the features shows itself. The breathing may indeed be somewhat increased in frequency, but this is because the patient instinctively keeps the thoracic movements as shallow as possible, for fear of increasing the pain. By an effort of the will he can, if he chooses, freely expand the chest. There are even exceptional cases in which drawing a deep breath gives momentary relief to his suffering.

With regard to the state of the heart's action and of the pulse during a paroxysm of angina pectoris, the accounts of different writers have differed widely; and what we shall find to be the probable pathology of the disease favours the conclusion that all cases are not alike in this respect. It is said that the impulse and the sounds of the heart are sometimes unaltered in character throughout the seizure, and the pulse regular and neither frequent nor weak. Dr Walshe, speaking from his own experience, declares that at least towards the close of a paroxysm, when the patient is about to recover, there may be no acceleration nor any irregularity of beat. But Parry long ago described the pulse as being more or less feeble, according to the violence of the attack; and Dr Gairdner expresses the same opinion. In many cases it is expressly noted that the pulse has been small and irregular in rhythm, but not always increased in frequency, and sometimes morbidly infrequent. The failure in the circulation is also shown by deadly pallor of the face, by coldness of the limbs, by the presence of clammy perspiration. In cases about to prove fatal, the pulse becomes imperceptible a little while before death.

The mental faculties usually remain unimpaired throughout the seizure; but after its subsidence the patient is said sometimes to have no remembrance of anything except the intense agony which he has undergone. Thus the state of the brain in angina pectoris would appear to resemble that during collapse, and not that during syncope. But Dr Walshe says that the sight sometimes fails more or less completely. There may be slight convulsions, or even tonic spasms of a severe kind, with opisthotonos.

There is in some cases violent and continued eructation, or vomiting; or the whole abdomen may become distended with flatulence. A copious flow of watery urine sometimes occurs as the attack is passing off. Trousseau relates a case in which the paroxysms, though frequently repeated, and though each lasted only about a minute, were always accompanied by an irresistible desire to micturate.

The duration of an attack of angina pectoris is usually a few seconds or minutes; but attacks may recur again and again for an hour or longer. A patient of the author's remained for many hours stooping over the end of a couch, refusing to move for fear of the return of the pain. But sometimes when a seizure occurs during walking, it ceases as soon as the patient stands still. Trousseau remarks that very different attitudes are assumed in different cases. One patient will be motionless on his back, another will incline backwards on his chair or on pillows, a third may place himself on all fours, resting on his knees and his elbows, a fourth may bend forwards as far as possible.

Fatal event.—When this disease proves fatal, the heart is found at the autopsy to be relaxed and flabby, even though there is marked cadaveric rigidity of the muscles generally. Dr Walshe says that there is an almost complete absence of blood from the cardiac cavities, which fact certainly looks as though a ventricular systole not followed by an active dilatation had been the last act of life. Sometimes death appears to be absolutely instantaneous; Dr Walshe relates an instance in which the patient had been reading quietly in bed, and in which the thumb and the forefinger were found in the pamphlet on which he had been engaged, the bedclothes being also quite undisturbed. In some of these cases, even where there have been former attacks attended with severe pain, it seems probable that the fatal seizure must have been so brief as to be painless; and one may perhaps suspect that sudden death, in persons who have never been known to suffer from angina, is not seldom essentially of the same nature. But sometimes death is more gradual, being preceded by gradual failure of the pulse, laboured breathing, and unconsciousness. We have seen that, as a rule, the paroxysms of angina pectoris return again and again; there is generally an interval of some years between the first attack of the disease and its fatal termination. Whether a single paroxysm ever occurs without being followed by any others, seems to be doubtful, though it is certain that the patient himself may do a great deal to prevent their recurrence by avoiding exertion and emotion. Dr Walshe speaks of having himself seen a patient who appeared to have been first attacked twenty-four years previously; and there is some reason to believe that John Hunter had begun to have seizures of angina twenty years before his death. But the longest case is one related by Dr Murrell, in which the patient had suffered for thirty years, the diagnosis of angina pectoris having been formally given by Sir Risdon Bennett twenty-six years before he came under Dr Murrell's notice. In some instances the disease returns very frequently, so that the whole number of paroxysms must be very large. On the other hand, Dr Walshe relates an instance in which there were only three, one being a year, and the other half an hour, before the third, which proved instantaneously fatal Dr Latham met with two cases in one of which death occurred fourteen days, in the other ten days, after the first attack. The most rapid (not instantaneous) case on record is probably that of Arnold, of Rugby, who, having never suffered from angina, went to bed on the 11th of June, 1842,

in apparent health, but was first seized with the pain at about five a.m. and died about a quarter past seven.

Pathology.—The pathology of angina pectoris is still obscure. Following Laennec, Romberg, and Friedreich, Trousseau and Anstie maintained that it should be regarded as a neurosis, or a "visceral neuralgia." The same opinion is formally upheld by Eulenburg in 'Ziemssen's Handbuch,' but perhaps a more correct statement would be that he rejects it, since he would exclude from the disease, in its "purely nervous" form, the very cases involving danger to life, on which the foregoing description is based, and which from the days of Heberden to the present time have been regarded as the true and typical examples of it.

For a neuralgia to prove habitually fatal is without precedent; moreover, angina pectoris differs from all neuroses in being generally, if not always, associated with the existence of organic lesions in the heart or in the great vessels, although it would seem that no one lesion is constantly present. This, at any rate, is true of the cases that destroy life, and that occur in middle-aged or old persons, or sometimes in young adults, as in a case of Dr Balfour's, to be presently mentioned. Lastly, it is unlike a neuralgia to show, as angina does, an enormous preponderance of males over females—as many as ten of the former to one of the latter—among those who are attacked by it. On the other hand, it is right to mention that Anstie declares that those who suffer from the disease are always of nervous temperament, this being shown by the frequent occurrence of other neuroses in different members of their families; and, like Trousseau, he insists on the existence of a relationship between angina pectoris and asthma.

Within the last few years observations have been made which tend to show that angina pectoris may depend upon organic lesions affecting the cardiac nerves and ganglia. The earliest record of the occurrence of such lesions, indeed, dates back to 1841, when Heine ('Muller's Archiv,' 1841) published a case treated by Skoda, in which Rokitansky made the autopsy and found the right phrenic nerve, the *N. cardiacus magnus* and the descending branches of the left vagus, each involved in pigmented nodules, doubtless altered lymph-glands. The symptoms during life, however, consisted, not in paroxysms of angina, but in attacks of intermittency of the heart's action, continued during a period of from four to six beats, and attended with a feeling of inexpressible anxiety. In 1864 Lancereaux ('Gaz. Méd.,' 1864) had an opportunity of examining the body of a man who had long suffered from angina pectoris and who had at last died suddenly; he found a raised patch in the aorta between the orifices of the coronary arteries, with injection and thickening of the corresponding part of the external coat of the vessel; in this injection the adjacent cardiac plexus took part, and some of its fibres were surrounded by a nucleated material, and presented a greyish, finely granular appearance of their myelin. Similar changes in the nerves are recorded and figured by Peter ('Traité des Maladies du Cœur,' 1883) in two cases which came under his care, and he cites a fifth instance ('Bull. de la Soc. Clin.,' 1878).

Since the year 1867, however, clinical experience has taught in a very decided manner that the immediate exciting cause of the paroxysms of angina pectoris is a sudden *rise in the tension* of the systemic arteries. A similar view had been previously suggested by Traube. But what first proved its correctness was a series of observations made by Dr Lauder Brunton on a patient in the Royal Infirmary of Edinburgh. The man was

affected with aortic regurgitant disease, and was liable to frequent attacks of angina-like pain. Dr Brunton found that during these attacks the sphygmographic tracing of the pulse became rapidly altered, the curve being lower, the dicrotism disappearing, and both the ascent and the descent being more gradual—changes indicative of a marked increase of arterial tension. These observations led Dr Brunton to propose the inhalation of nitrite of amyl as being likely to relieve the pain, and the trial of this agent was attended with brilliant success. The pallor and the coldness of the face and of the limbs that accompany severe seizures can hardly be cited as evidence in themselves of arterial spasm, for failure of the heart's action might produce the same effects. Trousseau, however, draws attention to the fact that the pallor is sometimes followed by a reddish or livid-bluish hue, and the same thing was noticed in one case by Anstie ('Trans. Clin. Soc.,' vol. iii) and attributed by him to paralytic dilatation succeeding spasm of the arterioles.

Under the name of "Angina Pectoris vasomotoria" Nothnagel has recorded ('Deutsches Archiv,' iii) a series of cases which seem to have an important bearing upon this question. Their peculiarity lies in the fact that the earliest and most conspicuous symptoms of the paroxysms from which the patients suffered were coldness and pallor with numbness and stiffness of the limbs; the palpitation, the feeling of oppression at the chest, the giddiness, the sense of impending death, being all apparently secondary and attributable to the increased efforts which the heart was called upon to make to overcome the resistance opposed to it. The attacks were also definitely traceable to external cold, and were relieved by hot foot-baths and frictions; in fact, the state of the peripheral circulation seems to have been very similar to that which in other patients leads to paroxysmal hæmoglobinuria. Pain appears to have been a much less marked symptom than in ordinary angina; however, a dull pain is mentioned, seated chiefly in the cardiac region, but in one case extending over the whole of the left side of the chest, and sometimes down the left arm.

On the whole, Nothnagel's cases prove that a sudden increase of tension in the peripheral arteries due to a cause acting upon the body from without is capable, in some persons, of giving rise to phenomena approaching those of a paroxysm of angina pectoris. The next question is how we are to suppose the arterial spasm to be brought about in the more usual form of the disease. Now, if the anatomical researches of Lancereaux and Peter are to be credited with the significance which these observers attach to them, nothing seems to be more simple than to suppose that from the cardiac nerves and ganglia there is transmitted to the vaso-motor centre an impression which causes it to throw the muscular walls of the smaller vessels throughout the body into contraction. But it is necessary to exercise a good deal of caution before we adopt such a view. The lesions discovered by the French pathologists were after all in every instance secondary. If the nerves lying adjacent to areas of chronic thickening and induration at the base of the heart (or, indeed, elsewhere throughout the body) were to be systematically dissected, is it not probable that they might very often be found involved in the adhesions, even when no symptoms had existed that could possibly be traced to them? It is surely significant that, like Rokitansky, Peter found the phrenic nerves affected in exactly the same way as the cardiac nerves.

One point in which the phenomena of the paroxysm of angina pectoris seem to differ from what might be theoretically expected on the view that it

is essentially dependent upon an increased tension in the peripheral arteries, is its not being invariably, or even generally, attended with a reduction in the frequency of the pulse; among Nothnagel's cases, too, there is only one in which a fall from 80 to 64 or 60 beats in the minute is noted.*

It must, however, be remembered that in the severe and dangerous form of angina pectoris, which is regarded as typical of the disease, organic changes in the heart and large vessels are usually, if not always, present. Among recent writers Anstie stands almost alone in maintaining that fatal seizures may occur in persons in whom no such disease exists. As a rule, the most conspicuous lesion is either a soft, flabby, or fatty state of the cardiac muscle, or else a chronic inflammatory change in the coats of the aorta at or near its origin, leading to atheroma, to calcification, and perhaps to aneurysm: Dr Gairdner has specially insisted upon the frequency with which symptoms of angina accompany even small aneurysms, arising very near the heart and projecting into the pericardium. It has, however, long been a question whether one ought not to regard, as more especially connected with angina pectoris, another lesion which is undoubtedly often found associated both with fatty heart and with arteritis deformans of the aorta, namely, obstruction of the coronary arteries. This was first suggested by Jenner, in a letter to Parry, and afterwards in one which he addressed to Heberden in 1778, but which he did not send, out of consideration for the feelings of his friend John Hunter, whom he rightly believed to be at that time a sufferer from angina. Sometimes the coronary arteries have their orifices more or less completely obliterated by disease of the aorta itself, their coats in the rest of their course being healthy; sometimes they are converted into thick calcified tubes in nearly their whole length.

But it would be an untenable position to maintain that obstruction of the coronary arteries is constantly present in cases of fatal angina, either with or without other more conspicuous lesions of the heart or of the aorta. And it is no less certain that these vessels are often found in the *post-mortem* room to be greatly narrowed when there were no symptoms of angina during life. Again, the long duration of the disease in some cases seems to be inconsistent with the idea that any of the organic lesions above described can have existed throughout its whole course. Perhaps after all it may be that the paroxysms of angina owe to the lesions in question their severity and their tendency to prove fatal, but do not stand to them in the direct relation of effect to cause. For, if the disease be regarded as a struggle on the part of the heart to overcome an excessive resistance in the arteries, enfeeblement of the cardiac muscle, whether as the result of fatty change or of a mere deficiency of blood supply, cannot but add greatly to the embarrassment of the heart and the danger of the patient.

From this point of view it seems probable that many instances of the affection which Dr Walshe describes as "pseudo-angina," and which is admitted to be of far more frequent occurrence, are fundamentally of the same nature; but it would still doubtless be necessary to exclude cases dependent upon hysteria or upon flatulent distension of the stomach. Within a single year the writer saw two young clerks in the same London bank, each of whom described attacks that appeared indistinguishable from those of angina pectoris, although their age rendered it very unlikely that

* Landois's physiological explanation of angina, on which Nothnagel's theory is founded, will be found in Eulenburg's article in 'Ziemssen's Handbuch' (Bd. xii, 2, S. 45—48), and also in Ross's 'Treatise on Nervous Diseases' (vol. i, p. 731).

the affection would prove dangerous to life. But that the age of the patient does not always form a safe criterion is well shown by a case of Dr Balfour's, that of a man, aged only twenty-four, who died after four months' illness with paroxysmal pain in the epigastrium, and in whom (as had been correctly diagnosed during life) the base of the aorta presented a ring of atheromatous thickening, by which the mouths of the two coronary arteries were greatly narrowed. Such a case, however, is altogether exceptional; for, as Dr Walshe says, angina pectoris (exclusive of his pseudo-angina) is rare before the fiftieth, and excessively so before the fortieth year.

Morbid anatomy.—Symptoms of angina ending in death have been found associated most often with atheroma, thickening, obstruction or obliteration of the coronary arteries. It was so in Hunter's case (1793). "The coronary arteries had their branches which ramify through the substance of the heart in the state of bony tubes, which were with difficulty divided by the knife, and their transverse sections did not collapse" (Home). There was atheroma and dilatation of the ascending aorta and calcification of both aortic and mitral rings, but the valves were probably competent and the cavities were not dilated. Sir Everard Home notes with much emphasis two "milk-spots" on the surface of the heart, but omitted to weigh it ('Life,' by Adams, p. 203).

In Robert Hall's case (1831) Dr Pritchard reported "the muscular structure (of the heart) to be soft, like macerated cellular membrane; the left ventricle was judged to be one third larger than usual. The aorta contained in several places patches of bony matter." The valves are not mentioned nor the coronary arteries (Memoir by Gregory, note D.).

In Arnold's case (1842) Dr Bucknill and Mr Hodgson found the aorta and all the cardiac orifices healthy. The muscular structure of the heart in every part was remarkably thin, soft, and loose in its texture. The walls of the right ventricle were in some parts not much thicker than the aorta. There was but one coronary artery, which with some difficulty admitted a small director ('Latham's Lectures,' vol. ii, p. 377).

In the case above quoted from Dr Balfour (1877) the heart weighed thirteen ounces, the cavities were slightly dilated and hypertrophied, the mitral and tricuspid orifices enlarged, the aorta atheromatous, and the coronary arteries "both so extremely contracted as barely to admit the point of an ordinary surgical probe." The muscular structure was healthy to the naked eye, and under the microscope "presented no abnormality, except the presence of a considerable number of reddish-brown pigment granules in some of the fibres" ('Clinical Lectures,' p. 301). In a second case of angina, in a woman of eighty, Dr Balfour found disease of one coronary artery, with dilated ventricle and thin, pale, fatty muscle.

Angina (or symptoms clinically resembling it) most often appears in cases of atheroma of the aorta, whether leading to aneurysm or to valvular lesions with dilatation of the heart, and very rarely in cases of mitral disease.

Ætiology.—With regard to the remote causes of angina pectoris, as distinguished from the exciting causes of the paroxysms, there is little to add to what has already been incidentally mentioned. There has been some discussion whether the disease is related to gout; probably if such a connection exists, it is indirect, and through the medium of arteritis deformans of the base of the aorta, which is of frequent occurrence in gouty subjects. It is, however, a remarkable circumstance, which was first pointed out by Sir Gilbert Blane, that angina is much more frequent among well-to-do persons, than among the poor. Dr Gairdner throws some doubt upon the facts given,

but there can be little question of the general experience on this point; Dr Walshe says that his experience scarcely supplies him with more than a solitary well-defined example of the affection in hospital practice.

It must be remembered that many physicians have large hospital practice and see few rich patients, while with others the reverse is the case; that the fatal diseases of public men excite interest and are fully recorded; and that the attacks of angina are so sudden and pass off so quickly that a patient subject to them would scarcely apply for admission to a hospital. Moreover, the first seizure is sometimes fatal. Hence almost the only cases properly recorded among the poor would be either out-patients happening to be attacked on their weekly visit, or in-patients admitted to hospital with organic disease of the heart or aorta.

However it may be accounted for, the fact remains that, as Sydenham remarked of gout, and as we found it to be true of megrim, an unusual number of the cases of fatal angina pectoris—always a rare disease—which have been recorded since Heberden's paper appeared, have occurred in men who were remarkable for their ability. Dr Gairdner has set forth the argument in favour of the philosopher Seneca having been subject to angina. This was the opinion of Sir John Forbes; but the absence of severe pain and the fact that Seneca died a voluntary and gradual death by venæsection surely preclude the inference. Rather do the "history of the case" and the patient's own account of his attacks point to chronic bronchitis or possibly phthisis, ending in emphysema with spasmodic asthma; and this was the judgment of Parry and of Stokes. John Hunter undoubtedly died of angina pectoris, and so did Dr Arnold. Dr Chalmers's death during sleep had no likeness to angina. But it was doubtless the cause of the last illness and death of a still more eloquent preacher, Robert Hall. A patient who described the distress of an attack as "a sense of dissolution, not the fear of it," was "one of the most gifted men" his physician, Dr Reynolds, "ever knew, and one most competent to analyse sensations." Excluding on the one hand angina-like attacks of pain and dyspnœa in cases of aneurysm or disease of the aortic valves, and on the other the exaggerated descriptions of neurotic and hysterical patients, the editor has never seen classical angina pectoris in hospital practice. The only one he has met with was in a man between fifty and sixty, short and stout, endowed with great energy and ability in business. He had no discoverable disease of the heart or aorta, the lungs or the kidneys, and after being less than a year subject to attacks of increasing severity, all characterised by cardiac pain shooting into the arms, by a sense of imminent death, and by complete immunity in the intervals, he died in one more severe than any that preceded it.

"Males are most liable to this disease, especially such as have passed their fiftieth year" (Heberden). Dr Walshe quotes Sir John Forbes's collection of 88 cases: of these 80 occurred in men and only 8 in women, while out of 84 patients 72 were more than fifty years old.

In some cases (as Arnold's) a tendency to angina pectoris has appeared to be transmitted by inheritance. Dr Gairdner remarks upon its greater frequency in persons who are corpulent and of sedentary habits. It is, however, to be observed that the liability to attacks of angina interferes greatly with the activity of those who had before been accustomed to take exercise.

Beau described eight cases of angine de poitrine due to tobacco-smoking; similar ones have been recorded by other French physicians, and one by Eulenburg. Mostly they are not fatal, and belong to Walshe's pseudo-angina; but in one recent case the patient died ('Brit. Med. Journ.,' August, 1887).

A point worthy of notice is that persons suffering under frequent attacks of angina pectoris often have a haggard, frightened aspect; this is in many cases due to their sleep being broken by bad dreams; some patients are actually afraid to lie down in bed for fear of the occurrence of a seizure.

Prognosis.—It is important to remember that the cardiac affections most frequently associated with angina pectoris are such as it is at present impossible to diagnose with certainty. Consequently, one must never be led to give a favourable prognosis by the fact that on examination of the chest one has failed to detect evidence of organic disease. Dr Walshe, however, states that in every one of twenty-four cases which had been examined by him during life he was able to make out physical signs of some morbid change either in the heart, or in the aorta, or in both. And Dr Balfour says that he has never met with an instance of angina in which signs of dilatation of the heart were not present. Dr Latham's experience, again, is very similar; among thirteen cases there were only three in which neither increased dulness on percussion nor any murmur on auscultation over the heart or the aorta was present; and even in those three cases the cardiac impulse was most feeble, while the sounds, "though natural in kind," were "raised to their highest intonation and diffused over the entire front of the chest." In the next chapter, however, we shall have to consider how far signs of dilatation and of other changes in the muscular walls of the heart can be relied on.

But besides being unable to assert, in any case of angina pectoris occurring at a time of life when organic changes in the cardiac muscle are apt to take place, that the disease is free from danger to life, one is also never justified in giving an opinion as to its probable duration. A patient's first seizure may have been mild, yet after a longer or shorter interval it may be followed by another of such severity as to prove fatal. Or whereas the first may have been very severe, the subsequent ones may be far less so.

Treatment.—In the treatment of angina pectoris a great deal more can be done than formerly. The older physicians could recommend nothing better than large doses of laudanum and brandy. But we now, knowing how slowly absorption from the stomach takes place, always prefer to give *morphia* subcutaneously. Dr Balfour seems to have shown that it is perfectly safe to use *chloroform* freely, so as completely to narcotise the patient. When the pain is very severe, this is the only method by which it can be relieved; in protracted paroxysms, he follows it at once by subcutaneous injection of morphia, so that the chloroform sleep may pass into the morphia sleep, from which the patient awakes after some hours, free from suffering but exhausted, and generally with some œdema of the lungs.

As a rule, however, one should rather have recourse to the *nitrite of amyl* or to nitro-glycerine. The advantage of the former agent is the rapidity of its action. The best way is to employ the glass capsules, each of which contains from three to five minims of it. One of them is broken within the folds of a handkerchief, and the vapour is inhaled as freely as possible. In from fifteen to twenty seconds the face flushes, a sense of fulness in the head is experienced, the pulse at the wrist loses its tension, and the pain ceases. Dr Balfour has found in two cases that nitrite of amyl kept in a stoppered bottle gradually loses much of its efficacy in cutting short the paroxysms of angina, notwithstanding that it still flushes the face. Clinical experience does not appear to confirm Anstie's apprehension that this remedy might possibly induce cerebral hæmorrhage if the arteries of the brain should be diseased.

Since the year 1877, *nitro-glycerine* has been largely used in the treat-

ment of angina pectoris; it was first tried by Dr Murrell. Although its physiological action is less rapid than that of nitrite of amyl it is yet commonly quick enough to cut short an attack if taken at the very commencement. But for this purpose it must be employed either in solution or in the form of Martindale's chocolate tablets. Pills, unless broken up by mastication, are too slow in their effects. The proportion most commonly used is an alcoholic solution of the strength of 1 per cent. It may be given in a drachm of water, when it is almost tasteless; or if there be flatulence, in peppermint water with a little chloric ether. The dose necessary to give relief varies very widely in different patients; sometimes several doses have to be taken in succession at short intervals before the pain can be entirely got rid of. In beginning the treatment, it is generally best to prescribe at first half a minim or one minim of the solution; in one case Dr Murrell pushed the dose until 110 minims (more than a minim of pure nitro-glycerine) were taken at a time; but most patients are liable to experience alarming effects from fifteen to twenty minims. It gives rise to headache, a rushing noise in the ears, a sensation of fulness in the neck, and sometimes to nausea, languor, and drowsiness or even complete insensibility. These symptoms generally become more marked as the dose is increased; but with time a certain amount of tolerance of the remedy is often established.

The great use of nitro-glycerine, however, is in preventing the recurrence of seizures. In many case it does this perfectly, the patient being able after a time to leave off taking it, and remaining apparently quite well and able to walk long distances and even uphill without discomfort.* The plan which Dr Murrell advises is to give a dose of the remedy every three hours regularly, besides additional doses at each repetition of a paroxysm. The author has met with two cases in which persons thus freed from their symptoms ultimately died suddenly; and anyone who has suffered from the disease in a well-marked form should always afterwards lead the most quiet life that he possibly can, avoiding all kinds of exertion and excitement. One of the two patients just mentioned had returned to his business on the Stock Exchange. Rest must, indeed, be regarded as an essential part of the treatment of angina pectoris. Probably all the general measures found useful in the management of cases of thoracic aneurysm and severe cardiac disease find their application here also.

Before the introduction of nitro-glycerine, arsenic was the medicine which proved most efficacious in warding off the seizures. In a good many cases it yielded very satisfactory results.

It is right to add that Dr Balfour says of nitro-glycerine that he has "used it without any benefit in the treatment of angina."

* As an instance of the successful administration of nitro-glycerine in what Dr. Walshe would doubtless have regarded as "pseudo-angina," I may relate the following case, which occurred to me in 1882. A bank clerk, about twenty-one years of age, had for some weeks been suffering from what he described as a "sudden tightness of the chest," which would stop him in walking, so that he could not walk more than thirty or forty yards. He experienced a pain at the lower end and a little to the right of the sternum as well as behind near the spine at about the same level. He also noticed that he could not lie on the right side in bed without discomfort. His own impression was that the seat of the affection was in the lungs rather than in the heart. I found that he had a pulse of 120, but this was in part due to nervousness, for it soon afterwards fell to 104. No sign of any organic cardiac affection could be detected. I prescribed for him one minim of the solution of nitro-glycerine three times a day. In about a week he lost his complaint entirely, so that (as I afterwards learnt) he took only a single bottle of the medicine containing twelve doses.—C. H. F.

AFFECTIONS OF THE MUSCULAR WALLS OF THE HEART

DILATATION AND HYPERTROPHY.—*Physiology of the process—Its estimation—Its anatomy—Diagnosis—Symptoms—Ætiology—Treatment.*

FIBROID DISEASE.—*Anatomy—Causes and pathology—Cardiac aneurysm—Symptoms—Course and event.*

ACUTE MYOCARDITIS.—*Its origin, anatomy, and results.*

FATTY DEGENERATION.—*Fatty overgrowth and infiltration—Intrafibrous fatty degeneration: its histology, causes, and symptoms—Granular or pigmentary, and calcareous degenerations—Rupture of the heart.*

THE present chapter will be devoted to those organic affections of the heart which depend primarily on changes in its muscular substance.

PRIMARY ENLARGEMENT OF THE HEART.—HYPERTROPHY AND DILATATION.—We shall find hereafter that hypertrophy, generally attended with dilatation, of the heart is an almost inevitable result of all the more grave affections of the cardiac valves; and that another very frequent cause of the same changes is chronic Bright's disease. In each instance it is clear that the organ enlarges because the work which it has constantly to do is increased.

Theory of hypertrophy.—How increased activity produces hypertrophy is hard to say; the necessary condition for overgrowth is no doubt hyperæmia, but this probably begins as the result of increased waste of tissue from exertion The passive tissues hypertrophy either in direct consequence of hyperæmia, as the clubbed fingers of cardiac disease and venous congestion generally, or from some unknown stimulus as in the overgrowth of the bones seen in osteitis deformans, or of the spleen and lympharia in leuchæmia and Hodgkin's disease. Of the active organs, those of the nervous system, whether belonging to the central, the conducting, or the peripheral and receptive apparatus, appear to be incapable of true hypertrophy, however much exercised: the so-called cerebral hypertrophy (described at p. 647) is probably always a form of interstitial sclerosis. Glands hypertrophy from the stimulus of overuse, as the liver in beer drinkers, the kidneys in diabetes, or one kidney when the other has been destroyed by some local disease. The voluntary muscles hypertrophy remarkably—by increase of the fibres both in number and in size—in response to exercise; but the effect of training in removing interstitial fat is often sufficient to mask the true muscular development and make the firm and powerful muscles of an athlete less bulky than those of an indolent ree-liver.

Moreover, in this as in other physiological developments, the degree of

change is limited by the laws of each individual organism. However well nourished and well worked, the muscles will not hypertrophy beyond a certain point for each man, just as the possible degree of speed or flesh-forming is limited for each horse or ox, and the possible attainments of the senses or the intellect are limited for each human being.

Involuntary muscular fibre undergoes hypertrophy from overuse at least as readily as that which is set in action by the will: the ciliary muscle grows larger as the lens becomes less elastic, the bladder hypertrophies behind a stricture, the intestine above a chronic obstruction. In like manner the ventricle or the auricle hypertrophies as the result of narrowing of its orifice of egress, by which the discharge of its contents is obstructed. In the left auricle and left ventricle this is frequently seen; on the right side of the heart, stenosis of the pulmonary and still more of the tricuspid orifice is rare; but when present, the same effects are produced, except that for some unknown reason hypertrophy of the right ventricle produces more hardening and closeness of texture, with less increase of bulk, than that of the left.

The obstruction need not be from narrowing of the ostia through which each cavity discharges its contents: it may depend on mechanical hindrance to the flow of blood further on. Mere dilatation of the arch of the aorta can have no effect of this kind, but this dilatation is always associated with loss of elasticity; and, directly or indirectly, rigid arteries lead to hypertrophy of the left ventricle, probably by increased tension during systole. Increased friction from roughness of the tunica arteriosa intima, contraction of the arterioles, obstruction in the capillaries from external pressure, retardation of the flow in the veins from deficient muscular exercise or deficient respiratory movements—all these conditions increase intraventricular blood pressure and thus stimulate the muscular walls to hypertrophy. In the same way obstruction in the pulmonary capillaries produces hypertrophy of the right ventricle.

Increased stimulus, however, does not always call forth adequate hypertrophy. Impaired nutrition of the muscular fibres, from want of food, from imperfect digestion and absorption, or from loss of blood, may prevent them responding as they should; just as labour and gymnastic exercises, if undertaken by the ill fed, the anæmic, or the invalid, or if disproportioned to the powers of childhood or old age, will not cause hypertrophy of voluntary muscles, but rather atrophy and increased weakness.

Moreover, increased fluid pressure will always tend to dilate the walls of the containing cavity, even when its elastic as well as its contractile force is increased by hypertrophy of its muscular coats. Accordingly, some degree of dilatation is rarely absent, even when compensatory hypertrophy is well developed. This is seen in the dilated stomach which follows pyloric stenosis, the dilated œsophagus which forms a pouch above a stricture, the dilated intestine above a chronic obstruction, and the dilated renal pelvis, ureter and bladder, which result from stricture of the urethra.

In the heart, dilatation may occur with but little hypertrophy, particularly in the auricles, from mitral or tricuspid regurgitation; and hypertrophy may occur with little or no dilatation, as in uncomplicated cases of chronic Bright's disease; but as a rule the two conditions are met with together, both at the bedside and in the deadhouse. Nevertheless we can, in the majority of cases, recognise as the leading process (better perhaps by studying the physiology of the circulation during life than the cardiac

anatomy after death) either the compensatory and favourable one of hypertrophy, or the ingravescent and unfavourable one of dilatation.

We shall see hereafter that the most difficult cases of cardiac hypertrophy to explain are those of chronic Bright's disease, but there is little doubt that the immediate cause of the change in the ventricle is increased blood pressure from obstruction in the systemic circulation.

There is one form of obstruction which is less obvious because it is scarcely pathological. It is that caused by over-exertion in what often appears to be healthful labour or athletic sports. Each contraction of a muscle, if extreme, and particularly if long sustained, causes pressure on the arterial trunks which pass through or beside it.* This leads to increased blood pressure in the aorta and left ventricle, and becomes a stimulus to cardiac hypertrophy and a cause of dilatation of the heart and aorta, as well as of irritative arteritis of the latter.

Bodily exertion, even if it is repeated day after day, has necessarily its intervening periods of rest; and the healthy heart naturally possesses a considerable amount of reserve force beyond that which is required to carry on the circulation in ordinary circumstances. Consequently, although the strain upon it is augmented by muscular efforts of all kinds, it is generally able to meet the call without suffering damage. Even if its walls should to some extent yield to the increased pressure which they have to bear, they generally recover themselves afterwards, when the body returns to a state of rest, and the blood current resumes its usual tranquil course. But such is not always the case. It sometimes happens that violent or frequently repeated bodily exertion leads to permanent enlargement of the heart. We must suppose that one factor concerned in bringing about this result is a deficiency of power in the muscular fibres. Either they are originally weak, or their strength may have been lowered by previous disease or by an insufficient supply of food. Again, it is probable that there are cases in which a like enfeeblement of its walls renders the organ unable to maintain even the natural tranquil circulation. This feebleness is not due to fatty degeneration, or other obvious organic changes in the fibres, but to a state of the heart like that which in the voluntary muscles is recognised as weakness, leading to fatigue on moderate or even slight exertion. It seems likely that the immediate effect of the failure of the heart to perform its work efficiently is a yielding or *dilatation* of its walls, and that *hypertrophy*, or the increased growth of their substance, sets in afterwards. But, as a rule, it is not possible to trace this clinically.

Definitions.—As regards the application of terms, which has sometimes been somewhat confusing, it seems reasonable that "simple hypertrophy" should mean an increase in the amount of the heart's muscle, its cavities remaining unaltered in capacity; "simple dilatation," an increase in their capacity, the amount of muscle remaining stationary. The former is frequently observed, especially as a result of Bright's disease. And although the latter is perhaps never actually seen in the *post-mortem* room, it must yet be supposed to occur at the commencement of very many cases of heart affection. These definitions involve the necessity of admitting that a dilated heart is hypertrophied if its weight as a whole is above the normal standard, even though every part of its walls may be far below the natural thickness; and it was probably in order to avoid this apparent inconsistency that Dr

* The smaller arterioles which supply the muscle itself are probably protected by their physiological dilatation at the moment of stimulus of the motor nerve.

Walshe has defined as "simple dilatation" a condition in which "the walls are of such thickness as would be normal had the capacity been unchanged." But such a conception really rests upon an arbitrary basis, because, for a dilated heart, the normal thickness of walls should be proportionately augmented, in order to maintain its functional power; below this it is only a question of varying degrees of failure of "compensation." In fact although simple hypertrophy is in its physiological effects the very opposite of simple dilatation, yet in all the mixed forms of these affections it is to the latter rather than to the former that the case really approximates, however much increased the bulk may be.

Measurements.—In the *post-mortem* room, then, in order to ascertain whether or not a heart is hypertrophied one has only to weigh it after being emptied of its contents. According to Dr Peacock ('Reynolds' System,' vol. iv) the ordinary range of the weight of the organ is, in males affected with acute non-cardiac diseases, from nine to eleven ounces, with chronic non-cardiac diseases from eight to ten ounces; in females affected with acute disease from eight to ten ounces, with chronic disease from seven to nine ounces. But in large and powerful men who have been killed by accident or have died after a short illness, the weight may sometimes be as much as twelve ounces, or even more, without exceeding the limit of health.

It is much less easy after death to determine the presence of dilatation, at least in its slighter degrees; when it is well marked there can of course be no doubt about it. Dr Peacock gives the following figures as representing the normal dimensions of the two principal chambers:

	Males.			Females.		
	In Lines.	In Millimetres.	In Inches.	In Lines.	In Millimetres.	In Inches.
Circumference of heart	103·7	233·32	9·209	104	234	9·236
Girth of right ventricle	55·4	123·85	4·919	58·4	131·4	5·184
„ left „	48·3	108·67	4·289	45·6	102·6	4·049
Length of cavity of right ventricle . .	43·3	96·42	3·821	44·3	99·67	3·925
„ „ left „ . .	37·6	84·6	3·333	37·1	83·47	3·197

But it is very difficult to make sure how far the apparent size of the organ is modified by the state of contraction or of relaxation of the muscular fibres. The difficulty arises in its extreme form in regard to what has been described as "concentric hypertrophy," a condition in which it is supposed that the wall of the ventricle grows in thickness inwards, so that its cavity actually becomes smaller than natural. But although some recent German writers are still disposed to admit that concentric hypertrophy occurs in some rare cases, it is probably a condition to be entirely removed from pathological anatomy. In the *post-mortem* room hearts may at first look as though they might be specimens of such a kind, but they always yield to a little stretching with the fingers and regain their normal size. Even when a hypertrophied heart seems to have moderately large chambers it still remains a question whether in a fully relaxed state of their walls they might not be larger; in other words, the pathological proof of the presence of hypertrophy without dilatation is less simple than might be supposed.

It is only when the capacity of the cardiac chambers is not above the normal that one is justified in taking the thickness of their walls as proof of the presence or absence of hypertrophy. Dr Peacock gave the following as the normal measurements (1854). The figures in the third columns are those given by Bizot (1838).

Thickness of the	Males.			Females.		
	In Lines.	In Millimetres	Mm.	In Lines.	In Millimetres.	Mm.
Walls of the right ventricle: base .	[illegible]	4·16	4	1·85	4·16	3
" " " midpoint	1·98	4·35	3	2	4·5	2
" " " apex .	1·42	3·19	2	1·3	2·92	2
" left " base .	5·15	11·58	10	4·9	11·02	9
" " " midpoint	6	13·15	11	5·6	12·6	10
" " " apex .	2·4	5·4	8	2·5	5·62	7
Septum between the ventricles . .	5·73	12·89	11	4·7	10·57	9

But even when dilatation is present, the determination of the thickness of the several chambers is still of great importance, as enabling one to form some estimate of the degree to which the heart was competent to carry on its functions. In different cases there are very wide differences. One left ventricle measuring five or six inches in length may have its walls only two or three lines thick; another not more capacious may have them upwards of an inch thick.

As a rule, the weight of a heart, dilated and hypertrophied independently of valvular lesions and of Bright's disease, does not exceed fifteen or twenty ounces. But in one case observed at Guy's Hospital, it reached thirty-three ounces. Dr Peacock ('Reynolds' System,' vol. iv) speaks of hearts hypertrophied without any material valvular lesion or obvious source of obstruction in the aorta, and reaching the weight of from twenty-six to forty ounces. But it is not quite clear whether the presence of chronic renal disease was also excluded in these cases; in one of them, to which he refers as having been exhibited by Dr Bristowe at the Pathological Society in 1853, it is possible that the state of the kidneys was really the cause of the cardiac affection, although it is right to add that this view was rejected after due consideration by Dr Bristowe himself.

In the cases which are now mainly under consideration, we have no means of determining the rapidity with which the heart undergoes hypertrophy; probably the process is very slow and gradual. But when it arises secondarily to other lesions, of which the starting-point can be fixed, it has sometimes appeared to be much more rapid than could have been anticipated. Dr Stone ('Lancet,' 1879) has related two examples of injury to the aortic valves by blows upon the chest, in each of which, if the heart was healthy at the time of the accident, it must have gained weight at the rate of nearly an ounce a week during the four or five months that elapsed before the patient's death. And Dr Goodhart ('Path. Trans.,' vol. xxx) has published a case, the history of which would suggest that the organ within three or four weeks grew to a weight of nineteen ounces, as the result of an attack of pericarditis. Dr Stone remarks that the increase in size of the pregnant uterus is less rapid, namely, two thirds of an ounce a week; but

this assumes that the growth of the organ is uniform during the whole period of gestation, which is not likely to be the case.

There has been much discussion as to whether cardiac hypertrophy depends upon an overgrowth of existing fibres or upon a formation of new ones. Schroetter (in 'von Ziemssen's Handbuch') states that the fibres measure 0·03 mm. in a hypertrophied heart, as compared with a normal thickness of 0·007 mm. Friedreich also is said to have arrived at 0·025 mm. as the mean of ten measurements of the fibres of a hypertrophied left ventricle. But Rindfleisch says that he failed to discover any such difference, and his conclusion is that the fibres, being unlike those of all other muscles in forming a network, undergo a further splitting up, which leaves them apparently of the same size as before.

As already remarked, different cases seem to differ as to the order in which dilatation and hypertrophy are developed. In some instances it is probable that dilatation occurs first as the result of over-distension or of weakness of the cardiac muscle, and that hypertrophy follows later. In others hypertrophy appears to be the primary condition. In either event, that which finally brings about a failure of the heart's functional activity is commonly the supervention of a further degree of dilatation, which causes the hypertrophy to be relatively inadequate. It is usually said that such an ultimate breakdown of the organ depends upon the occurrence of fatty changes in its muscular tissue. But Cohnheim ('Vorlesungen,' i, p. 72) throws doubt upon this view. There has, he says, been much exaggeration in the statements that have been made as to the frequency of fatty degeneration in hypertrophied hearts; and even when it is present, he thinks it just as likely to be the effect of the disturbed compensation as its cause. His own view is that there is a simple "fatigue" or "exhaustion" of the fibres, unattended with any anatomical change that can be recognised with the microscope. Dr Allbutt ('St Geo. Hosp. Rep.,' vol. v) has drawn attention to a very interesting fact, which perhaps has a bearing on this question. It is that in the file cutters of Sheffield, who are constantly using the arm in rapid flexions, the biceps muscle undergoes great enlargement, but that after a few years it again wastes, and now falls far below its normal size. If the functional activity of hypertrophied muscles has thus a more or less definite period of duration, it is obviously important to relieve them, as far as may be, of all strains and extra calls upon them, so that the time when they are to break down may be postponed to the furthest possible limit.

Diagnosis.—The clinical recognition of hypertrophy and of dilatation of the heart rests partly upon percussion, partly upon inspection and palpation, partly upon auscultation.

By *percussion* one determines to what extent the heart comes into contact with the anterior wall of the chest, uncovered by the lungs. Consequently, the results of percussion are modified not only by the state of the heart, but also by that of the lungs. If the lungs are emphysematous, the area of dulness due to the heart may be diminished, notwithstanding that the organ is enlarged; if the lower and anterior part of the chest on the left side is flattened, or if the corresponding part of the lung is collapsed, the dull area may be increased, though the heart is no larger than natural. There is even a difference in the extent of cardiac dulness, according as the breath is drawn deeply in or forced out, so that for accurate percussion it is necessary that the breath should be held. A good way

of arriving at a satisfactory result is to make a series of marks with a soft pencil upon the patient's chest indicating the different points at which dulness begins to pass into resonance round the circumference of the organ One thus obtains a more or less triangular figure, representing the region within which an absolutely dull note is obtained. Above, it forms an angle, which in normal circumstances is situated at the upper border of the fourth left costal cartilage close to the sternum. From this point two lines are traced downwards, diverging in their course. One of them corresponds with the left border of the sternum, for although the right ventricle uncovered by lung lies behind the lower part of this line as far as the median line, the tone yielded by the bony tissue when percussed masks the dulness that should theoretically be present and prevents any accurate definition of the space occupied by the heart on this side.* The other line extends downwards and outwards, passing to the inner side of the nipple until it reaches a point at which the apex of the heart can be felt beating. Along this line the transition from dulness to resonance takes place gradually, so that above it one can trace another line running more or less parallel and about half an inch distant, which indicates the upper limit of a region of partial dulness, just as the lower one indicates the upper limit of absolute dulness. The upper line begins above at about the level of the third rib. To complete the triangular figure, a base line has to be drawn from the lower end of the sternum to the point at which the heart's apex beats. It can scarcely be traced by percussion, because the cardiac dulness passes insensibly into that caused by the left lobe of the liver. But Dr Gee says that sometimes a distinct heightening of pitch and increase of resistance can be made out in pasing from one organ to the other.

It must be added that, when the stomach is distended with gas, the resulting tympanitic sound is not infrequently transmitted by lateral conduction beyond the region which the stomach actually occupies. In fact, such a sound may often be elicited by tolerably firm concussion over the very spot beneath which the apex-beat can be felt. But in percussing over the heart, it is essential that the stroke should be light and free, and made from the wrist. The statements above made with regard to the natural extent of the cardiac dulness are applicable only when the patient is standing upright or lying upon his back. When he lies over to the left, the left side of the triangular area shifts further to the left, even though the heart may be of the natural size.

When the heart is enlarged, the upper angle of the area of cardiac dulness commonly remains at the fourth rib. Dr Walshe speaks of it as sometimes reaching even as high as the second rib; but such a change in an upward direction is unusual unless there is some morbid condition beyond hypertrophy and dilatation of the heart. On the other hand, the position of the two sides of the triangle varies widely from the normal. The right one may reach to the right border of the sternum or even half an inch or an inch further still to the right; this is an indication of increase in size of the right ventricle. The left line may sweep outwards as far as the nipple or even still further to the left, and it is carried much lower than usual, the apex-beat being situated at a lower level in the sixth interspace or even in the seventh; this is a sign that the left ventricle is

* Guttmann says that the bone may be rendered less vibratile if the hand be laid over the upper part of it, or if an assistant press firmly with the hands placed upon the rib cartilages on each side of it; in these circumstances a dull sound may be obtained.

enlarged. The two ventricles are generally affected together ; but if the right one alone is dilated, one effect of it is that the apex-beat is displaced outwards without being lowered. A pulsation may thus also be felt in the epigastrium. Dr Walshe states that distension of the right auricle contributes largely towards the increase of dulness to the right of the sternum at the upper part of the cardiac region. He says, too, that distension of the left auricle may cause abnormal deficiency of resonance in the third and second left intercostal spaces. And he even speaks of having recognised an impulse of presystolic rhythm in these several positions as the result of enlargement of one or the other of the auricular chambers. A necessary consequence of the displacement of the two lines marking the sides of the dull area when the heart is enlarged is that this area acquires a more or less definitely quadrilateral instead of the normal triangular form.

It is to be observed that for practical purposes nearly all that can be learnt from percussion of the heart, so far as concerns the diagnosis of enlargement of the left ventricle, may be gained by tracing one diagonal line from the fourth costal cartilage near the sternum to the site of the apex-beat, and another line at right angles to it from the lower end of the sternum upwards and outwards to the point at which the absolute cardiac dulness ceases. In normal circumstances the first diagonal line should not exceed two and a half inches in length ; when the heart is much enlarged it may extend to four or five inches. The second diagonal line should measure about one and a half inches ; it may be increased to more than two inches.

Now, it must not be supposed that percussion affords an infallible indication of enlargement of the heart, even when the lungs are healthy, being neither emphysematous nor retracted. On the contrary, very great hypertrophy of the left ventricle may be present (as for example in cases of Bright's disease) without there being any increase in the area of dulness. The organ seems to bury itself within the hollow of the left lung, so as not to come more widely into contact with the chest wall than in normal circumstances. One never has a better opportunity of demonstrating this fact than in cases of cerebral hæmorrhage ; in such cases one often finds at the autopsy an enormously hypertrophied heart, notwithstanding that a few hours previously it was impossible to detect any clinical evidence of such a condition.

If percussion fails to reveal the presence of enlargement of the heart, it is doubtful whether any other results can be obtained from *inspection*, or from *palpation*. It is possible that the character of the apex-beat may be altered, notwithstanding that it is in its natural position ; but great caution is required in diagnosing hypertrophy from this sign alone, at least while the action of the organ is disturbed by excitement.

When it has been determined by percussion that the heart is enlarged, the question arises whether the enlargement is due to dilatation or to hypertrophy ; and the solution of this question, so far as it can be solved, is to be found in a careful examination of the cardiac region by the eye and by the finger. In either case the heart's impulse may be seen and felt over a much more extended area than that of the natural "apex-beat." When there is great hypertrophy, it is often diffused, laboured and heaving, so that even a stethoscope with the observer's head resting upon it, is lifted as though by an irresistible power. In some cases, while the ribs immediately below the left nipple are pushed forwards, other parts of the chest wall are sucked in.

On the other hand, when dilatation preponderates, the impulse may consist (to use the words of Dr Walshe) "either of a short feeble slap, followed by a sudden fall back of the organ, or of a more prolonged faint tremulous motion." To the eye it has an undulatory character. Another peculiarity of the heart's action which indicates dilatation rather than hypertrophy is irregularity in the force of successive beats, or in their rhythm. Dr Walshe also remarks that there may be a want of perfect uniformity in the point at which the organ strikes the parietes. There has been some difference of opinion as to whether enlargement of the heart ever by itself causes an increased prominence or bulging of the præcordial region and a widening of the intercostal spaces, as compared with the state of the corresponding parts on the opposite side of the chest. Most observers admit that this is sometimes the case, especially in young subjects; but Schroetter, following Skoda, maintains that it occurs only when there is also pericarditis, by which the textures forming the chest wall are softened.

Little direct information as to the existence of hypertrophy or of dilatation is yielded by *auscultation;* its value lies rather in affording some guidance as to the state of nutrition of the cardiac muscle. But it is to be particularly noted that in cases of simple hypertrophy, with little or no dilatation, the first sound is not louder than usual but fainter, and is often dull and muffled; Dr Walshe describes it as "prolonged and weakened, sometimes almost to actual extinction, the sensation reaching the observer's ear being rather one of impulse than of sound." When dilatation and hypertrophy occur together, the first sound may be occasionally loud, higher-pitched than natural, and widely audible over the surface of the chest. But if with dilatation there is a soft, flabby state of the muscular tissue, the first sound is still weak, faint, toneless, and of high pitch. Alterations in the second sound are not infrequently to be observed, but they depend upon the state of the arterial tension, and afford only indirect indications of the condition of the heart itself.

The question whether a systolic murmur is ever due to dilatation of the ventricles, apart from regurgitation through the mitral or the tricuspid orifice, is a very difficult one. But it is at least certain that one or both of the cuspid valves may be rendered incompetent, either from failure of the papillary muscles to adjust their flaps properly, or as a mere result of the widening of orifices which necessarily takes place as the heart increases in size.

Seitz ('Deutsch. Arch.,' xi, xii) has drawn special attention to the fact that the movements of an enlarged heart within the pericardial sac are sometimes attended with sounds having a grazing or scraping quality, so as to be very like those produced by pericarditis. In one case observed by him, these continued up to the time of death, and no doubt was entertained as to the existence of pericardial inflammation; yet at the autopsy the serous membrane was found perfectly healthy, without any thickening or opacity of its surface. Dr Walshe, too, speaks of "knocking and rubbing additions to the first sound at the apex" as not very uncommon. One such instance occurred several years ago at Guy's Hospital which led to a diagnosis of pericarditis that proved to be absent on *post-mortem* examination.

Ætiology.—We have now to consider what are the special causes of dilatation and hypertrophy of the heart. In the first place, as stated at the beginning of this chapter, two of the most important causes are chronic Bright's disease

and lesions of the cardiac valves. In a great many cases it is impossible at the bedside to determine whether the state of the kidneys is such as to account for the cardiac enlargement, or whether there is a primary valvular lesion—generally affecting the mitral rather than any other valve—to which it may be due. Even in the *post-mortem* room the very same points often remain doubtful, or at any rate the facts are so far ambiguous that different pathologists interpret them differently. Formerly, in cases of heart disease with dropsy, the diagnosis of "mitral regurgitation" was deemed sufficiently accurate, and physicians frankly admitted that they were often unable to tell whether the left auriculo-ventricular orifice would after death be found narrowed or widely dilated. But now everyone recognises that in those instances in which it is dilated there is no lesion of the valve itself, but that the primary morbid process is dilatation of the left ventricle. Still it may be held—and until recently this was the common opinion—that regurgitation through the widened opening occurs secondarily, and constitutes the essential feature of the disease, being directly concerned in bringing about the induration of the lungs, the nutmeg change in the liver, the dropsy, and all the other obvious symptoms. Of late, however, the tendency has been to disregard to a great extent the presence or absence of mitral incompetency, and to look upon the impairment of the propulsive power of the ventricle as the really important thing in the cases in question. That is the view which the author long taught, and he even went further and entertained doubts as to whether secondary mitral regurgitation really occurs so constantly as is supposed by those who regard a systolic apex-murmur as a sufficient proof.

As regards the kidneys, we shall find, when Bright's disease is under discussion, that these organs may be of good size and not unhealthy in look, and yet may be shown by the microscope to have undergone changes so extensive as fully to account for any enlargement of the heart that may be present. The renal origin of such cases is by no means always, or even generally, indicated during life by albuminuria, a low density of urine, or the presence of casts, even when the autopsy leaves no doubt about the matter. And there are a great many other cases in which, after the most complete histological examination of the kidneys, different pathologists would express divergent opinions as to whether the cardiac affection should be looked upon as secondary to the renal, or the renal to the cardiac. Another possible cause of enlargement of the heart has been recently found by some observers in extensive pleural adhesions. But it is allowable to doubt whether the cases supposed to be of this kind which have been recorded by Bäumler and by Brüdi ('Deutsches Arch.,' xix) really warrant the conclusions drawn.

It must be remembered that the right side of the heart is not only subject to dilatation and hypertrophy from obstruction in the pulmonary circulation analogous to that in the systemic, but also from mere abolition of large capillary tracts, as in carnification of the lung by chronic pleural effusion.

Leaving now these intricate and doubtful questions, we have:

(1) A clear and unquestioned cause of primary enlargement of the heart in *over-exertion* of the organ. Within the last few years its importance has been made manifest in various directions. Da Costa in his observations on 'Irritable Heart' among soldiers of the United States army states that in twenty-eight out of a hundred cases there was evidence of hypertrophy.

In one of them death occurred from strangulated hernia eleven months after the commencement of cardiac symptoms; the left ventricle, though not apparently larger than natural, had its walls seven eighths of an inch thick at the thickest part. Fräntzel ('Virchow's Archiv,' 1873) drew attention to a like affection as having developed itself in nineteen soldiers engaged in the Franco-German war, especially among those who took part in the arduous march to Orleans, or in the attack upon Belfort. He also refers to some statements, according to which recruits in the German army frequently become affected with hypertrophy of the heart as the result of prolonged and heavy marches, especially in summer. Fräntzel is disposed to doubt the correctness of these observations, but our own Army Medical Department has been for many years familiar with the prevalence of the disease among British soldiers even in time of peace. In 1870 Dr Myers published an essay in which he showed that cardiac affections in general were considerably more common in soldiers than in sailors, and in the Foot Guards stationed chiefly in London than in the men of the Metropolitan Police. And in a large proportion of cases he says that there is neither valvular disease nor disease of the aorta, but an extreme excitability of the heart leading after a time to enlargement of the organ. The general opinion formerly was that the cause lay in the crossbelts, heavy accoutrements, and tight clothing which the men used to wear, and by the urgent advice of Dr Maclean and Dr Parkes the old form of knapsack was abolished, and a "valise equipment" was adopted in its stead. Dr Myers laid especial stress upon the effect of the tightness of the tunic collar in constricting the neck. It would be very interesting to know whether the prevalence of cardiac dilatation and hypertrophy among soldiers has been diminished within the last few years, but according to Prof. Veale ('Army Med. Dep.,' Report xxii) the necessary statistical data are wanting. He states, however, that this affection and the "palpitation" which is its most conspicuous symptom are still very frequent, and after careful inquiry into all the circumstances of a hundred cases, he assigns it in different instances to no fewer than seventeen more or less distinct causes. But it seems far more likely that some one cause is really responsible, and probably the real solution of the difficulty has been found by Surgeon F. A. Davy, who refers it mainly to the "setting-up drill," during which recruits are compelled to "swell the chest" so as artificially to expand it ('Army Med. Dep.,' Report xviii). To this they are subjected for four hours a day during a period of about six months, having to march, and even to "double," with the chest in an abnormal condition. Dr Davy shows that, in consequence of free expiration being prevented, the functions of the lungs and of the heart must be very seriously interfered with; and he appears to have ascertained by direct observation that soldiers under the drill, even when they are standing, have the frequency of the respirations increased to about 40, and the pulse to 110 in the minute, that the heart's rhythm is often disturbed, and that the impulse of the organ is altered in position, is more forcible, and is felt over a wider area than natural. It is surprising that Dr Veale does not allude to this view of the matter; but Dr Myers has often seen recruits completely exhausted after their morning's drill, which (one would suppose) ought not to have any such effect upon healthy young men. The notion is, of course, that the soldier's figure is improved, by the capacity of the chest being enlarged, but as Dr Davy remarks, this is of no advantage when obtained at the expense of its mobility.

Among civilians, the ill-effects of over-exertion of the heart, though they had been cursorily alluded to by many previous writers, seem to have been first fully recognised by Dr Peacock in reporting, in 1864, upon the health of the miners of Cornwall, who, besides heavy hammerwork in the day, have to climb ladders of immense height in order to get out of the pit every evening. He found that many of them suffered from cardiac dilatation. Then came an important paper in the 'St George's Hospital Reports,' vol. v, p. 23, by Dr Allbutt; but the cases that he observed hardly fall into the category with which we are now directly concerned, inasmuch as he assigned a conspicuous place in the sequence of events to chronic changes in the aorta and in the aortic valves leading at length to aortic regurgitation. More strictly in point is a series of articles by Seitz in the 'Deutsch. Archiv' for 1873 and 1874. He showed that at Zürich cases are tolerably frequent in which during life there is great uncertainty as to the exact diagnosis of the cardiac affection from which the patients undoubtedly suffer, and which after death are best explained on the view that the disease is a primary enlargement of the organ. The patients were almost all men, and engaged in heavy labour of one kind or another. But the most striking paper of all is perhaps one by Münzinger, in the 'Deutsches Arch.' for 1877, on what he terms "the Tübingen Heart." It appears that at Tübingen heart disease, without any valvular lesion, is very commonly seen both in men and in women who work as labourers in the neighbouring vineyards, situated often upon the slopes of hills, up which heavy burdens of manure have to be carried. A point on which great stress is laid is that these poor people are very badly fed, living on potatoes and puddings, and scarcely ever tasting meat. Dr Allbutt too, alludes to insufficiency of food as an important factor in the ætiology of cardiac affections due to overwork and strain; he cites two cases of Dr Paget's which appeared due to the habit of taking long and active exercise while fasting; and he expresses the opinion that one reason why young men of the upper and middle classes do not more often suffer ill-effects from athletic sports is that they habitually live well. Thus it would seem that one must take into account not only the absolute amount of exertion which a person may have undergone, but also the condition of the cardiac muscle at the time. Muscular work of all kinds of course calls upon the organ for increased efforts to maintain the needful circulation. In strong, vigorous subjects there is a reserve force which is equal to all but the most excessive demands upon it. But in weakly, ill-nourished persons, the heart may fail under comparatively slight efforts. And there probably may be great differences in the vital endowments of the cardiac muscle itself in different individuals, altogether apart from the state of nutrition of the body generally.

It is obvious that these remarks are applicable likewise to hypertrophy and dilatation occurring as the result of valvular lesions or of Bright's disease. In such cases the amount of enlargement of the heart required to effect compensation for a certain degree of leakage, or to overcome a certain degree of obstruction to the arterial current, must largely depend upon the previous state of the cardiac muscles.

It is not to be supposed, however, that any change in the muscular fibres recognisable by the microscope is an essential feature of cases of enlarged heart from overwork. Some few fibres may be found fatty or granular, but such an appearance is altogether exceptional.

(2) Among patients belonging to the middle classes, Traube ('Berl. klin.

Woch.,' 1872, p. 223) was inclined to refer cardiac hypertrophy to "excessive *smoking* and to congestion of the portal system, resulting from sedentary habits and excess of food." The probably injurious effect of tobacco has been taken into account by all the writers who have discussed the ætiology of cardiac affections in the British army, but there seems to be no clear proof of its taking a prominent place among their causes. As for indulgence in eating, it doubtless falls under the category of those conditions which produce hypertrophy by increasing the arterial tension. These will be discussed hereafter, when the relations between blood pressure and Bright's disease are under consideration.

(3) Since the recognition of the fact that *anæmia* induces a fatty change in the muscular fibres of the heart, it has naturally occurred to pathologists that those forms of the affection from which recovery takes place very possibly form the starting-points of subsequent cardiac disease. The question has been fully discussed by Dr Goodhart ('Lancet,' i, 1880); but although he has shown that in women who are actually suffering from chlorosis the heart's impulse is diffused and displaced outwards, and that when anæmia proves fatal this organ is found to be dilated, he has not brought forward any clinical proofs of the development of permanent mischief from this cause.

(4) Very much the same may be said of the supposed production of enlargement of the heart by the exanthemata and other *febrile diseases*. Pyrexia is known to damage the muscular tissues, and Dr Goodhart has recorded ('Guy's Hosp. Rep.,' xxiv) four or five instances in which sudden or nearly sudden death has occurred during scarlatinal dropsy and in which the heart has been found dilated or fatty. But in the cases in question it is difficult to say how much was due to increased arterial tension resulting from nephritis, and how much to the antecedent pyrexial state. And even if it be true that during enteric and other fevers the heart may become for the time dilated, it has yet to be shown that the organ is liable to remain in a morbid condition after convalescence. Professor Veale, in his paper above referred to, on the causes of palpitation and cardiac disease in soldiers, says that the most common of them all is fever, chiefly malarial. But when he declares, in support of this view, that the physician must have had "either small experience, or very limited powers of observation who cannot call to mind many instances of permanent weakening of the heart after fever," he appeals to a court of which the verdict would scarcely be in his favour.

(5) Another occasional cause of primary enlargement of the heart appears to be acute *rheumatism*. At least, now and then cases have occurred which seemed to admit of no other interpretation. The most striking of these was that of a girl aged eleven, who died in Guy's Hospital of cardiac dropsy six months after a rheumatic attack. The most conspicuous lesion was dilatation of the left ventricle, which had reached such an extent that although its walls measured only from one eighth to a quarter of an inch in thickness, the organ weighed ten ounces. There had probably been regurgitation through the mitral orifice, for the papillary muscles were much wasted, but the valve itself was healthy or only slightly thickened. How such an effect is produced by acute rheumatism, apart from pericarditis, is not certain. Dilatation of heart may, however, be caused by extension of pericardial inflammation to the subjacent muscle.

Symptoms.—The symptoms of primary enlargement of the heart vary widely in different cases. In the earliest stage of the affection they consist

partly in *palpitation* and subjective sensations of pain or discomfort in the cardiac region, partly in an increased *frequency of pulse*, which is often irregular or intermittent in rhythm. These phenomena have all been fully discussed in the last chapter. But another symptom, of which the absence is conspicuous in the merely functional diseases of the heart, is *dyspnœa*. This at first comes on during exertion only; the patient finds that he cannot walk so quickly as before with comfort, or that going uphill or ascending two or three flights of stairs makes him feel short of breath. From this condition there are all gradations up to a point at which even the slightest bodily movement becomes almost impossible. Inasmuch as in health muscular exertion makes the beats of the heart more frequent and calls for greater vigour of systole, there is no difficulty in understanding how it disturbs the action of the organ when diseased. And in cases of enlargement of the heart it has been specially noticed (as, for example, by Dr Veale among the soldiers whose cases he studied) that after even slight exertion or excitement the rate of the pulse becomes altogether disproportionately accelerated. But what is not so obvious is why this disturbance should give rise in the patient to a sensation of dyspnœa, even where the left ventricle is the seat of the affection, the right being healthy so far as can be ascertained. The explanation seems to be that in spite of its augmented frequency the heart after all fails to forward the blood through its left chambers with the needful rapidity; there is therefore an accumulation in the pulmonary vessels, and the right ventricle has to make increased efforts to propel its contents onwards. And as the result is a state of the pulmonary circulation identical with that which is produced in other cases by a deficient supply of air to the lungs, it is not surprising that the same feeling of shortness of breath is experienced. But the consequent increase in the frequency and depth of the respiratory movements would appear to be, in cases of disease limited to the left ventricle, rather prejudicial than useful to the patient, since it must still further augment the accumulation of blood in that chamber.

In many cases an early effect of cardiac dyspnœa is that the patient is unable to sleep with the head low. Instead of one pillow he has to use two or three. In extreme cases he cannot lie down at all, and is obliged to sit up in bed or to lean forwards. This condition is termed *orthopnœa*. It seems clear that the necessity for its adoption lies in the fact that it facilitates the descent of the diaphragm, which in the recumbent posture is hampered in its movements by the pressure of the abdominal viscera, and especially of the liver.

The assumption in the previous paragraph, that in cases of primary enlargement of the heart the left ventricle is the chamber earliest affected, is not altogether in accord with the view of some recent writers, many of whom are inclined to think that the right ventricle often undergoes dilatation, while the left still remains in a normal state. Thus Dr Allbutt relates, in the 'St George's Hospital Reports,' how on one occasion, towards the end of a long day's mountaineering in Switzerland he was rather suddenly seized with a strange and peculiar *besoin de respirer*, accompanied by a very distressing sense of distension and pulsation in the epigastrium; placing his hand over the heart, he felt a labonring diffused beat all over the epigastrium, and by percussion he ascertained that the right ventricle was very greatly dilated. He threw himself on the grass with his shoulders raised, and in a few minutes had the satisfaction of finding the distension,

the oppression, and the dulness recede. He could then run and even move about on the level, but the instant he began to ascend the symptoms returned, so that it was only with great caution that he could proceed. During the following night he was awakened again with severe palpitation and dyspnœa, which, however, passed off as soon as he went to the window and drew a few deep inspirations.

In 1866 the late Dr Daldy published a little work on 'Disease of the Right Side of the Heart,' in which, beside relating some similar cases to that of Dr Allbutt, he attributed to this cause a number of other symptoms. There is perhaps no theoretical objection to the view that the whole of the heart takes part in the excessive strain produced by violent bodily exertion, and that if the right heart happens to be the weaker of the two it may suffer before the left. But, granting the occurrence of such cases, one ought, if they do not invariably end in recovery, to find them continuing up to the time of death as uncomplicated examples of enlargement of the right chambers, the left remaining of normal size and thickness. We are familiar with the fact that dilatation of the left ventricle leads to a secondary dilatation of the right ventricle; but it does not seem possible that the order of events can be reversed. All pathologists, however, are agreed that if those cases in which enlargement of the right side of the heart caused by pulmonary emphysema or by severe bronchial affection be excluded, such an affection is scarcely ever seen in the *post-mortem* room unaccompanied by a like affection of the left side. The following is a very rare case of the kind. A man, aged forty-one, was admitted into Guy's Hospital in 1880 on account of dropsy of the abdomen and legs. On examination there was a loud systolic murmur at the ensiform cartilage, musical in quality at that spot, and propagated towards the right nipple as much as towards the left. On account of the rarity of primary disease of the right chambers this diagnosis was given doubtfully, but the autopsy left no question of the fact. The heart, which weighed 16½ oz., was extremely broad and rounded in shape; the right ventricle was large and massive, and had large fleshy columns, but the left one was quite small and flaccid; the right auricle formed the greater part of the base of the organ; the tricuspid orifice admitted more than five fingers; the edge of the valve was thick and opaque. Now, it had been a striking feature of this case that there was no orthopnœa; the patient, though very dropsical, lay quite low in his bed. At the time the easy state of the breathing was regarded as a further argument against the view that a cardiac affection of whatever kind was the cause of the ascites and of the anasarca; for it has hitherto been the universal opinion that dyspnœa must necessarily be produced by any lesion of the right side of the heart interfering with the blood supply to the pulmonary capillaries. But it admits of doubt whether this opinion is well founded, and the point is one well worthy of consideration in future cases.

Systematic writers upon diseases of the heart have been accustomed to draw a contrast, which does not appear to be well founded, between the effects of *dilatation* of the *left* side of the heart and those of *hypertrophy* of the same chamber. Dr Walshe, for instance, speaks of the pulse, in cases of pure hypertrophy, as full, tense, and resisting; he describes persons so affected as having a florid countenance and bright full eyes, and as liable to sensations of rushing of blood to the head, and to dull aching or throbbing cephalalgia. But these statements, however true of cases in which the cardiac affection is dependent upon an antecedent state of augmented

arterial tension, as in Bright's disease, appear to be altogether inapplicable to those in which the causes of hypertrophy of the left side of the heart are such as have been described in the present chapter. The obsevations of Da Costa, Myers, and Veale, all point to the conclusion that whether hypertrophy or dilatation be the result of overstrain of the heart, the effect on the circulation (if any is noticeable) is always that it is retarded, or at least that its activity is impaired. The organ, in fact becomes enlarged because in its natural state it is unequal to carry on its function; and the hypertrophy never passes beyond what is required for this purpose.

Prognosis and treatment.—The slighter degrees of enlargement of the heart subside when the cause of them is removed. But when hypertrophy has developed itself, it is only as the result of long and patient treatment that a cure can be looked for. One most essential thing is physiological rest to the organ. Among drugs the most useful appears to be *aconite.* Da Costa testifies most decidedly to its value in cases in which the cardiac impulse is unduly forcible; he generally gave one or two minims of a tincture three times as strong as the British tincture, three times a day; or gr. $\frac{1}{60}$th—$\frac{1}{30}$th of aconita. Dr Walshe speaks very highly of the same remedy; the dose which he recommends is gr. $\frac{1}{8}$th of the alcoholic extract of aconite. The usefulness of this medicine in cases of primary hypertrophy is not inconsistent with what has been stated above as to the symptoms of that affection, for when under the influence of rest and of good nourishing food the cardiac muscle recovers its tone and the dilated chambers resume their natural size, there is no longer any necessity for so forcible an action of the organ. In some cases *veratrum viride* may be employed with advantage; Da Costa administered drop doses of the fluid extract, or five to ten minims of the tincture, three times daily. *Bromide of potassium* is another remedy which is mentioned favourably by Dr Walshe. It does not seem that iodide of potassium is of any service.

If, however, rest be not taken in cases of overstrain of the heart, and if the early symptoms be neglected, the result is that sooner or later the hypertrophy fails in its purpose. Or perhaps there is from the first a condition of dilatation rather than of hypertrophy. In such circumstances *digitalis* is often of the greatest possible service.

As the ultimate issue, cardiac hypertrophy and dilatation tend to pass into a condition of "imperfect compensation" or "asystole," leading to a series of changes in the lungs and in the liver, and to dropsy of the dependent parts of the body, precisely like the effects of valvular disease, which we shall hereafter discuss.

Fibroid Disease of the Heart.—Unlike fatty degeneration and other diffused lesions, *fibroid disease* is in well-marked cases constantly limited to a definite area of the wall of the organ.

Dr Quain has indeed described in his Lumleian Lectures for 1872 ('Lancet,' 1872, vol. i) an affection which he terms "connective-tissue hypertrophy," in which he says that the muscular fibres are surrounded by connective tissue in all stages of its development—round-cells, spindle-cells, and bundles of fibrillæ.* The thickness of the heart's wall is in such cases increased, but the most striking peculiarity is its firm, tough, leathery feel. Dr Quain says that slight degrees of this change have been overlooked, and

* See on the histology of fibroid disease of the heart, a valuable paper by Dr F. C. Turner, in the 'Trans. Internat. Med. Congress,' 1881, p. 427.

he refers to a greatly enlarged heart, weighing forty and a half ounces, which had been for thirty years preserved as a specimen of cardiac hypertrophy in the museum of St George's Hospital, but in which upon examination the increased size was found to depend only in part upon muscular overgrowth, the connective tissue being also greatly in excess.

Such a primary diffused hypertrophic sclerosis of the heart is rare, although it frequently accompanies the consecutive hypertrophy of Bright's disease and of valvular lesions.* But we have had a large number of instances of circumscribed fibroid disease. No fewer than eleven such cases came under observation in the *post-mortem* room of Guy's Hospital in one period of less than twelve months (1873–74), and altogether twenty-six cases have occurred there at different times. In its slighter degrees it consists in the presence of streaks and patches of a milky white colour in the substance of the muscular tissue. The wall of the heart is thereby rendered more hard and resisting to the knife, but it may also acquire a succulent and spongy appearance, and when incised its cut surface looks uneven. Microscopically there is seen a perfectly developed connective tissue, forming wavy bands, running in the same direction as the muscular fibres, some of which, or the remains of them, are still embedded in it. Or there may be a dense fibrous plate, looking like a piece of tendon, and consisting of a glassy substance with regularly arranged fissures or spaces, indicating the planes of fibrillation. It creaks when cut, and sometimes it contains calcareous salts in such quantity as to make it crackle under pressure, like an egg-shell.

The seat of this lesion is sometimes the apex of the heart, sometimes some part of the anterior or posterior wall of the left ventricle, sometimes the septum. It seldom or never begins in the right ventricle, but it may invade it by extension from the left. The fibrous substance is occasionally surrounded on all sides by muscular tissue, touching neither the endocardium nor the pericardium; and it is then very likely to be overlooked, unless all parts of the organ are completely sliced up. In other cases it reaches one or both surfaces to a greater or less extent. The endocardium then shows a marked local thickening and opacity, while the visceral pericardium is found covered with lymph, or adherent to the parietal layer. Some observers have thought that fibroid disease of the heart is generally secondary to pericarditis or to endocarditis, which spreads to the muscular wall of the organ. But this view is clearly inapplicable to many cases; and so far as the endocardium is concerned, there is an insuperable difficulty in the fact that inflammation of this structure, apart from the valves, is itself unknown. As regards the relation between pericarditis and fibroid disease, it is to be noted that adhesion is often present only just where the fibroid material reaches the outer surface of the heart, and that even when the whole serous sac is obliterated, the two layers are often found to be very firmly connected together at that spot, but elsewhere so loosely that it is easy to separate them. Moreover, if extension from a general pericarditis occurred, one would expect to find connective tissue dipping into the muscle at a number of different points, which is not the case. And in one instance pericarditis developed itself so as to be recognised by physical signs about two months before death, at a time when the fibroid affection must have been already of long standing.

When the part of the left ventricle that is affected by fibroid disease

* It was present in two of Dr Turner's cases.

includes the base of either of the fleshy columns of the mitral valve, the process often spreads into that column, which becomes shrunken. This condition is distinct from the far commoner one in which the summits of the mitral columns undergo conversion into fibrous tissue by extension from the tendinous cords attached to them.

In all cases of fibroid disease of the heart there is considerable destruction of muscular tissue at the seat of the lesion; and when the whole substance of the wall is involved from one surface to the other, we have often found not a trace of the normal structure between. The thickness of the wall is, as a rule, much diminished. In one case only was the result to produce an obvious increase of bulk; in that instance the septum was the part affected.

Pathology.—With regard to the pathology of this morbid change there is still some uncertainty; perhaps it is not the same in all cases.

1. One view is that it results from a primary process of chronic inflammation, a *myocarditis*, arising spontaneously, or from rheumatism, or perhaps in consequence of a blow or fall upon the chest. At first, it is said, there is an infiltration of leucocytes; these subsequently develop into connective tissue. The atrophy and disappearance of the muscular fibres at the seat of the disease is regarded as a secondary effect of the compression which they undergo. The author has never been able to discover a small-celled infiltration, even at the margins of the fibroid patches; but perhaps the reason may have been that the morbid process was no longer advancing when the patient died. In four of twenty-seven cases taken from the *post-mortem* records of Guy's Hospital there was history of a former attack of acute rheumatism. Another probable cause of chronic myocarditis is alcoholic intemperance.

2. In some instances *syphilis* gives rise to an affection which in its later stage is probably not distinguishable by its characters from fibroid disease due to other causes. At an earlier period it would doubtless be characterised by the presence of gummata, as in eight cases cited by Lancereaux. It is worthy of notice that the development of gummata in the heart is by no means limited to the left ventricle; in two of the cases in question the right ventricle is said to have been alone affected, and in one the right auricle. Among our twenty-seven cases of fibroid disease of the heart, there were four in which, from the presence of specific lesions elsewhere, the existence of syphilis could be safely asserted. In only one of them were gummata detected; these consisted of a mass as large as a bean within the wall of the right ventricle, and of a number of small, hard yellow points embedded in a reddish gelatinous substance at the growing edge of the fibroid material in the left ventricle. In one instance, the morbid process was limited to the septum, this being changed through nearly its whole thickness into a tough fibrous material with puckering and depression of the adjacent part of the endocardium. The disappearance of characteristic gummata in the more advanced stage of the disease corresponds exactly to what is observed in the liver, in the testis, and in other organs.

3. The formation of *thrombi in the cardiac cavities* may give rise to an inflammatory change, extending through the wall of the part of the heart to which they adhere. A well-marked instance of this occurred in the auricular appendix. It is surely not improbable that the ultimate result might be the formation of a patch of fibroid disease; and such an origin might explain the frequency of fibroid disease at the apex of the left ventricle, for

this point is very apt to become the seat of thrombi during the course of enteric and other fevers.

4. In a series of eleven cases of fibroid disease of the heart brought by the author before the Pathological Society in 1874 there was one in which the cardiac muscle presented in addition a peculiar form of degeneration, consisting in its conversion into a dry-looking, greenish-brown substance, of the texture of wash-leather. At one part this formed a thin flat layer, embedded in the substance of the heart, and appearing as a narrow sinuous line in a vertical section. Under the microscope it was found to be merely muscular tissue, which retained its striation, and showed remarkably well the branching and reuniting of the fibres. Its characters contrasted strongly with those of the muscular fibres which lay within the area of the fibroid change; they exhibited but slight striation or had undergone fatty degeneration. In the case of another patient, aged sixty-two, with regard to whom the probable existence of fibroid disease of the heart was repeatedly discussed at the bedside, we found on *post-mortem* examination that the posterior wall of the left ventricle was much thinned, and was to a large extent converted into a lustreless yellowish-green substance, almost exactly like that just described; no fibroid material, however, was present. From these two cases, it appeared clear that the peculiar change in the muscular tissue must be the primary affection, and that the fibroid development must be secondary, and Dr Ormerod, twenty years ago, suggested ('Brit. Med. Journ.,' 1863) that the latter was due to a "process of conservative substitution, designed to fortify the walls of an attenuated and weak heart." But it is only quite recently that the true relation of the one morbid process to the other has been demonstrated to be as follows.

Weigert seems to have been the first to point out ('Virchow's Archiv,' vol. 79, 1880) that fibroid disease of the heart is often the result of a change analogous to the formation of *infarctus* in other viscera. This has since been confirmed by the observations of Hüter (*ibid.*, vol. 89, 1882). He found precisely the same dry greenish or yellowish-brown patches as those above described, and traced them to obstruction of branches of the coronary arteries, sometimes resulting from thrombosis, sometimes from embolism, as in one case of endocarditis affecting the mitral valve in a young subject. And he recorded no fewer than eighteen cases of fibroid disease of the heart in each of which the affection was associated with sclerotic changes in the coronary arteries, corresponding more or less closely in distribution with that of the fibroid patches. At the same time he does not think that the formation of actual infarctus is a necessary step in the process by which coronary arterio-sclerosis leads to fibroid disease. It is sufficient that there should be a molecular change in certain of the muscular fibres, from interruption of their blood supply. The fibroid patches themselves he regards as the result of an inflammatory process set up by the disintegrating tissue elements. The author drew attention in the 'Pathological Transactions' for 1874 to the relation between fibroid disease of the heart and *arteritis deformans* of the systemic arteries generally, and commented on the fact that the former lesion was present in a very remarkable case, accompanied with absence of pulse in the limbs, which had been recorded by Dr Wilks, and to which reference will again be made under diseases of the blood-vessels. In the second of the two cases in which were found the greenish-looking patches now known to be infarctus, the coronary arteries were extremely diseased, and some of their branches

were completely obliterated; but one could not make out that the two morbid changes were definitely related to one another.

Dr Wickham Legg, in his Bradshaw Lecture (1883), has thrown doubt upon the correctness of Hüter's view, and has expressed the opinion that the lesions found in the coronary arteries in his cases were only such as might have been anticipated from the advanced age of the patients. The importance of the peculiar change in the muscular tissue antecedent to the fibroid development seems not to have struck him. Our experience, however, at Guy's Hospital, quite confirms what he says as to the frequent absence of all obvious disease of the cardiac muscles notwithstanding that the coronary arteries are much obstructed. He cites a case of Dr Greenfield's, in which their orifices were indicated only by small vertical thickenings on the inner surface of the aorta; microscopically all that could be found was a slight but widely distributed fatty degeneration of many of the muscular fibres of the heart. In the year 1880 three instances occurred, in each of which arteritis deformans of the aorta had led to great narrowing of the mouths of both arteries and to almost complete obliteration of at least one of them. The cardiac muscles in these cases were either healthy or at most a little soft and pale.*

It might have been expected that the loss of contractile power of so much of the cardiac wall, inevitably resulting from the transformation of the muscular substance of the ventricle in its whole thickness into fibrous tissue would seriously impair the efficiency of the organ. But Cohnheim has found experimentally that in the rabbit a large part of either ventricle, or even the entire lower third of the heart may be held fast in a clamp so as to be completely deprived of its function without the arterial pressure becoming lower in consequence. And clinical experience shows that fibroid disease sometimes gives rise to no symptoms whatever; in three of the above-quoted twenty-seven cases in which it was discovered at the autopsy, the patient had died from some other cause. On the other hand, the frequency with which dilatation and hypertrophy are associated with this morbid process proves that in many instances the systole of the ventricle is more or less interfered with; in ten of our twenty-seven cases the heart was considerably enlarged, weighing from twenty-one to thirty-five ounces.

A peculiar alteration in the shape of the ventricular cavity is produced by the presence of fibroid disease in its wall; it becomes deepened from before backwards, so that the mitral valve lies much further from the anterior surface of the heart than usual. Occasionally the valve is also separated from the posterior surface by a considerable interval.

Cardiac aneurysm.—During the systole, any portion of the wall of the heart that has undergone the fibroid change must be exposed to great pressure; one can easily imagine it forming a protrusion like those that are observed in the frog's heart when the action of digitalis is beginning to manifest itself.† Indeed, when the whole thickness of the cardiac muscle is destroyed, a permanent yielding of the affected part almost always results. Sometimes it forms a shallow pouch, sometimes a sac of greater or less size, communicating with the ventricle by a comparatively narrow opening.

* It seems impossible to reconcile Hyrtl's and Cohnheim's statement, that the coronary arteries and their branches possess no anastomoses, with the results of the injections made by Dr Legg and by Dr Samuel West, who found that they could readily fill one artery from the other, the two communicating by branches over the surface and the apex of the heart.

† See a paper by the author and Dr Stevenson, in the 'Proc. Royal Soc.,' for 1866.

This condition is now generally known as "cardiac aneurysm." The term was used by Corvisart and earlier pathologists to denote general dilatation of the cavities of the heart. With this, as described in a previous section, we are not here concerned, nor with the destruction by ulceration of a part of the wall of the heart in connection with a like affection of the valves which is still sometimes spoken of as "acute aneurysm." Leaving these affections out of consideration, cardiac aneurysm, in the modern sense of the term, depends almost always upon a pre-existent fibroid change in the muscle. Dr Legg in his Bradshaw Lecture ('Med. Times. and Gaz.,' 1883, ii) has cited three cases which appear to show that it may be produced by fatty degeneration; but there is no doubt that these cases are quite exceptional. The reason doubtless is that in fatty hearts the morbid process is too widely diffused, and the ventricular systole too feeble, for great pressure to be thrown upon any one part of the chamber.

Aneurysm of the heart, like fibroid disease, is met with at all periods of adult life up to a very advanced age. Among Hüter's eighteen cases of fibroid disease, only four of which occurred in patients under sixty years of age, there were but four in which aneurysms were present; the ages were fifty-six, sixty-two, seventy-three, and eighty. Both affections are far more common in men than in women.

Cardiac aneurysms do not usually attain a great size, but may occasionally become as large as the fist. They then of course project from the surface of the heart; but the smaller ones, especially if there are several of them, are sometimes excavated within its substance. In a remarkable instance ('Path. Trans.,' 1874) the wall of the left ventricle was tunnelled out in all directions into cavities, of which the largest was as big as a walnut. Fibrin is often deposited in large quantities in the interior of the sac of an aneurysm of the heart; in the specimen just referred to many of the cavities were filled with an adherent greenish gelatinous substance, containing curd-like degenerating flakes. Dr Wilks has placed on record (*ibid.*, vol. viii) a case in which there was found attached to the apex of the heart a cured aneurysm of the size of a pigeon's egg, of which the walls were calcareous and the interior completely consolidated.*

Symptoms.—Clinically fibroid disease and aneurysm of the heart can very seldom, if ever, be diagnosed. The presence of the former affection should perhaps be suspected when cardiac symptoms without evidence of valvular lesions are present in a patient suffering from senile gangrene or showing other signs of general arterial disease. A syphilitic history, too, may lead one to infer that the heart has been the seat of gummata, out of which a fibrous tissue ultimately has developed, and it is important not to overlook the possible existence of syphilis as a cause of cardiac disease, on account of the good results that may be anticipated from a prolonged course of mercury and of iodide of potassium. One striking instance of this has come under the author's notice, and Dr Balfour may be cited in testimony that cases of "excited action of the heart with hypertrophy" have yielded to antisyphilitic treatment.

The physical signs of fibroid disease of the heart are undistinguishable from those of enlargement due to overgrowth of the muscle. In either case, a systolic apex murmur may or may not be audible; at one time it may be present and not at another.

* I well remember the autopsy, for it was the first at which I was present when I entered as a student of Guy's Hospital, in October, 1856.—C. H. F.

In a few instances the pulse has been unusually slow, varying from 28 to 48 in the minute.

When there is a large aneurysm projecting from the heart's apex, a careful mapping out of the area of cardiac dulness might possibly suggest the real nature of the case; but the disease would still have to be diagnosed from an aortic aneurysm pushing downwards into the region ordinarily occupied by the heart. In one instance Skoda is said to have observed bulging of an intercostal space overlying the seat of a cardiac aneurysm.

The *result* of fibroid disease of the heart is sometimes obstruction to the pulmonary and systemic circulation, and consequent dropsy; this was the case in nine among the above-cited twenty-seven cases. In one instance the period that elapsed from the beginning of the patient's illness up to the time of his death was remarkably short, only seven weeks. But other cases have run a protracted course, and have at first been benefited by treatment with digitalis and diuretics, for that which impedes the flow of blood is, after all, not the fibroid affection itself, but the failure of compensation on the part of the wall of the ventricle in general.

In a great many cases, however, the heart has gone on discharging its functions quite naturally, so far as can be known, until the patient has suddenly fallen down dead. For example, Dr Whipham ('Path. Trans.,' xxi) has recorded the case of a gentleman, aged twenty-nine, who fell dead from his horse while riding in Hyde Park, having started in good spirits and apparently perfectly well, and having never before exhibited any symptoms of cardiac disease. The abrupt stoppage of the organ in such circumstances is at present altogether unintelligible. But it is perhaps worthy of notice that there is an exact parallel for it in the results of the experimental ligature of one coronary artery, or even of a large branch of one coronary artery, in the dog, as practised by Cohnheim ('Virch. Arch.,' vol. lxxxv). After this operation the heart for a little while goes on beating with perfect regularity and maintains the arterial pressure at its normal level. But at the end of about ninety seconds its pulsations become somewhat less frequent and their rhythm is slightly disturbed; and about half a minute later both ventricles suddenly stop at the same instant, after which no stimulus whatever succeeds in restoring their contractions. Evidently, therefore, it is not sufficient, in searching for the cause of sudden death, to examine the orifices and main trunks of the coronary arteries. An arrest of the blood-current through any one of the principal branches must be supposed capable of accounting for it, and equally so whether there be embolism, or whether a more chronic change in the vessel has gradually led to the same result.

Acute Myocarditis.—Acute or subacute inflammation of the muscular tissue of the heart may take place under special conditions, but it is only positively recognised when it occurs in connection with endocarditis or pericarditis. In the latter case it is not at all unusual to see the layer of muscle immediately beneath the serous membrane involved in the inflammatory process; it is pale and soft, whilst the microscope shows it to have undergone a change in texture of a granular or fatty kind. In some exceptional cases, the whole thickness of the walls is seen to be affected; then dilatation is apt to occur, and may lead to speedy death or become a permanent morbid condition. At times long subsequent to the inflammatory attack, its effect may be seen in a fibroid patch, showing on section numerous white fibrous streaks interspersed with the muscular fasciculi (p. 935).

A more acute inflammation of the muscle, or rather one of a different kind, is met with in *pyæmia*, in which numerous small abscesses may be found scattered through the tissue of the organ. A single circumscribed abscess is sometimes met with in connection with endocarditis and disease of the valves. The diagnosis of myocarditis can only be conjectural.

Fatty Disease of the Heart.—(1) *Adipose overgrowth and infiltration.*—The natural layer of adipose tissue beneath the pericardium is often found increased in elderly persons, and within limits this may be regarded as a physiological condition which gives rise to no symptoms during life. It chiefly affects the right side of the heart, especially at the base of the ventricle.

But sometimes the fat grows in upon the muscular fibres so as to thin the cardiac wall, and this becomes a cause of atrophy of one or both ventricles. Occasionally the *adipose growth* may penetrate right through the wall until it meets the endocardium, and this is particularly seen near the apex of the right or the left ventricle.

Again, there may be *fatty infiltration*, the adipose tissue increasing between the fibres. A large amount of interstitial fat in the muscles of the limbs, as in cattle fatted for the market, and in men who drink largely of beer, is probably indicative of over-feeding and under-work. When it affects the diaphragm it may become more serious. But when the cardiac tissue is so infiltrated, as frequently occurs in conjunction with adipose overgrowth and sometimes alone, it is probable that, though no certain signs or symptoms of its presence arise, it may be the immediate cause of death. Obesity is notoriously a bad condition for recovery from surgical operations and other injuries, and a "weak heart" is often the cause of the want of repair and fatal issue.

(2) *Fatty degeneration.*—This is a different pathological condition, recognised by Laennec. The heart may be free from adipose tissue, but its tissue is pale, soft, and flabby. On the inner surface, particularly of the musculi papillares of the left ventricle, pale yellow zigzag markings are seen, described in the 'Med.-Chir.-Trans.,' vol. xxxiii, by Dr Quain as "tabby degeneration." Under the microscope the fibres are found to have lost their striæ, and black granules appear instead, at first in transverse lines, as if the change had affected disc after disc. Next the dark granules become larger and acquire a bright glistening centre, and all trace of structure disappears.

According to Dr Hermann Weber,* the amount of ethereal extractives from such hearts is not greater than normal. Dr Stevenson, however, as quoted by Wilks and Moxon, found the fatty matter nearly doubled, and Krylow has apparently settled that there is a decided relative increase, though much less than one would have anticipated.

The most frequent *causes* of this fatty degeneration of the heart (which is quite independent of obesity either of the heart or other parts) are anæmia, and certain poisons. It is most constant and well-marked in those remarkable cases of idiopathic anæmia first described by Addison, and since known under the name of "anémie grave" and "perniciöse Anämie." It is also often found in cases of leuchæmia and Hodgkin's disease, and occa-

* "Zur Lehre von der fettigen Entartung des Herzens," 'Virchow's Archiv,' xii, 326 (1857). Dr Weber has informed the editor that subsequent extension of his inquiries confirmed his previous conclusion. The wasting of the normal adipose tissue and the small extent of the degenerative process, often limited to the left ventricle, may explain these results. Böttcher, however, and Valentiner support the same conclusion as Stevenson.

sionally in phthisis, cancer, and other wasting disorders. It is a constant appearance in fatal cases of poisoning by phosphorus, when the liver is also the seat of remarkable fatty degeneration; and the same thing has been observed in poisoning by arsenic, by mercury, and by lead. It has sometimes but not constantly been observed when the coronary arteries have been much diseased. Lastly, acute fatty degeneration often occurs as a superficial change immediately beneath an inflamed pericardium.

The *symptoms* of true fatty degeneration are very obscure and doubtful. It is usually surmised from our knowledge of pathology rather than diagnosed by physical signs.

The cardiac impulse is described as being weak but irritable, or sometimes "slapping," *i. e.* distinct but short. The first sound is often accentuated and has lost its booming character, so as to resemble the second. The radial pulse may be quite unaffected. General symptoms of lividity, dyspnœa, irregular pulse, &c., probably only appear when there is concomitant dilatation.

When a person past fifty, pale and thin, with a white, soft, "satiny" skin and early arcus senilis, suffers from dyspnœa, and his heart gives a short, sharp first sound, the presence of fatty degeneration of the heart is probable; and it becomes almost certain if grave anæmia is also present.

The *result* of fatty degeneration is undoubtedly in not a few cases sudden and fatal syncope. Often, however, it is found after death has occurred in other ways. It does not appear in itself to lead to dilatation, to which it is rather secondary; but it certainly may end in rupture of the heart.

A brown or *granular degeneration* of the cardiac muscles has been frequently observed, but its relations to fatty degeneration and its pathological significance are still obscure. Spongy or cavernous degeneration has been observed in the fœtal myocardium by Virchow.

The rarest form of cardiac degeneration is that which has been described by Köster as *calcareous infiltration*. A good account of it, with two original specimens figured, is given by Dr Coats in his 'Manual of Pathology.'

Rupture of the Heart.—This rare and interesting pathological condition owes its practical interest to the importance of discriminating it from rupture as the result of injury. In the latter case the lesion is almost always in the right ventricle or one of the auricular appendages. In idiopathic rupture it is almost always in the left ventricle. Of fifty-five cases, forty-three affected the left ventricle and seven the right; the auricles are still more rarely found ruptured. Traumatic rupture of the heart is rather more common in the right ventricle than in the left. In most cases the muscle is already weakened by fibroid, fatty, or granular degeneration; but several instances are on record in which no such changes have been detected.

The age of the patient is generally over sixty. George the Second died of rupture of the heart at the age of seventy-six: it is remarkable that he was the first English sovereign who exceeded the age of seventy.

The rent does not always go through the entire thickness of the wall, and may possibly in such cases be recovered from; but usually hæmorrhage into the pericardium ensues from the torn vessels, and causes death as certainly, though not so rapidly, as when a larger rent opens directly into the ventricular cavity.

PERICARDITIS

Rarity of idiopathic pericarditis—Its antecedents—Anatomy—Fibrinous, serous, hæmorrhagic, and purulent effusion—Physical signs—Symptoms—Event—Adherent pericardium—Hydropericardium—Treatment of pericarditis.

Ætiology.—Inflammation of the pericardium is very rarely idiopathic. Professor Bäumler, in the fifth volume of the Clinical Society's 'Reports,' has published three cases of idiopathic pericarditis in adults, and the editor once saw an uncomplicated idiopathic case with Dr Dalton, of Norwood, in a healthy man about forty. Sometimes acute pericarditis has followed prolonged bodily exertion—for example, after a long march ; or it has apparently been set up by exposure to cold, generally along with pleuro-pneumonia. Scarcely any acute disease is more rapidly fatal than double pleuro-pneumonia with pericarditis, but in these cases the pulmonary symptoms override those of the pericardial inflammation.

Pericarditis is sometimes the first manifestation of acute rheumatism, pain and swelling of the joints coming on only when it has existed for two or three days. Hence, when a patient dies of pericarditis after a very short illness, one can hardly exclude the possibility of a rheumatic origin of the attack. But this explanation applies especially to children, in whom such a rheumatic pericarditis preceding any affection of the joints is much more common than in grown-up persons.

If we exclude those cases in which inflammation of the pericardium is set up by wounds or direct mechanical injuries, it can, in the great majority of cases, be traced to one of two causes—either (1) to some general morbid condition, or (2) to a pre-existing local disease of a neighbouring part.

(1) Next to acute rheumatism, Bright's disease of the kidney is by far the most common cause of pericarditis.

In Russia pericarditis has been often observed to occur in scurvy, but this has not been confirmed by English physicians. Gout has also been mentioned as a cause, but this has probably acted only indirectly through the renal disease which so often complicates it. Tubercular deposits may set up pericarditis, but this is not commonly observed at the bedside, and occurs chiefly when several serous membranes together are attacked by tubercle, independently of the organs covered by them. Formerly pyæmia was said frequently to give rise to this and to other serous inflammations, but it is now believed that pericarditis occurs in pyæmic cases only when suppuration has first attacked the heart's substance; the affection would thus come under our second head.

(2) Pleuro-pneumonia (of the left side especially) is often associated with pericarditis, but generally inflammation attacks both organs simultaneously, and cannot be strictly said to pass from one to the other. Mediastinal tumour or abscess, disease of the ribs, or even of the mammary gland,

are other causes of pericarditis, and it not uncommonly arises intercurrently in cases of cardiac dropsy towards the end of life. Usually, however, in these cases the kidneys will be found diseased as well as the heart.

Anatomy.—The morbid appearances characteristic of pericarditis are in part the same as in other serous inflammations. In an early stage the membrane becomes minutely injected and loses its lustre. Then lymph appears upon its surface, often first around the roots of the great vessels. As this lymph increases in amount, however, it assumes appearances which differ from those ordinarily seen in pleuritis or peritonitis. It forms thick concentric layers over the heart's surface, which may be stripped off in succession. The parietal pericardium likewise becomes lined with lymph, and between the two surfaces there is more or less serous or sometimes sero-purulent liquid. In consequence of the incessant movements of the heart, the surfaces now become remarkably roughened. Sometimes they bristle with a number of papillæ, more often they look honeycombed, or resemble the interior of the paunch of a ruminant. Another good comparison is that made use of by Laennec, and afterwards by Hope, who say that the surface looks like that which would be produced by squeezing some butter between two flat pieces of wood and then suddenly separating them. So shaggy does the heart sometimes look in these cases that the name of *cor hirsutum* was formerly given to it. The progress made by pathology in little more than a century is shown by the fact that Haller described this "hairy" heart as occurring especially in bold and adventurous men.

In other cases, advancing pericarditis leads to the effusion of more and more fluid. When the disease is acute, this cannot much exceed the amount that it is possible to inject into the sac after death, viz. twelve to eighteen ounces; when this limit is reached, the diastole of the ventricles is interfered with and great distress is produced, terminating in the rapid death of the patient. But in chronic cases a much larger quantity of fluid may accumulate in the pericardium, and more than three pints have sometimes been found. The serous effusion is sometimes tinged with blood, particularly in scorbutic and cancerous cases.

Suppuration appears to be much less frequent than in other serous sacs, but this probably depends rather on the preponderance of rheumatism as a cause than on any special tendencies of the membrane itself to one morbid change rather than to another. It is not uncommon in cases of Bright's disease.

In very rare cases the fluid in pericardial effusion has been known to undergo decomposition with the evolution of fœtid gas. This condition, *hydro-pneumopericardium*, somewhat less infrequently arises from the extension of disease from a mucous surface (as that of the œsophagus or stomach) to the serous sac.

The inflammation often extends from the pericardium to adjacent parts. Thus pleurisy may be set up, especially on the left side. In other cases the mediastinal tissues become affected, so that the parietal pericardium is fixed to the sternum by dense adhesions. The areolar tissue above the heart may participate in this change, and the left innominate vein may have its coats very greatly thickened and its cavity plugged with coagulum, a point of some clinical interest, as accounting for the occurrence of œdema of the left arm, without tendency to general dropsy.

Another very important extension of pericarditis is to the heart. The outermost strata of muscle are then found soft, and of a pale yellow or dull

greyish-red colour. On microscopic examination it is found that superficial fatty degeneration of the myocardium has taken place.

Signs.—The diagnosis of pericarditis in practice turns upon the discovery of the physical signs of the disease, and can scarcely be based upon the other symptoms taken by themselves. The earliest of the physical signs is generally the friction-sound or *rub*. It is true that in some cases, even before this can be heard, the onset of pericarditis may be suspected from the heart's action becoming disturbed and tumbling, and the first sound noisy and prolonged, but even then the detection of a rub first converts this suspicion into a certainty. A pericardial rub sometimes lasts for weeks, the disease leading to little beyond the effusion of lymph; in other cases it rapidly disappears, after being audible for a few days, or only for some hours. This often is due to the fact that the two serous surfaces have become separated by fluid, and consequently no longer rub together as the heart moves. The presence of this fluid is indicated by special signs, the most important of which is an increase in the area of the cardiac dulness; and in practice it is generally found that the augmented dulness is first discoverable at the base of the heart. Instead of the percussion-note in the third left interspace being but little less resonant than at the corresponding point of the opposite side, it may be completely dull, and this dulness often reaches as high as the second rib and has been known to extend above the clavicle. When the quantity of fluid is considerable, the left lung is pushed to one side and compressed, and dulness on percussion may exist over so large a part of the left side as to cause the case to be mistaken for one of pleuritic effusion. Another sign of pericardial effusion, if extensive, is bulging of the præcordial region with widening of the intercostal spaces, and occasionally the diaphragm may become so depressed that the epigastrium bulges forwards.

Comparatively small quantities of fluid may suffice to separate the heart from the chest wall, and its impulse may consequently be diminished or imperceptible, but this sign is far from being constant. On the other hand, an impulse can often be felt in the fourth interspace, slightly external to the line of the natural apex-beat.

In general it may be said that the positive value of the physical signs above enumerated is very great: friction-sound is conclusive as to the presence of lymph; and increased dulness upwards, if developed during an attack of acute illness, proves that liquid has been poured out into the pericardial sac. On the other hand, it is by no means certain that the absence of a pericardial rub can be regarded as disproving the existence of acute pericarditis when some other severe disease is present. One may fail to discover pericarditis a few hours before death in cases of double pleuropneumonia, and yet the heart may be found covered with recent lymph.

Symptoms.—These vary remarkably in different cases; and in Bright's disease pericarditis is often altogether latent. Pain in the cardiac region and in the epigastrium may be most intense and agonising, and may radiate widely over the chest, and down the left arm to the elbow; while pressure over the heart or in the pit of the stomach may cause the greatest distress. But in other instances the patient feels no pain, nor is there any tenderness. It was maintained by Bouillaud and by Addison that pericarditis is painful only when it is associated with pleuritis, the pericardium itself being insensitive both in health and disease. But on the other hand pleuritis is often present when pain is by no means severe.

The heart's action, again, may be regular or irregular, quiet or greatly disturbed and attended with distressing palpitation. When these symptoms are present in an intense degree there is much dyspnœa; the patient can hardly speak for want of breath and because of the tightness of the chest; his features are anxious and drawn; his nostrils dilate with each inspiration; he generally reclines on his back, with his head raised; but it is remarkable that when copious effusion has occurred, he often lies by choice flat in his bed, with scarcely a pillow, since the least elevation of the head produces a tendency to syncope.

Earlier writers on the subject mention the occasional occurrence of violent cerebral disturbances in acute pericarditis. Maniacal delirium, rapidly fatal, has sometimes been the principal symptom; and the case has been regarded as one of cerebral inflammation, until the autopsy showed that the pericardium was the seat of disease. But similar cerebral symptoms occur in acute rheumatism, independently of pericarditis, when the temperature is greatly raised; and this fact raises the question whether, when pericarditis is present, it is really concerned in the production of the delirium. The same may also be said of the choreic movements occasionally observed in pericarditis, for there is a very close relation between acute rheumatism and chorea. Apoplectiform stupor, hemiplegia and convulsive attacks may probably be traced to embolism of the cerebral vessels, from endocarditis.

Dysphagia has been mentioned as a symptom of pericarditis; and has been referred to the pressure on the œsophagus by the sac distended with fluid. Walshe disputes this opinion, and refers it to a nervous or dynamic origin.

Event.—It is a rare thing for acute pericarditis in any of its ordinary forms to be the sole or even the direct cause of death. By Louis the average mortality was estimated at one in six cases; but in the pericarditis of acute rheumatism the immediate danger is far less than this. In Bright's disease, death often follows quickly upon the occurrence of pericarditis as a complication; but even then we may often doubt whether it has been much concerned in causing the fatal result. Sometimes, however, fluid effusion accumulates in so large a quantity as to hamper the heart, apparently by interfering with its diastole.

When recovery is about to take place the symptoms gradually subside, and generally disappear in about twelve to twenty days from the commencement of the disease; the fluid effusion diminishes by absorption, and if the corresponding surfaces of the heart and parietal pericardium are still roughened by lymph, a *redux rub* may be heard on auscultation. After a time this lymph also in great part disappears; but before this occurs the pericardial surfaces are commonly glued together to a greater or less extent, and become permanently adherent. There has been much difference of opinion as to whether such adhesions necessarily occur in every case of pericarditis in which lymph has been effused. Some of the best observers think so, but the fact is open to doubt.

Adherent pericardium.—The physical characters of pericardial adhesions vary greatly in different cases. Sometimes, especially after the lapse of a long time, they are reduced to a mere film of connective tissue, which the fingers can tear through with but little difficulty. In other cases they are exceedingly tough, so that the only way to denude the heart is to strip off all the tissues superficial to the muscular fibres. Then, again, they may be uniformly of great thickness; or they may include masses of altered lymph, accumulated in certain parts of the pericardial sac, and especially round the great vessels.

Lastly, the inflammatory material may in course of time undergo calcification, and the heart thus appear to be enclosed in a bony case.

This condition of obliteration of the pericardial sac by adhesion did not escape the notice of the older pathologists, but it was then supposed to be a congenital defect. Since its real nature has been understood, physicians have contended whether it is or is not of clinical importance; and the general conclusion appears now to be that this depends on the quality of the adhesions. Thin areolar connections appear not to hamper the heart's movements in any way, but a thick mass of hard fibrous tissue surrounding the organ may give rise to serious symptoms. By Hope and others it was maintained that an adherent pericardium always tended to cause hypertrophy of the heart's substance; but it is now known that this was a mistake. If one or more of the chambers is hypertrophied in such a case, this is the effect of some previous disease or of a coexisting valvular affection. On the other hand, the presence of thick pericardial adhesions is often associated with atrophy of the ventricular walls. This may in part be the result of the myocarditis which often accompanies pericarditis rather than of the adhesions themselves, but it is precisely in such cases that an adherent pericardium most often gives rise to symptoms and that it can (if ever) be detected by physical signs.*

The early auscultators attempted to diagnose adherent pericardium by various signs which were not long in being shown to be fallacious. At the present time the point on which most stress is laid is the occurrence of systolic depression at the site of the impulse, while one or two intercostal spaces above this recede at the same time. Slight retraction of the spaces close to the sternum during the systole is by no means uncommon even when the pericardium is healthy; but it appears probable that obliteration of its cavity may generally be inferred when a considerable region of the chest wall is drawn in. Still, however, we may doubt whether this can occur without the pleura over the heart being adherent and the left lung being withdrawn from its natural position, and if so, it might perhaps be met with as a result of old pleurisy apart from any pericardial adhesion.

Some observers have also endeavoured to diagnose pericardial adhesion from the fact of the heart's dulness not being diminished during inspiration or from the position of the organ remaining unaltered when the patient lies on different sides; but these signs are still more uncertain.

Milkspots.—We frequently find the visceral pericardium thickened and opaque in patches, on the prominent parts of the right ventricle in front or of the left behind, or on the auricles. These "milkspots" or "corns," as they have been termed, are not adherent to the parietal layer, and are not produced by acute endocarditis. They are the result of friction, and are seen most often on a hypertrophied heart, and when there is more than usual friction, as from a soldier's old-fashioned crossbelt. They are probably of no clinical significance (cf. Paget's paper in the 'Med.-Chir. Trans.,' vol. xxiii).

Mediastinal complication.—A remarkable pathological condition was first recorded by Griesinger in 1854, in which not only the pericardium is found adherent and enormously thickened, but also the adjacent anterior mediastinum is united with it in a continuous mass of indurated fibrous tissue.

Kussmaul described two similar cases in 1873, and observed in both of

* See the late Dr Barlow's remarks in the 'Guy's Hosp. Reports' for 1847, and Dr Wilks's article in the same 'Reports' for 1871.

them the "pulsus paradoxus," *i. e.* an irregular and frequently an intermittent pulse which becomes altogether imperceptible with each inspiration. Moreover, the distended jugular veins, instead of collapsing as usual with the free entry of blood to the right auricle which follows the expansion of the thorax, became fuller with each inspiration, apparently from the dense fibrous adhesions of the mediastinum dragging upon the innominate veins and superior cava. Similar cases have been described by Traube, Bäumler, and Bauer.

Hydropericardium.—The serous sac may be distended with fluid under two conditions—excessive inflammatory exudation, and passive effusion as part of general dropsy. In the latter case it probably is seldom recognised during life and seldom gives rise to serious symptoms. Moreover, since these large effusions are practically confined to cases of renal dropsy, it will commonly be found that (as in hydrothorax) the effusion is not pure serum but shows by the presence of fibrin that the process is active as well as passive. Accordingly, we may regard hydropericardium as practically the result of inflammation and not of mere dropsy.

When the fluid has distended the sac, the normal cardiac dulness is increased as in dilatation with hypertrophy of the heart, but it is increased upwards and to the left, not downwards, except in the last stage of enormous accumulation. It is then that orthopnœa, irregular pulse, and præcordial oppression are most marked.

Such cases appear for the most part as sequelæ of rheumatism or Bright's disease when the acute symptoms have passed off, and then the diagnosis is not difficult, particularly if the case has been watched from the beginning.

Treatment.—The treatment of pericarditis, according to modern practice, is far from being active, and would have been regarded as very inadequate by the earlier auscultators, who sought to recognise the disease at the earliest possible moment, in order to combat its progress by antiphlogistic measures. It is most instructive to peruse the graphic and confident description by a writer of literary skill like Latham ('Lectures on Diseases of the Heart,' xii—xv) of the signs of acute pericarditis, of the bold treatment of the disease by bleeding and by mercury, and of its rapid subsidence under these measures. At the present time few would regard the danger as imminent, and probably none would believe that the classical treatment would avert it. Venæsection is now rarely employed in this affection, and if leeches are used it is only with the object of relieving distress and dyspnœa, for which purpose they have undoubted value. Mercurials are scarcely ever prescribed: the influence of mercury on inflammation is denied by many of the most competent observers, and pericarditis offers little opportunities for testing its value, since the natural duration of the disease varies greatly in different cases. The therapeutical measures which we now adopt are as follows:—The patient is kept as quiet as possible in bed, the præcordial region is covered with a poultice, a thick layer of cotton wool, or a hot flannel; light fluid nourishment is given to him, with a saline or effervescing mixture, and opium or chloral is prescribed for pain and restlessness.

Nevertheless, not only may a few leeches applied over the sternum in the early stage of pericarditis relieve distress and perhaps limit the inflammation, but, when there are symptoms of embarrassed circulation with orthopnœa and distress, an irregular pulse, arterial anæmia and venous con-

gestion—the abstraction of four or five ounces of blood from the arm is found in some cases to give remarkable relief, and probably is never injurious. But the remedy is used to meet a special complication and not with the intention of curing the disease.

Some physicians apply a blister as soon as a pericardial rub is audible. But beside the practical inconvenience of this treatment, its benefit is more than doubtful, and in cases of Bright's disease blisters should, if possible, be avoided. To relieve pain and quiet the heart's action, Dr Bäumler, from personal experience, much prefers the application of a bladder of ice.

When, however, large effusion has taken place, a blister, quickly followed by a poultice so as to promote free effusion of serum, is undoubtedly efficient in hastening absorption; at any rate it has been followed by a rapid diminution of the dulness which for several days before had remained much more extensive than natural. Iodide of potassium is frequently given, with the hope of favouring absorption, and its efficacy in some allied affections is perhaps sufficient ground for employing it.

When urgent dyspnœa and threatening suffocation arise from the presence of pericardial effusion, paracentesis should be considered, and its performance is justified by the imminence of the danger or by the apparent failure of a large blister to relieve. This was suggested more than two centuries ago by Riolanus, but it appears to have been first practised by Romero, of Barcelona, in 1819 (quoted as the first of fifty cases, in a dissertation on "Paracentesis Pericardii," by Hindenlang, 1879). A hypodermic syringe may first be inserted to remove all doubt as to the diagnosis. A slight incision is then made, and the trocar is passed gently into the fourth or fifth left intercostal space, about an inch away from the sternum so as not to wound the internal mammary artery.

The operation has now been performed in a sufficient number of cases to make it trustworthy; and when, as sometimes happens, the pericardium is found after death enormously distended with pus, we regret that paracentesis has not been done. Aran even injected iodine into the pericardial sac, after removing about two pints of fluid; and the patient recovered. One of the most successful cases of paracentesis pericardii has been published by Dr Samuel West. The patient was a lad of sixteen, who had been suffering from increasing dyspnœa for three weeks before he applied for admission to the Victoria Park Hospital. The physical signs led to the belief that a large pericardial effusion existed, and he was accordingly tapped. Fourteen ounces of pus were withdrawn. In a few days the same amount was again taken away; but the fluid still re-forming, an incision was made into the sac through the fifth interspace and a drainage-tube inserted. Two quarts of purulent fluid were thus removed, and the patient gradually but completely recovered ('Med.-Chir. Trans.,' vol. lxvi).

ENDOCARDITIS

AND

VALVULAR DISEASE OF THE HEART

ENDOCARDITIS.—*The subacute, simple, or benign form—Its relation to rheumatism and its ætiology generally—Its anatomy—Its signs.*

CHRONIC, FIBROID, OR SCLEROTIC ENDOCARDITIS.—*Its origin and anatomy.*

THE ULCERATIVE, OR INFECTIVE FORM.—*Its origin, diagnosis, anatomy, and results.*

VALVULAR LESIONS.—*Their anatomy and ætiology—Effects upon the heart itself and upon other organs—Diagnosis: the murmurs attending on valvular lesions—Their physical cause—Their rhythm—Their locality and conduction—Mitral stenosis—Its signs—Tricuspid stenosis—Mitral regurgitation, and tricuspid—Aortic and pulmonary stenosis and regurgitation—Special prognosis—Functional murmurs—The pulse in cardiac disease—Sphygmographic tracings—General symptoms of valvular lesions—Treatment of organic diseases of the heart.*

Congenital diseases of the heart—Cardiac disease in children—Relative frequency and general prognosis of valvular lesions.

INFLAMMATION of the lining membrane of the heart is very common, and appears under three distinct forms.

SIMPLE ACUTE ENDOCARDITIS.*—The first is an acute endocarditis very closely resembling acute pericarditis, pleurisy, and peritonitis. The structure of the lining membrane of the heart is almost identical with that of the great serous cavities, the chief differences being the absence of close relation with the lymphatic system; this and the fact of the former membrane being continually bathed with the circulating blood are the most important peculiarities of the endocardium to bear in mind. It is remarkable that although the endocardium is continuous with the intima of the arteries and veins no acute idiopathic inflammation of the latter is met with.

The process is acute or subacute in course, mild in its effects on the condition of the body in general, serous and fibrinous (or, as the German pathologists call it, "croupous") in the character of its exudation, adhesive and contractile in its effects. Adhesions, however, can only occur where the parts affected come into contact and hence are limited to the neighbourhood of the valves; "puckering" and the formation of delicate bead-like nodules of fibrin are the characteristic effects on the valves, which have led to the epithet "warty" (endocarditis verrucosa) and "vegetative" being applied to this form of inflammation. Away from the valves it results in opacity and thickening like the milkspots of the pericardium.

* *Synonyms.*—Endocarditis simplex *v.* benigna—E. verrucosa et vegetans—Rheumatic endocarditis.

This process is usually limited to the left chambers of the heart; it is extremely rare to find the right auricle, ventricle, or valves affected, except along with those of the left side, and in the great majority of instances they do not even then share in the inflammation. The exception that the right side is *more* frequently affected by endocarditis in the fœtus seems clearly to show that the more active function of the systemic half of the heart is the cause of its being more obnoxious to disease in extra-uterine life.

Ætiology.—By far the most frequent cause of acute endocarditis is rheumatism, *i. e.* the acute febrile disease associated with multiple synovitis which is often called rheumatic fever. The so-called "chronic rheumatism," "muscular rheumatism," "rheumatic gout," "gonorrhœal rheumatism," and the other disorders which are still often vaguely styled rheumatic show that they have no claim to the title by not producing endocarditis.

The only other diseases which probably produce endocarditis as an occasional complication are chorea, pyæmia (see three cases published by the author in the 'Path. Trans.' for 1866), and scarlatina. Some authors mention smallpox, measles, and diphtheria, and others acute or chronic Bright's disease, but the evidence on which these statements rest is far from conclusive. Even scarlatina is perhaps only operative by its liability to be followed by multiple synovitis, which is itself in most cases genuine rheumatism; a choreic murmur has been most often preceded by rheumatism; and pyæmic endocarditis is very rare and of no clinical importance. Hence we may practically consider acute non-infectious endocarditis as "rheumatic" in the strict sense of the word.

It usually begins early in the course of the disease and occasionally even before the synovitis has appeared, or altogether without synovitis, as seems proved by the testimony of such authorities as Graves, Stokes, Trousseau, and Latham.

Acute endocarditis is more frequent in the rheumatism of children than in the case of adults, and far more common between puberty and thirty years of age than later.

Anatomy.—Endocarditis is recognised by the increased vascularity of the serous membrane and by an exudation which causes some roughness of its surface and thus leads to deposition of fibrin on the valves. It is this lodgment of fresh fibrin on the inflamed surface which constitutes the peculiarity of endocarditis and causes it to differ from inflammation of other parts. The living blood is always ready to deposit fibrin on a roughened surface, and especially when the current is in any way checked. Moreover, as Dr Moxon has conclusively shown, these fibrinous concretions again set up inflammation in neighbouring parts by their rubbing against the surface.

The first indication of endocarditis is seen in redness and vascularity of the valve, afterwards by the production of a row of granulations along the lines where the cusps meet. These are bead-like elevations formed along the delicate curved line on each side of the corpus Arantii where the sigmoid valves touch, and also along the corresponding edges of the mitral or (rarely) the tricuspid valve. If examined microscopically these nodules are found to consist of ordinary inflammatory cell-elements. The whole body of the valve may also become infiltrated with these inflammatory products, and thus being swollen and softened is ready to undergo still further changes. In recent endocarditis these granulations along the valves are the first and only indications of the inflammation. At a later stage they become larger and confluent,

and hanging down into the stream of blood, attract coagulating fibrin, which then collects upon them in large quantities. It is thus seen that vegetations on the valves have a double origin, being in part derived from the valve itself and having therefore a true inflammatory source, and in part derived from a deposition of fibrin from the blood; the two products running into one another and not being clearly separable.

Symptoms of endocarditis.—The presence of endocarditis is indicated by the occurrence of a murmur or bruit heard over the region of the heart. If, for example, in acute rheumatism a bruit arise near the apex of the heart we suppose that inflammation has been set up in the neighbourhood of the mitral valve. If it occur at the base we believe it may be associated with inflammation of the aortic valves; in the latter case the diagnosis is less certain, because bruits of a temporary, probably hæmic, character are often met with in acute rheumatism. The fact of the disappearance, however, of the bruit by no means warrants the positive denial of endocarditis, since there is good reason to believe that an inflammation of the valves may be recovered from; witness the case of the disappearance, on recovery from chorea, of the bruit which in many cases appears to be due to an organic change in the valve. The late Dr Sibson made the observation (and it has been corroborated by others) that, preceding the occurrence of a bruit in rheumatic fever, the first sound may often be prolonged.

The general symptoms of endocarditis are very slight. In itself it probably produces little pyrexia, and only moderate acceleration of the pulse, nor is pain by any means a constant symptom.

The *prognosis* is favourable as far as the endocarditis is concerned. The evils arise first from embolism, and secondly from the deformities of the valves which are likely to be produced.

Chronic Fibroid Endocarditis.*—Acute or subacute inflammation of the valves often leads only to puckering and incompetence of the cusps, with consequent regurgitation of the blood-stream; but often there is so much thickening and contraction, that obstruction is the result, with or without concomitant leaking from incomplete closure. Chronic endo carditis produces similar effects; but while in many cases it is merely the last stage of acute endocarditis, it is in many others a slow and insidious process from the first. Without previous rheumatism, the valves slowly thicken and the ostia contract. This process, chronic from the beginning, is found associated with gout, alcoholism, and cirrhosis of the kidneys. It is not infrequent in youth, but becomes more common in adult and later periods of life. It is often complicated by deposition of phosphates of lime and magnesia, with carbonates of the same bases, in the indurated fibroid tissue. It is thus nearly identical anatomically with atheroma of the arteries, and is associated with the latter affection clinically as well.

The relation to rheumatism is through the acute form; probably more than half of the cases are thus rheumatic in origin. See the author's paper in the 'Guy's Reports' for 1871, and Sir Dyce Duckworth's in the 'St. Bartholomew's Reports' for 1877.

From an analysis of the records in Guy's Hospital, Dr Pitt has found that sclerotic endocarditis, as it affects the mitral valves, and leads to

* *Synonyms.*—Sclerotic endocarditis—Valvular atheroma—Chronic fibroid contracting endocarditis—Endurcissement cartilagineux (*i. e.* fibrous) et osseux (*i. e.* calcareous) des valvules du cœur (Laennec).

stenosis of the left auriculo-ventricular ostium, is frequently associated in men with the more chronic forms of Bright's disease (renal cirrhosis) and in women with uterine disorders which lead to ascending nephritis, or to consecutive renal cirrhosis.

The late Dr Peacock and some other writers have believed that a large number of these cases are congenital, but probably this is very exceptional.

The origin of this sclerotic form of valvular inflammation is therefore to be sought (1) in the acute or subacute rheumatic form, (2) in gout and chronic Bright's disease, (3) in strain on the aorta and its valves from over-exertion, while (4) a certain number of cases appear to be idiopathic from the first, and perhaps congenital.

The symptoms, the prognosis, and the treatment are identical with those of the structural valvular lesions which it produces.

Acute Ulcerative Endocarditis.*—Of late years it has been possible to draw a marked distinction between the simple benign form of cardiac inflammation above described and a more acute, more dangerous, and septic form. It may be compared with the virulent forms of peritonitis which occur in the puerperal state. Beside the usual fibrinous products of endocarditis (which are present in exuberant amount) there is a breach of surface of the endocardium, often leading to rupture of chordæ tendineæ or perforation of a valvular cusp. Moreover, micrococci or septic bacteria are found in the masses of fibrin, and the morbid process is accompanied by pyæmia and other symptoms of septicæmia. Lastly, the detached fragments of fibrin, which in the case of ordinary endocarditis produce only mechanical results by blocking the arteries into which they are carried, become here the means of transport of infective microphytes and ptomaines, which excite similar suppurative, "pyæmic" inflammation whenever they lodge as emboli. Hence this form of endocarditis has been termed "malignant" by Dr Osler in his Croonian lectures (1885), and the whole process was described by Dr Wilks as one of internal or arterial pyæmia.

The *origin* of this form of endocarditis, like that of the preceding, is far most frequently in rheumatism. It often is found as a secondary process engrafted on a chronic inflammation with deformity of a valve. It very rarely begins as a secondary result of chorea, scarlatina, or pyæmia, and is unknown as a consequence of Bright's disease, of gout, or of syphilis. Ulcerative endocarditis, like the benign form, is for the most part confined to the left side of the heart, but sometimes affects the pulmonary or tricuspid valves in addition. It only produces a similar affection of the arteries when an embolus lodges.

Its *diagnosis* depends, first, on the same physical signs as that of the benign form, for the mechanical effect of both on the valvular mechanism is the same; and secondly, upon the raised temperature, and the signs of infective embolism of distant parts. When we find a patient in a state of fever and discover a cardiac bruit; when to this are added hemiplegia, aphasia, or other symptoms of cerebral embolism, albuminuria, and hæmaturia, pointing to embolism of the kidneys, or increased splenic dulness, with a palpable tumour and tenderness in that region; or when acute aneurysm forms in the arteries of the limbs, we may then safely diagnose ulcerative endocarditis.

Anatomy.—The first stages are like those of the benign form, but the

* *Synonyms.*—Acute diphtheritic endocarditis (Eberth)—Malignant endocarditis (Osler)—Infective endocarditis.

fibrinous masses are found more abundantly, and are more easily detached. Hence embolism is particularly common.

When these vegetations are long and move about, they come into contact with the surface of the ventricle and there set up a fresh inflammatory process; so that the part touched is also soon covered with a patch of fibrin. In this manner a vegetation on one valve may bore a hole through another with which it comes in contact. There are few cases in which these secondary effects are not seen, and of the valves, the mitral seems more likely to be affected than the aortic.

Subsequently the texture of the valves becomes involved, the tissue is loosened, and the valves become much altered in structure. The inflammation may go on to *ulceration*, and a rent or perforation may take place so that a complete hole may form in the aortic or mitral valve, which then becomes surrounded by vegetations deposited from the blood. At other times an aortic valve may be found partially detached from its base, or the chordæ tendineæ of the mitral ulcerated and broken; their loose ends floating about and covered with fibrin. As a consequence also of these inflammatory changes, thickening and adhesion or coalescence of the valves may take place, leading to obstruction at the orifices, or on the other hand retroversion of the valves, leading to reflux or regurgitation.

Acute ulcerative endocarditis is not so common an affection as the more chronic process. It often spreads by friction of fibrinous concretions. These rub against the wall of the cavity and there produce an ulceration followed by a further deposition of fibrin, or, proceeding further onward, invade the muscle until an abscess is formed. This finally discharges its contents into the heart and constitutes an *acute aneurysm*. It usually occurs at the root of the valve, and it may sometimes reach the surface of the heart, when if it bursts it sets up a fatal pericarditis. Although the fibrinous concretions spoken of are probably the usual instruments in the production of the aneurysm the latter may also arise independently of them.

In some cases of ulcerative endocarditis the inflammatory products become detached, and infecting the blood, set up a fatal blood-poisoning; micrococci are frequently met with in them, as was first shown by Heiberg, of Copenhagen. The result is infectious embolism causing "pyæmic" abscesses of the spleen, kidneys, and other viscera, including the heart itself, and aneurysms of distant arteries.

Occasionally a diastolic bruit suddenly occurs in the course of rheumatic fever. This implies that the inflammation of the aortic valves has caused ulceration and laceration of one of the segments.

The *prognosis* of ulcerative endocarditis is always very grave. The patients frequently die from pyæmia, but sometimes they recover remarkably. No efficient *treatment* is known; but quinine is given in large doses, and stimulants are usually exhibited.

Chronic Valvular Lesions.*—These changes may, as explained above, arise out of acute endocarditis or they may be chronic from the beginning. One of the commonest changes is for the valves to become thickened by a growth of connective tissue which by subsequent contraction considerably alters their shape; and calcareous matter also is frequently deposited in them. On these sclerotic valves vegetations may arise, so that it is often difficult on

* The description of valvular disease, from this point to p. 978, is from the pen of Dr Wilks.

examining a specimen to decide whether the malformations are the result of a process which was from the first acute, or whether the more striking acute process has not supervened on a previously thickened valve.

Anatomy.—In the case of the *mitral valve* the effect of the chronic disease is very often to produce stenosis or narrowing of the aperture; the wall of the valve, especially towards its free edge, is thickened and the segments may closely cohere. Sometimes there are deposited in it masses of calcareous matter, and this gave rise to the old name of "ossification." The chordæ tendineæ may undergo a similar change and coalesce, so that each papillary muscle gives origin to a single large fibrous column, which may be more or less fluted, or pierced with one or two slits indicating the line of separation between the chordæ of which it was made up. At the same time the cords become much shortened, so that the edge of the valve is drawn down and assumes a "funnel shape," or more rarely appears as a kind of diaphragm between the auricle and ventricle with a small hole in its midst. This is usually styled the "button-hole mitral," and more particularly so if the opening is in the form of a slit. This may be so narrow as scarcely to allow the insertion of the tip of the finger, instead of three fingers, which ought readily to pass through a healthy valve. It is a remarkable fact in cases of stenosis of the mitral valve where much thickening is observable and where a history of rheumatism might have been expected, that often no such evidence can be discovered: the symptoms sometimes can be traced back to childhood and in some cases the disease may have been congenital. It is possible, however, that thickening of the valves, being of a very slow growth might have arisen during some of the slighter forms of rheumatic fever when endocarditis was not suspected.

Mitral stenosis is much more common in women than in men and is always to be suspected in childhood if the heart is affected.

Inflammation of the mitral valve need not, however, always produce stenosis, but the reverse; the chordæ tendineæ may become elongated, and so, being unable to preserve the valve in a due state of tension, allow it to become retroverted towards the auricle; or without retroversion by the mere lengthening of the cords the due apposition of the segments is prevented and the valves rendered inefficient. It must, however, be remembered that regurgitation through the mitral valve, owing to its inefficient closure, by no means proves structural changes in the valve, since it often arises from over-distension of the ventricle.

In the case of the *aortic valves* similar changes may take place; the edges may unite or all three valves join together to form a "funnel" with an aperture at its smaller end towards the aorta. The attached borders between the valves become obliterated so that only a line of projection is left to indicate the original segments. Many good authorities have regarded these cases of stenosis from adhesion as congenital.

In other cases a valve may become thickened and distorted so that it hangs down a mere shapeless mass. This of course produces regurgitation. Sometimes the valve is retroverted; at other times it is perforated by ulcerative endocarditis, or may be found dragged down as if forcibly torn away from the aorta. It may here be remarked that in cases of regurgitation both of the mitral and aortic valves there may often be observed a reticulated patch of fibroid tissue on the auricle or ventricle, and these "water marks" show where the regurgitant stream of blood has impinged.

The *valves of the right side* of the heart may be affected in the same way,

though more rarely. The tricuspid may occasionally show a thickening of its curtains and sometimes be narrowed into a buttonhole. The pulmonary valves are very rarely affected, except in cases of congenital origin from intra-uterine endocarditis of the right side.

Ætiology.—As regards the causes of the changes in the valves, endocarditis accompanying rheumatism is the most common; a similar inflammation may, however, be observed sometimes in pyæmia and scarlatina, and the result of endocarditis is not infrequently seen in chorea, manifested by a fine row of beads along the meeting edges of the mitral and aortic valves, more especially the former. There can be no doubt that the chronic changes found in the aortic valves are frequently brought about by hard labour, especially when the exertion has been of that kind where the arms are rhythmically thrown backward and forward as in rowing or sawing; this is the reason why aortic disease is so rare in women compared with men, and that when it is observed in the former a history of acute rheumatism is nearly always found. It was noticed by older writers that aortic disease was met with much more frequently among sawyers than other labourers, in consequence of their work being done in a pit by lifting a heavy saw.

Although it is universally admitted that hard labour and exercise with the arms of a peculiar kind will in course of time cause damage to the valves it has never been generally accepted that violent exertion may cause a sudden injury to a valve, from which all subsequent organic alterations may spring. The question is constantly arising in consequence of patients who are the subjects of valvular disease maintaining that they were perfectly well until, on a given occasion, they made some unusual exertion by attempting the ascent of a hill, running to catch a train, or, as it was said by two patients, mounting a lofty cathedral spire; after these unwonted feats they became breathless, were unable to advance further, and ever since have been the subject of distress which the medical man had recognised as cardiac. The difficulty in arriving at a just conclusion arises from the fact that these persons are usually middle-aged and had never tested the integrity of their organs before the occurrence of the dyspnœa, and also from the fact that if death occurs two or three years afterwards and the heart is carefully examined, any supposed injuries or lacerations are so changed by subsequent inflammatory processes that it is impossible to declare what is new and what old. On the whole it is probable that healthy valves do not suddenly give way under any physiological strain, but after such repeated strains have produced a chronic valvular inflammation the damaged structure may give way under sudden effort. Moreover, a tendinous chorda or a cusp affected with ulcerative endocarditis may be suddenly ruptured.

Effects of valvular disease on the heart.—It can be easily understood how the deranged mechanism of the heart due to disease of the valves must react on the muscular walls of the cavities and necessarily disturb their normal working power; it is also manifest that this derangement must affect the circulation throughout all the organs of the body. It is highly important to be cognisant of these changes during the life of the patient, since they are productive of marked and peculiar symptoms.

It may be laid down as a rule that obstruction in any cavity or passage causes hypertrophy in the muscular structure behind it, as seen in the case of the bladder in stricture or in the intestine in a chronic obstruction; in

like manner stenosis of the aortic orifice produces hypertrophy of the left ventricle, and stenosis of the mitral a like hypertrophy of the left auricle. Regurgitation through a valve causes enlargement of the cavity, from its being continually over-distended; it follows, therefore, that the left ventricle will be dilated in aortic regurgitation, and the left auricle in mitral regurgitation; in the latter case the enlargement is not excessive, seeing that the blood is thrown back directly on the lungs, which have to bear in part the pressure. It is evident that any obstruction on the left side of the heart must affect the lungs, and so react again on the right side of the heart. For example, in cases of primary pulmonary obstruction, as in chronic bronchitis, where the blood flows with difficulty through the lungs, the right ventricle of the heart becomes hypertrophied, and as a natural consequence the right auricle also; there is thus produced the well-known hypertrophy of the right side of the heart in bronchitis, when the texture of the cardiac walls may be noticed to be remarkably tough. If again we take the case of mitral stenosis we find that the left auricle, in order to propel the blood through the narrow orifice, has more work thrown upon it, and becomes in consequence much hypertrophied, and at the same time its lining membrane opaque and thickened; the blood in the lungs in like manner must be retarded and the right side also becomes enlarged, as in bronchitis. In some cases of mitral stenosis, owing to the whole current of blood being reduced to the measure of the mitral orifice, the left ventricle becomes somewhat diminished in size, and the whole heart smaller than in health. In the cases of disease in which there is an impediment to the flow of blood as well as regurgitation, the left ventricle becomes hypertrophied and dilated to a very great size. This impediment to the escape of blood from the aorta arrests the flow behind, and therefore the left auricle participates in the enlargement, and as a consequence the right side of the heart also. In such a case the whole heart is immensely enlarged, and is called "bovine." It frequently weighs forty-five ounces, and much heavier hearts are sometimes met with.

Effects upon other organs.—If will be convenient to mention in this place the changes which take place in other organs from the long-continued congestion. In the case of the *lung* this may be so great that blood is brought up during life, and the organ is found after death to have blood effused into the tissue; this may be scattered through the substance or seen as large round circumscribed dark masses, constituting what is called "pulmonary apoplexy." These usually occur at the lower parts of the right lung. If the blood do not actually burst through the vessels the engorgement goes on until the capillaries are completely blocked and the alveoli of the lungs become almost obliterated; in consequence of this the lung becomes very dense, hard, and fleshy, and sinks in water, the cut surface being smooth. This condition is sometimes called "splenization." Sometimes, after a time, exudation takes place, a large quantity of pigment is formed, the alveoli are thickened, and the lung becomes dark and granular; this is styled "brown induration." If more marked inflammatory products are thrown out, and some formation of connective tissue takes place, the consolidation which then arises, combined with the granular pigment, produces an appearance which has been termed "brown indurated pneumonia." A close examination discovers the remarkable changes which have occurred in the vessels of the lung from the long-continued pressure; sometimes they have become varicose, and atheromatous degeneration of the pulmonary arteries is frequently seen.

The *liver*, which from the long-continued engorgement becomes much enlarged, is found to be in the state denominated "nutmeg." The arrested flow of blood in the hepatic veins produces a similar congestion of the portal circulation as well as a stagnation of bile in the ducts, the capillaries become choked, fatty degeneration of the cells takes place, and the secretion becomes arrested. The appearance of the liver is altered in a most characteristic manner, the fatty degeneration of the circumference of the lobule giving it a white border, and this white being mixed with the red of the blocked hepatic veins and the yellow of the obstructed ducts produces the nutmeg or "myristicated" appearance. It is still a question whether production of new connective tissue may also take place. Several authors speak of this condition going on to cirrhosis, so that in course of time a structural disease of the liver is produced. This, however, has never been thoroughly proved.

The *spleen*, in like manner, is found in cases of ordinary cardiac disease to be hard, dark, and small, sometimes with fibrinous wedges from emboli. In cases of ulcerative endocarditis it is swollen and soft, as usual in pyrexia from any cause, and often contains congested wedges or infarcts from the presence of septic emboli. These are sometimes found softened down into pyæmic abscesses.

The *kidneys* are large, hyperæmic, tough, and coarse-grained. In the end the congestion leads to a form of nephritis which, without producing the typical form of the large white kidney, may gradually cause shrinking, and thus simulate the appearance of the true cirrhotic kidney.

The *stomach* is intensely injected, often with petechial spots or hæmorrhagic erosions, and its surface covered with mucus.

Altered sounds arising from disease of the valves. — These are called "murmurs" or "bruits;" they are of various kinds, one of the most common being the bellows murmur, or *bruit de souffle;* others are harsh, sawing, or grating, and some musical.*

As to the cause of these morbid sounds, the generally accepted opinion has been that they were due to friction, that is, that the blood, pouring over a roughened surface or passing through a narrow channel, sets up a new sound by the rubbing of its particles against the irregular edges. This was not, however, the original explanation, and of late years the older doctrine has come into vogue as more reasonable and more in accordance with clinical facts. One of the original investigators was Corrigan, who says, "When an artery is pressed upon the motion of the blood in the artery immediately beyond the constricted part is no longer as before. A small stream is now rushing from a narrow orifice into a wider tube, and continuing its way through the surrounding fluid. The rushing of the fluid is combined with a trembling of the artery, and the sensation to the sense of hearing is the *bruit de souffle.*" This doctrine, long put aside, has been more recently supported by Chauveau, who by means of various experiments has shown that when blood passes into a dilated vessel a sonorous jet *(veine fluide)* is produced, and that this vibration of the current

* It is worth mentioning that the French term *bruit* is applied to the natural sounds of the heart; so that what is called a "murmur" in English, is always distinguished in French as a *bruit anormal* generally, or as a *bruit de souffle, bruit de râpe, bruit d'oboe,* &c. *Bruit de souffle* is literally a "blowing murmur," but the ordinary English equivalent, "bellows murmur," is also a translation of the French *bruit de souflet*, a phrase used by Laennec, and stated by Littré to be identical in meaning with the former.

causes the bruit. The sound, therefore, is produced, not at the point of obstruction, but just beyond it, and this is what Corrigan had already pointed out in cases of stenosis of the aorta. The same explanation applies when a bruit is produced by the pressure of the stethoscope on an artery causing an artificial constriction. And to the same cause is due the *bruit de diable*, heard in the jugulars of anæmic subjects where the blood passes into a large dilated vein. In the case of constriction of the mitral orifice the conditions are most favourable for its production, the blood passing through a narrow aperture, and then breaking up into a number of jets in the larger ventricle beyond. Various illustrations have been given from other physical phenomena in corroboration of the doctrine; for example, when water runs swiftly through a narrow orifice it is not at this spot that a sound is produced; but it is beyond, where the stream breaks up into a number of smaller ones, that the noise is loudest. The bruits therefore are not now regarded as due to friction between the blood and the walls, but as due to oscillations in the blood itself or friction amongst its particles after it has passed through a channel of smaller into one of larger calibre. The strength of the sound will be influenced by the rapidity and force of the current, and this is what is observed in the case of the heart, for the sounds vary in intensity according to the strength of the propulsion.

The commonest bruit is that of which we have been speaking; others have been described under the names of *bruit de scie* and *bruit de râpe*. The vibrations which cause these bruits may sometimes be felt by the hand like the purring of a cat, and this thrill was denominated by Laennec *frémissement cataire*. Beside these bruits we meet sometimes a musical murmur (*bruit d'oboe*), the vibrations produced being so related that the symmetrical combination of the fundamental tone with the harmonic overtones produces a musical note. In these cases it is possible that some string or edge vibrates in the blood as the stream passes over it. The musical bruits are usually attendant on marked valvular disease and may sometimes be heard some distance from the body, and very readily down the arms or legs, which act as conductors or natural stethoscopes. By placing the fingers of the patient in the auscultator's ears, and thus converting him into a double stethoscope, such a musical sound is often well heard.

The presence of a bruit denotes the existence generally of disease or imperfection of a valve, but the nature of the affection is discovered by other means, viz. (1) by the rhythm of the bruit or murmur, (2) by its position or seat and by the direction which it takes.

Rhythm of cardiac murmurs.—In considering the action of the heart, it must be remembered that the physiologist has divided its movements into closing and opening, systole and diastole, and that during the former the first sound is heard, and during the latter the second, due to the closure of the sigmoid valves. The first sound is therefore styled *systolic* and the second *diastolic*, but it must be distinctly remembered that the closure of the aortic valves is only one of the events occurring during diastole, this taking place at its commencement, and that the filling of the heart and the contraction of the auricle also occur during diastole.

The heart's action may be looked upon in this wise. The ventricle is a large hollow muscle placed in the course of the circulatory vessels to propel the blood continually onwards; after receiving the blood from the ventricle the aorta contracts by its own elasticity and so keeps up the onward flow of the blood during the cardiac diastole. Behind the ventricle is placed the

auricle, which may be regarded as the dilated termination of the vein acting as a receptacle to supply the heart. The action, therefore, of the whole series is that of contraction of auricle, ventricle, and aorta in regular series. The blood is seen to flow along the veins into the auricle and through the open valve into the ventricle, then the auricle suddenly contracts, followed by the ventricle, and afterwards the aorta contracts. The contraction of the auricle is noiseless, but that of the other parts, ventricle and aorta, produce the first and second sound respectively by the sudden tension of the sigmoid and cuspid valves as they close.

It must be remembered that the series of contractions is complete before it begins again, that is, the aorta has ceased to contract before the auricle comes into action, and therefore the contraction or systole of these two parts is not synchronous. It was a want of knowledge of this, or a direct belief to the contrary by Laennec and others, which so long led to a misinterpretation of the different bruits.

Inasmuch as both auricle and aorta contract during the diastole of the ventricle, any sounds occurring during these times are called diastolic, but nevertheless they are not synchronous, the aorta contracting at the beginning of diastole and the auricle at the end. To avoid, therefore, confusion in terms we are in the habit of saying that any sound produced during the contraction of the auricle or that part of the diastole which is not occupied with the contraction of the aorta is *presystolic.*

If the time of the complete action of the heart be divided into fifths, about two fifths are occupied by the contraction of the ventricle, one fifth by the contraction of the aorta, and nearly the remaining two fifths by the diastole or filling of the auricle and ventricle. The short period of time not occupied by this is taken up by the quick contraction of the auricle.

If we watch the movements of the heart in a living animal we observe a deliberate movement of the ventricle followed by that of the aorta, but the auricle so momentarily contracts before the ventricle that it looks as if the movement of the one passed into that of the other by a kind of screw-like motion. The whole action of the heart indeed is screw-like, the ventricle is spiral, and as it contracts the blood is sent spirally into the aorta, which then takes on the same action, and the sigmoid valves close down, not simultaneously, but one after the other in a similar spiral manner. This spiral movement of the ventricle is due to the arrangement of six or seven distinct layers of muscular fibres crossing one another. The use of the papillary muscles is clearly for keeping the curtains tight during the con traction of the ventricle.

If we now take the three cavities in succession, auricle, ventricle, and aorta, and consider that the valves between these separate cavities may be variously injured, and that new and morbid sounds would thereby be produced, we can easily discern at what period of time these bruits would take place. We have already said that these valves may be affected in such a way as either to cause obstruction or allow regurgitation, and since we have mainly the left side of the heart to deal with, we have to consider the mitral valve between auricle and ventricle causing obstruction or allowing a reflux of blood, and then to consider the aortic valves in like manner. In the first place, it is obvious that if any obstruction at the mitral orifice caused a bruit, the latter would be ventriculo-diastolic (auriculo-systolic) auricular in rhythm, or, as it is usually called, presystolic. If, in the second place, the mitral valve was so diseased as to allow reflux, the sound so produced would

occur during contraction of the ventricle and therefore would be systolic. Thirdly, if disease of the aortic valve were productive of obstruction and a bruit occurred, the latter would take place during the contraction of the ventricle and be systolic. Fourthly, if the disease of these valves allowed regurgitation of blood through them and a bruit were produced during the contraction of the aorta, it would, for the reason before named, be styled diastolic. In considering, therefore, the ordinary lesions of the valves, those on the left side of the heart, the bruits produced by them would be four in number, one auricular, two ventricular, and one aortic, or, in clinical language, one presystolic, two systolic, and one diastolic. All these may be developed on both sides of the heart, making eight in number, but they are mostly produced on the left side only, the presystolic being due to obstruction of the mitral valve, the systolic murmur either to obstruction of aortic valve or regurgitation through the mitral, and the diastolic to regurgitation through the aorta.

The seat of the bruits or murmurs.—The valves of the heart are placed so near together, and so overlap, that the sounds produced when they are diseased are not easily distinguished by their position alone; it is rather by the course which these bruits take in accordance with the laws of conduction that we are able to separate and distinguish them. Nevertheless, it is well to know the relation of the valves to one another and their exact position on the surface. The heart retains a tolerably fixed place in the chest, so that if a perpendicular line be let fall along the left edge of the sternum two thirds of the organ would be on the right side of this line and one third on the left. The base of the heart is at the third rib, or higher if we reckon the appendages. The apex is in the fifth intercostal space or at the upper edge of the sixth rib, two inches below the nipple and one inside it, the nipple being placed pretty uniformly over the junction of the fourth rib with its cartilage.*

The area of cardiac dulness in each direction is about two and a half inches, the lung covering the upper part of the heart leaving a triangular space having its base line running from sternum to apex. The sigmoid valves are placed over the third left costal cartilage, reaching a little above and a little below the pulmonary at the upper edge of the cartilage, the aortic behind and a little below as well as a little nearer the median line; the mitral is found at the third intercostal space a little outside the aorta, the tricuspid is in front of the mitral and is nearer the median line behind the sternum. The aorta passes to the right, following the long axis of the heart, while the pulmonary artery passes to the left.

Although the stethoscope cannot separate the sounds at the seat of their production when these valves are diseased, yet (owing to their taking certain directions as we shall now describe) there are special parts of the chest which we regard as the spots where particular valvular murmurs are best heard, viz. the second right costal cartilage for an aortic bruit, the second left costal cartilage for a pulmonary bruit, the fifth right costal cartilage for a tricuspid bruit, and the apex of the heart (wherever situated) for a mitral bruit.

Conduction of cardiac sounds.—Sounds arising in the heart are conducted by the solid walls of the organ, by the direction of the flow of blood, and by the surrounding tissues outside the organ. If we consider the heart lying deep in the chest, the left side being posterior and covered by the right,

* This varies, however, not infrequently, and cannot be relied upon in the case of women or of children.

which is in front of it, we shall see how enveloped the mitral valve is and through what a mass of tissues any sound produced therein would have to pass before reaching the surface; and if, again, we consider that the valve is attached to the left ventricle, and that the latter touches the chest wall by its apex, it becomes obvious that any sounds produced in this valve would be more readily conveyed to this point than directly to the surface through the right side of the heart and the mass of tissues described. It happens, as anticipated, that all sounds produced in the mitral valve are heard best at the apex of the heart; this is consequently called the *mitral area*. Again, the sounds produced in the interior of the heart are conveyed more readily in the course in which the current runs; consequently the bruits produced in the mitral orifice by obstruction or regurgitation, although better heard at the apex than at the base, would each have its direction determined by the different course of the blood: the bruit produced by the obstruction (or the direct murmur) would be carried to the apex and there limited, while that produced by regurgitation would be carried back to the auricle and therefore heard in the dorsal region.*

In the case of obstruction of the aortic orifice the sound would be carried to the right along the aorta, whilst in the case of aortic regurgitation the bruit would be carried downwards. The course of this bruit is very remarkable and has received several interpretations from different authors. The blood flowing back through the imperfect aortic valves passes into the ventricle, and it might be thought that the bruit would be traceable down to the apex; this is sometimes the case, but rarely. As a rule, the bruit is carried down the middle of the chest, along the sternum, and is heard best at the lower portion of this bone just above the ensiform cartilage. The explanation probably is that the bony tissue of the sternum, lying directly over the aortic valves where the bruit is produced, is so much better a conductor of sound than the fleshy heart, that the bruit is more readily carried down the sternum than along the ventricle to the apex.†

The diagnosis, therefore, of valvular bruits depends upon their *rhythm*,

* It does not appear that the sound is "propagated in the direction of the blood-stream," for sonorous vibrations do not necessarily travel in the direction of the current of translation. It is rather that the direct or regurgitant murmurs are produced, as explained above in the text, not at the narrow passage, but beyond it, where the "fluid vein" is formed. Accordingly, a direct mitral bruit having its origin in the ventricle is propagated to the chest wall, where the ventricle strikes it, *i. e.* at the apex: a regurgitant mitral bruit being formed in the left auricle is conducted to the part of the chest wall nearest to that chamber, as well as by the auriculo-ventricular continuity to the apex; and, by the excellent conducting power of the ribs, along the axilla; a direct aortic bruit originating in the first segment of the aorta is conducted by its walls to the part of the chest wall nearest to it, *i. e.* to the third right rib, and upwards towards the right clavicle; and a regurgitant aortic bruit being formed just beyond the valves is usually best heard where the sounding-board formed by the sternum receives and conducts it. A direct pulmonary murmur, being formed in the pulmonary artery, is conducted, not by the stream of blood, but by the solid walls of that vessel in the direction in which it passes upwards and to the left. A regurgitant tricuspid murmur is not favourably situated for conduction, being formed in the right auricle, and is best heard either immediately over its place of origin or where the left auricular appendix comes in contact with the chest wall.

† Dr Balthazar Foster suggests an explanation of the different course of the bruit (if it is heard loudest at the apex) in the fact that different cusps are affected in different cases, and that if the left sigmoid near the mitral is affected, the blood falls directly back into the ventricle and the bruit is heard best at the apex, whereas if the right or posterior sigmoid valve is diseased, the regurgitant blood impinges on the septum, and the bruit is carried down the right side of the heart. Dr Sibson attempted an explanation by supposing that the sinus arteriorosus, being the shallowest part of the right ventricle, is a better conductor of sound than the left ventricle, and so a bruit passes directly through it.

position, and *direction*. Of the four commoner forms of disease just mentioned, a direct or obstructive mitral bruit is known by its being presystolic, and limited to the apex; a regurgitant mitral bruit is known by being systolic, heard best at the apex and conducted to the axilla and back; an obstructive aortic bruit, by being systolic, heard best at the base and carried upwards to the right; a regurgitant aortic bruit by being diastolic, heard at the base and carried down the sternum. If a regurgitant tricuspid bruit exists, it will be systolic and heard to the right of the ensiform cartilage.

We will now take each of these cases separately.

Stenosis of mitral orifice with presystolic murmur.—This murmur is heard during the latter part of the diastolic period and terminates abruptly with the first sound; it is therefore styled "presystolic." It coincides in time with the contraction of the auricle and its appendage, and is therefore called the *auricolo-systolic* bruit. It is limited to the apex, is characterised by its harshness, and is often "churning" or "grinding," and thus unlike the soft bellows murmur. It is also very variable in intensity, differs in loudness day by day and often according to the posture of the patient. If the finger be placed on the carotid, or on the point of cardiac impulse itself, whilst the ear is over the heart, the exact rhythm of the bruit may be ascertained. It is evident that if the natural first sound of the heart is due mainly to the tension of the mitral valve, it cannot be produced when the valve is converted into a fibrous ring; it is consequently short and sharp and resembles rather the second sound of the heart than the first. Herein lies one explanation of the long failure to recognise the presystolic bruit: the lengthened murmur followed by this short click naturally suggested a systolic murmur followed by the second sound; whereas the click was the modified first sound, preceded by a bruit which occurred during what in health is called the period of silence. The mistake was further strengthened by the almost total absence of the second sound at the apex; this being diminished in intensity partly from being lost in the long harsh auricular bruit and partly from the smaller amount of blood sent into the aorta. It may, however, be heard well at the base and may even be accentuated as in mitral regurgitation. Another difficulty arose in accepting the existence of a direct mitral murmur from the fact that in health no sound is produced during the passage of blood through the auriculo-ventricular orifice and also from the fact that the contraction of the auricle is momentary, whereas the alleged presystolic murmur is both long and harsh. The answer has been that the conditions in disease are altered: the ostium is contracted and the blood-stream obstructed, while the auricle is hypertrophied and its action slow; an abnormal bruit is produced by the passage of the blood, through the narrow ostium and under high pressure, producing a "fluid vein" as it enters the ventricle. It is possible that the first portion of a long presystolic bruit may be produced by the blood passing into the ventricle *prior to* the auricle's contraction, seeing that the auricle and its veins must exert considerable and very abnormal pressure upon it. The cardiographic tracing also which gives in health a slight elevation of the lever corresponding to the systole of the auricle, produces in these cases a long and elevated curve.*

* The above explanation of the presystolic murmur is that offered by Dr Gairdner in 1861 ('Edin. Med. Journ.,' vol. vii, p. 438), and is generally accepted in this country and abroad. It was ably defended by the author of the present work in his paper in the 'Guy's Reports' for 1871, and also in his article in 'Reynolds' System of Medicine.' It is, however, right to mention that the otherwise unsupported opinion of the late Dr Barclay, that the murmur is produced by the ventricular systole, has been revived in an elaborate

Beside the characteristic, rough, long, and ingravescent ("crescendo") presystolic bruit, there is another murmur which denotes mitral contraction. It is audible at the apex and is *diastolic* in rhythm, *i. e.* it occurs during the diastole of the ventricle.* But it differs from the presystolic murmur in that it does not run up into the first sound and impulse, but beginning after the second (or diastolic) sound it occupies the period of ventricular pause, and ceases before the first sound, from which it is separated by an appreciable interval.

The probable explanation is that in these cases the left auricle, being well hypertrophied, succeeds in emptying its contents through its narrowed outlet before the ventricle contracts, so that there is a short interval between the auricular and ventricular contraction. When the bruit is presystolic, the auricle does not succeed in expelling its contents before the ventricular contraction at once reverses the direction of the inflowing blood stream, and ends the obstructive murmur by the first sound or by a regurgitant systolic murmur.

Whatever is the explanation, this diastolic, or, as it is called for distinction, post-diastolic apex-bruit, when associated with other signs of mitral stenosis, is a sure evidence of that lesion. The difficulty is to distinguish it from a diastolic aortic regurgitant bruit, which is exceptionally most audible at the apex. The character of the bruit, which is usually less harsh and more blowing or musical, the evidence of dilatation of the left ventricle, and the peculiar character of the pulse, usually enable one to distinguish the latter.

Laennec recognised this diastolic apex-bruit as a sign of mitral stenosis, and his immediate followers taught the same. For example, Hope writing in 1832, says, "When the mitral valve is contracted, a murmur accompanies and sometimes entirely supersedes the second sound, being occasioned by the obstructed passage of the blood from the auricle into the ventricle during the diastole of the latter."

Another very frequent sign of a narrowed mitral orifice was pointed out by Laennec and confirmed by all subsequent writers—a purring tremor (*frémissement cataire*) felt when the hand is placed over the heart.† It is curious that this observation did not long ago lead to the discovery that the bruit under these circumstances is not systolic; for, if the heart beats slowly, the thrill is distinctly felt during the recession of the apex and not during the impulse. It is very remarkable what an insight Laennec had into the form of disease of which we are speaking. He illustrates his remarks by the case of a youth of sixteen in whom he says "the hand placed over the region of the heart felt the pulsation strongly and accompanied by the purring vibration; the stethoscope applied between the cartilages of the fifth and seventh ribs gave the following results:—contraction of the auricle extremely prolonged, accompanied by a dull but loud sound exactly like that

article by Dr F. C. Turner in the 'St Thomas's Hospital Reports' for 1876. The term "presystolic" was invented by Gendrin, but the recognition of a presystolic murmur as a sign of mitral stenosis is, as shown by the author in the paper above mentioned, undoubtedly due to Fauvel, whose article appeared in the 'Archives Générales' in 1843.

* It is sometimes called "*post-diastolic*," because it is heard, not *with* or replacing the second (diastolic) sound, but *after* it. In fact, the term "diastolic" may be applied in three senses: to the period of diastole of the ventricle, to the diastole of the auricele, or to the second sound.

† "Quand (l'orifice rétrécie) est à gauche, on sent quelquefois à la main un frémissement analogue à celui qui accompagne le murmure de satisfaction que font entendre les chats lorsqu'on leur passe la main sur le dos" (Laennec, 'Ausc. Méd.,' tom. ii, § 634).

produced by a file on wood. This sound was attended by a vibration sensible to the ear and which is evidently the same as that felt by the hand. Succeeding this, a louder sound and a shock synchronous with the pulse announced the contraction of the ventricle, which occupied only one fourth part of the time. From these signs I gave the following diagnosis:— ossification of the mitral valve, slight hypertrophy of the left ventricle."

Another frequent sign of mitral stenosis is *reduplication of the sounds* of the heart. It is obvious that if the two sides of the heart did not act together, four sounds might be heard instead of two. If, however, this want of synchronism occurred, it is still doubtful whether the two first sounds ever could be separated into two, so that double sounds are probably always due to the second being heard twice. In the case under discussion the heart's action is thrown out of gear by the hypertrophy of the left auricle and by the increased pressure on the right side of the heart. Under these circumstances, the two ventricles not acting together, the aorta and pulmonary artery are thrown out of order and a double sound is produced by the closure of the sigmoids.* This double beat, or, when combined with the first, this triple beat of the heart, is sometimes heard under other conditions of impediment to the circulation, but it is most common in the case of mitral stenosis. It is called in France by Bouillaud's name, *bruit de rappel*, and is like a hammer rebounding on the anvil.

For certain reasons the contraction of the auricle is sometimes not equal to the production of a bruit in stenosis, and then the case is characterised by the almost total *absence of the first sound.* This may arise from weakness of the auricle or from less pressure being exerted on it from behind. A thrill might still be felt, since a less number of vibrations than would produce a sound might be palpable to the hand.

In many cases when the mitral orifice is narrowed, the valve is so rigid that blood regurgitates through it; under these circumstances a systolic bruit exists as well as a presystolic; the two may be distinctly separated, though they sometimes run together.

As has been before remarked, we occasionally find cases of stenosis which have occurred in childhood and which have directly influenced the nutrition of the heart by diminishing the amount of blood circulating through the body; the size of the mitral orifice being a measure of this amount. The heart has consequently been much diminished in size.

The *pulse* is sometimes small and regular, and in uncomplicated cases of stenosis is not abnormal to the finger and shows no characteristic tracing by the sphygmograph. Occasionally, when the double beat is present, every systole of the ventricle does not fill the aorta, and the pulse is less frequent than the heart's action. When the pulse becomes irregular and compressible, *i. e.* acquires "mitral" characters, we may be almost sure that obstruction has become complicated with regurgitation.

The pulmonary veins and capillaries are of course engorged by the blood-stream being dammed up. Hence hæmoptysis is frequent, probably more frequent than in any form of valvular disease. Dyspnœa is often a marked symptom. But dropsy with albuminuria and the other signs of systemic venous engorgement only occurs in the later stages of the disease when the

* Dr George Johnson has suggested that one of these sounds may be not valvular, but due to the auricular systole. Dr Sansom thinks that it may occur from a tension of the mitral valve at the time when the blood is thrown back during the diastole of the ventricle, and that therefore it is not a normal second sound.

right heart has yielded and become dilated. Indeed, so long as the left auricle is well hypertrophied and the stenosis of the left ostium not excessive, the evil is compensated and no symptoms result.

Stenosis of tricuspid orifice with presystolic murmur.—This is occasionally met with; the bruit being heard best over the fifth right costal cartilage near the sternum. When after a time the blood is thrown back on the lungs and venous system, then all the other ulterior consequences take place as in the case of regurgitation.

Dr Bedford Fenwick has collected forty-six cases, the majority of which were in young women ('Path. Trans.,' 1881, p. 48). In most of them there coexisted similar stenosis of the mitral valve, and in half there was no rheumatic history giving a clue to their origin.

Imperfection of mitral valve causing regurgitation and systolic bruit.—The bruit is of a blowing character, heard loudest in front at the apex, heard also along the side of the chest under the axilla, and in the back over the angle of the scapula. Owing to the backward obstruction in the pulmonary vessels, the blood pressure in the pulmonary artery is raised and it contracts more forcibly upon its sigmoid valves; this produces an accentuation of the second sound, heard at the base towards the left side. The apex-beat may be slightly lower than natural. The *pulse* in extreme cases is small, compressible, and very irregular.

The other physical conditions are mainly due to the engorgement of the venous system from the blood being continually held back in the vessels. Thus the veins in the neck which are not usually observable are seen markedly to stand out, especially on the right side, where they can often also be seen pulsating. This distension sometimes arises from the impulse given by the carotids; in other cases it is due to the contraction of the auricle, and in others to a real regurgitation through the tricuspid. It is usually the external jugular which becomes prominent, although the internal may also be sometimes seen. The valves are clearly visible and become more so on coughing. The engorged right side of the heart may be felt beating in the epigastrium, and if one hand be placed in this region and the other in the right loin, the whole liver lying between the two hands may sometimes be felt pulsating (Dr F. Taylor, 'Guy's Hosp. Rep.,' 1875). The engorgement of the veins is shown by the enlargement of the liver and by dropsy, also by hæmoptysis; less frequently by hæmatemesis, and sooner or later in almost every case by albumen in the urine, which is scanty and high coloured.

Regurgitation through tricuspid orifice with systolic murmur.—If it be true that in a gorged heart reflux often occurs through the tricuspid, and if, as we know, this is not accompanied by a bruit, we may safely assert that no sound is produced by regurgitation through a healthy valve. When a bruit does exist, it is usually in very chronic cases of bronchitis, and where the valve has become thickened and inelastic, probably on account of its abnormally increased action.

Obstruction of aortic orifice with systolic bruit.—This generally arises when there is some roughness of the valves or disease of the aorta; a simple stenosis without regurgitation not being very common. The murmur is heard at the base of the heart and is carried across the sternum to the second right costal cartilage. The natural first sound heard at the apex is not quite so good as in the healthy heart and is partly covered by the bruit here faintly heard; the second sound which is produced by the closure of

the sigmoid valves is indistinct. The heart is hypertrophied and the apex beat is observed to be slower than natural and more external. The pulse is small, long, regular, and *slow:* almost the only case of a slow pulse with valvular disease.

Obstruction of pulmonary orifice with systolic bruit.—This is due, with the rarest exceptions, to congenital stenosis of the pulmonary orifice. The murmur is loud and harsh. The lesion is usually combined with imperfection of the ventricular septum and other congenital defects. In many cases (but not in all of even extreme atresia of the right sigmoid orifice) there is more or less marked cyanosis (*v. infra*, p. 979).

Imperfection of aortic valves with diastolic bruit.—We have already spoken of this bruit produced at the orifice of the aorta and carried down the sternum. The imperfect valve usually causes a murmur as the blood passes over it into the aorta, and thus, as a rule, in cases of diseased aortic valves a double aortic murmur is present; or, as it is sometimes called, a "see-saw murmur." The two bruits may differ in intensity, which is of the utmost importance in a diagnostic point of view; the louder the first the more favourable the case, and the louder the second the converse; although if the latter be prolonged it does not show so much disease as when the bruit is very short and the natural sounds altogether obscured; for the latter often denotes that the blood is falling back with great facility into the ventricle. This variety is sometimes musical, and includes the loudest of cardiac murmurs, so that it occasionally can be heard at some distance from the patient. Sometimes also a *thrill* accompanying the bruit can be felt by the hand.

The heart in these cases is much enlarged, the apex-beat being felt in the sixth or seventh space, and the area of dulness is increased in all directions. In young persons an actual projection of the chest may be seen. On account of the failure of support to the current of blood in the aorta and of the enlargement of the ventricle, the *pulse* is remarkable and characteristic. It has two well-marked features. First of all a large quantity of blood is sent out into the distended vessels and the pulse is therefore regular, large, and visible to the eye; and, secondly, this large full pulse suddenly collapses under the finger. The reason is that when this large amount of blood is sent into the aorta and the latter contracts, the valves behind, not being competent to support the column of blood, fail to sustain the pulse during diastole, and we are cognisant, on placing the finger over the artery, of this collapse. This pulse has many designations: Corrigan's, the "water-hammer," splashing, collapsing, receding, jerking, locomotive, kicking or shuttle pulse. If a novice, with no intention of feeling the pulse, grasp the wrist of a person suffering with aortic disease, he at once becomes aware of its peculiarity by the remarkable throbbing. Dr Galabin says it is the suddenness or sudden blow which renders the pulse characteristic. There is a want of relation between the amount of blood and the vessel that contains it; hence a very similar pulse is felt in cases of hæmorrhage, and "the pulse of unfilled arteries" is a name sometimes given to it. As might be supposed, the jerk is increased when the limbs are raised, so that by holding up the arm as we grasp the wrist, the character of the pulse is much exaggerated. The pulsation is also felt in the small arteries, as the digital, where it is not usually detected, and the pulsation may also be readily discerned in parts which are highly vascular. Thus, the retinal vessels may be seen to pulsate, and any patch of capillaries on the skin may

be seen to change colour at every beat of the heart. If an artificial blush be made with the nail on the skin and this be watched it may be seen to grow red and pale with every pulsation; this is known as the "capillary pulse." Sometimes the pulsation is propagated to the veins beyond when they also may be seen to beat. This is most observable when the veins are dilated by warmth, as after the hands or feet have been put in warm water.

The systolic bruit is often carried a considerable distance along the vessels and sometimes the diastolic also. In the femoral artery, for example, a double bruit may sometimes be heard. If not, it may frequently be produced by using pressure with the stethoscope.

It may be here remarked that all the signs just mentioned denote incompetency of the aortic valves; it does not follow, however, that they are diseased, as their want of closure may arise from dilatation of the ascending aorta or disease of its coats. For example, a yielding of the sinuses of Valsalva just above the aortic valves has been proved to interfere with their perfect action.

Dr Austin Flint, of Philadelphia, maintains that a presystolic murmur may sometimes be heard in cases of aortic regurgitation. He believes it is due to the vibration of the mitral segments even when the valve is inactive, owing to the over-engorgement of the ventricle from the reflux of blood into it from the aorta.

Imperfection of pulmonary valves with diastolic bruit.—This is the rarest of all valvular lesions, and probably never exists alone. It either accompanies congenital stenosis of the same orifice, or else endocarditis (probably of the most acute and ulcerative form) of the valves on the left side of the heart.

Relative prognosis.—As regards the relative gravity of the valvular diseases, probably aortic obstruction is the least serious; next to this, stenosis of the mitral valve, which with a compensating hypertrophied auricle may endure for years. Regurgitant diseases are far more serious, but mitral regurgitation is less so than aortic, for the latter condition often leads to sudden death.*

Concomitant lesions of the cardiac walls.—These valvular diseases, however, are all less important than those which commence in the cavities of the heart, for cardiac dilatation and fatty or fibroid degenerations give rise to the most urgent cardiac symptoms, often run a rapid course, and not unfrequently terminate in sudden death. To these we will now draw attention.

* This opinion of Dr Wilks is corroborated by Peacock and Bristowe, but there are authors who regard mitral stenosis as more serious than mitral incompetence.

Undoubtedly the gravest of all valvular lesions (excluding those complicated by acute ulceration, "malignant" endocarditis, which proves fatal not so much by its mechanical effects as by its poisonous character) is aortic incompetence with dilatation of the left ventricle. But well "compensated" aortic leaking may go on with care for years, and when mitral dilatation and incompetence become added, the danger is rather diminished than increased. The least grave single valvular lesion is aortic stenosis, with moderate compensating hypertrophy. Still, with inelastic arteries, these cases are occasionally cut short by sudden and fatal syncope.

Intermediate in danger between the two forms of aortic valvular diseases, are mitral obstruction and regurgitation. The former alone is far more often met with accidentally, without symptoms; and in all likelihood will last longer. The latter alone causes severe symptoms, but of all valvular lesions is most efficiently relieved by appropriate treatment. The most common mitral cases are probably stenosis with (usually secondary) regurgitation, and these are the gravest; next to cases of aortic incompetence they offer the shortest prospect of continued life, and they are far from exempt from danger of sudden death.

Before, however, speaking of these primary affections of the cavities of the heart, it is important to remember the changes which take place in them in connection with valvular diseases and may be considered compensatory or conservative; for it is evident that as long as these compensations hold good, the primary fault in the mechanism may be counterbalanced, and therefore it is the failure in these secondary beneficial alterations which mainly contributes to all the distressing effects of heart disease. The enlargement and hypertrophy of the several cavities in valvular disease overcome the imperfections of the valves, which would otherwise be at once felt, but as soon as these compensatory conditions fail, then the symptoms appear with increased force. Each particular case, however, must be considered separately: should the ventricle remain tolerably small in mitral regurgitation, it might perhaps be more effective than if it hypertrophied and sent back the blood on the lungs. But it may be said generally that as long as hypertrophy of the ventricles is equal to propelling the blood through the systemic vessels and lungs, the organism works fairly well, but should the ventricles become over-dilated or thinned, or should the muscle degenerate, then cardiac symptoms at once arise.

The state of the cavity, therefore, and of the muscle, is of equal or of more importance in considering the power of the heart than that of the valves. It follows, too, that if the cavities themselves should from any cause be the seat of primary disease the very worst form of cardiac affection is the result; and this is constantly verified by experience. Therefore, to consider heart disease as an affection of the valves only, is to regard it in a much too partial manner. In advanced age a large proportion of the cases of disease of the heart are those of the muscular tissue only, and if any incompetence of the valves be found this may have occurred as a secondary consequence. Again, in persons excessively fat, the organ may be overloaded with adipose tissue both on the right and left side, so that the ventricles become unequal to their work; or, what is more usual in persons advancing in life, the muscular tissue throughout becomes fatty or soft, and unable to propel its contents. In consequence of the weakness of the tissue the ventricle dilates; this prevents the due closure of the mitral valve; regurgitation may take place, a bruit is developed, and all the symptoms of mitral incompetency ensue. This is probably one of the commonest causes of mitral regurgitation. When the heart is examined after death, although no structural disease of the valve is seen, it is very obvious that with the largely distended vetricle the valves could not have closed. These changes in the muscle may come about in various ways; it may become fatty and degenerate in intemperate persons, or sometimes in the gouty. Dilatation may also occur after severe illnesses, and Dr Goodhart believes this is often the case after scarlatina. It no doubt occurs in rheumatism without inflammation of the serous surfaces, and it may arise after excessive hæmorrhages. In rheumatism, as before said, a real myocarditis may sometimes occur, and its results be seen in fibroid changes in the muscular tissue of the heart many years afterwards. Herein is a very striking example of disease of the heart originating in the muscular substance. The various symptoms and physical signs accompanying these enlargements have already been dwelt upon (pp. 923—926).

It is in these secondary valvular diseases of the heart that the origin of bruits and abnormal sounds has been so much discussed, and it is their investigation which has been mainly instrumental in disproving the old

theory that bruits depend upon the friction set up by a diseased valve. According to the more recent explanation, that they are due to the movement of a number of currents when the blood passes from a smaller into a larger space, it is clear that they may arise under a number of circumstances where no cause of friction obtains. We can see how a want of relation between the vessel and the blood, as where a vessel is not only dilated, but the fluid within scanty and poor, can produce a tumult among the particles and a bruit result. In anæmia such a bruit is heard at the base of the heart and directed generally towards the left side in the direction of the pulmonary artery; it is also heard in the carotids and as a continuous murmur in the jugular veins. Another explanation of the anæmic bruit heard so often in chlorosis has been given by Naunyn, and is strongly upheld in this country by Dr Balfour, that it is a valvular murmur and due to regurgitation through the mitral valve, that the ventricle fails fully to contract, that the mitral does not close, and the blood, being thrown back into the auricle, causes a bruit to be heard at the base over the auricular appendix of the left auricle. The theory is not generally accepted, for the reason that the auscultatory phenomena do not agree with those which all admit exist in undoubted mitral regurgitation. Should, however, the theory not hold good in this case, it is probable that there are temporary and functional relaxations of the mitral valve, by which for a time it ceases to be efficient, and yet after a time recovers itself as the blood improves in quality and the tissue in strength.

But even with these explanations many bruits exist whose origin has not been made clear; for example, the very common case of endocarditis evidenced by vegetations on the mitral valve is accompanied by a bruit, the cause of which is not at all obvious. It may safely be asserted, since the first sound of the heart is due mainly to the stretching and closure of the mitral valve, that the causes of the natural sound must be seriously interfered with when this valve is thickened or covered with vegetations; but this scarcely explains the existence of the loud *bruit de souffle* which is so often heard in chorea. It has been suggested that in such a case the incompetence of the valve may be explained on the supposition that, being loaded with vegetations, its specific gravity would be altered and therefore it would not float up in the natural way. In other cases where a temporary dilatation of the ventricle may be supposed to exist it has been thought that the blood, circling in eddies behind the curtains of the valve, might produce abnormal sounds.

There are still other bruits which have not yet received a full explanation; for example, a bruit heard under the clavicle, more especially on the left side and occurring intermittently. This in some cases is obviously in the subclavian artery, and has been thought to be due to muscular pressure on the vessel. In some cases, where there is a suspicion of phthisis, it has been thought that a compression of the pulmonary artery by a consolidated lung may have caused it.

Occasionally bruits may be heard which apparently depend upon movements of parts altogether outside the heart, and these are called *exocardial*. They depend sometimes upon adhesions between the sac of the pericardium and the pleura, which becomes stretched during the heart's action. They are clearly outside the heart when they alter or cease with the respiration; for example, altogether disappearing when the patient holds his breath in the extreme condition of expiration or inspiration. It has been suggested

that when these intermittent sounds resemble bellows murmurs, the heart may be beating against an isolated tongue of lung and that at every stroke a portion of air is driven out of it.

Beside the more ordinary murmurs arising from well-known causes which we have been describing, we occasionally meet with others which have a special origin, and these require all our acumen to unravel; for example a bruit might be caused by a communication between the aorta and pulmonary artery owing to the rupture of an aneurysm, and the sound be peculiar both in character and rhythm,—perhaps a diastolic murmur at the base of the heart. Then the bruit of a circumscribed aneurysm might be heard at any part of the chest; as might those arising from the various malformations of the heart. One of the commonest of these malformations is that in which the aorta and pulmonary artery come off together from the ventricles or are in some other way united; a loud systolic bruit is then heard all over the chest, especially at the base, and, unlike ordinary valvular murmurs, is often carried upwards. When carried to the left clavicle such a bruit generally denotes a congenital narrowing or partial closure of the pulmonary artery. It is very remarkable that in these latter cases the patients have generally died of phthisis (*v.* p. 980).

The pulse in valvular disease.—This is the wave produced when the blood is driven into the elastic arteries, and they contract again upon it. Many of the peculiarities connected with it have already been dwelt upon, so that we shall merely state concisely its principal characters. It only requires a moment's consideration to see what a large number of elements are in operation to produce a particular character of the pulse at any given time, and therefore what variations it is liable to undergo both in health and disease. If the heart and vessels be regarded as a piece of mechanism, with its valves like a bag and pipes of exit, it will be evident that the pulse must vary with the force and regularity with which the blood is driven out of the impelling organ; it must be affected also by any derangement of the valvular apparatus which close its openings; it will vary also according to the nature of the pipes or vessels through which the fluid flows, viz. their elasticity or rigidity. Since, too, the blood has to be driven not only into vessels, but into the tissues beyond, the readiness with which the latter receive the circulating fluid must necessarily affect the flow and impress itself upon the pulse. Then moreover the character of the blood itself must be taken into account, since fluids of different qualities circulate with different amounts of freedom, and finally as the coats of the vessels are living and their calibre influenced through the nerves as well as the heart, the state of the nervous system must be noted as affecting the pulse in a very marked degree. It is therefore obvious that the movement of the blood through vessels or the pulse is most complex and apt to be influenced by a large number of causes. If we repeat the experiment performed in Guy's Hospital many years ago by Mr Wilkinson King, that of attaching a bristle to the skin over an artery by means of a piece of wax, we at once perceive that the pulse is something more than an up and down movement—that the end of the bristle is making vibrations or curves. If this bristle, which Mr King called the "sphygmometer," could be made to take tracings, you would then see the natural movements of the pulse.

The first thing observable in a pulse is its *rapidity* or number; this, except under peculiar circumstances, is due to the rate at which the heart

is working. If the number be great we usually speak of a "quick" pulse, but, as above explained, we endeavour to limit the term quick to rapidity of the systole of the heart and consequent shortness of the pulse wave, and to use the word "frequency" for the number of pulsations in the minute (p. 897). The *force* of the pulse also depends in great measure on the heart, and the same may be said of its *regularity*. The terms *large* and *small* speak for themselves. The large, full, distended vessel is readily distinguished from the small and contracted one. Fulness, however, does not necessarily correspond with relaxation, nor does smallness with contraction. In the case of imperfect aortic valves or in hæmorrhage fulness may be due to a relaxed vessel, and firmness or tension of the vessels to contraction of their coats from various influences. The older writers used to speak of a "quick, full, and hard" pulse of some kind of inflammations as pneumonic, and of a "hard and wiry pulse" of other inflammations, as of the peritoneum. The *tension* of the vessel has been long observed as a peculiar quality of renal disease. It is important to distinguish between this hardness, which is produced by a temporary contraction of the coats of the vessels, from an actual hypertrophy or thickening of their walls. In Bright's disease both conditions exist, the former preceding the latter. Nervous, and particularly cerebral causes, operate powerfully in slowing the action of the heart, and the slow labouring pulse of brain disease is universally recognised. Then, besides the actual force and varied action of the heart and arteries, and the influence exerted over them by the nervous system, the state of the blood itself will affect the pulse. The freedom with which the blood is delivered into the tissues will react on the blood-vessels and affect the pulse; one occasional result of any impediment to a free circulation is the production of another beat in the vessel constituting one form of the so-called *dicrotic pulse*. Various mechanical derangements of the valves necessarily affect the pulse, as we have already seen. When one or two beats are left out, the pulse is said to be *intermittent*. Intermittence and irregularity have already been considered in connection with functional affections (*supra*, p. 899). It may be observed that irregularity of the pulse is sometimes its normal condition; in a case where this was unknown by the medical attendant during the final illness of the patient it was regarded as an important symptom, but the heart was found perfectly healthy after death.

The *sphygmograph* of Marey consists essentially of a needle which is moved by the pulsating artery, while its vibrating end is made to trace itself on paper. The pulse, as felt by the finger, shows itself in such a tracing as one wave followed by another, and sometimes by a third. The second wave is occasionally felt by the finger, and if so the pulse is called "dicrotic;" the tracing needle, however, shows its existence in all cases. These waves or pulsations are very similar to those which are felt by the hand or foot when placed on an elastic pipe through which water is being pumped. It is the first wave and its height that measures the force of the pulse; when large, it shows strength of the heart and distension of the vessel; when wanting, it shows a weak heart or a relaxed vessel, as in aortic valvular disease.

If, however, we look at a sphygmographic tracing, we see yet another stroke preceding the curves of which we have been speaking. This is often nearly perpendicular, and reaches high above the following curves. It is still a question whether this constitutes any part of the tracing, whether, indeed, there is anything in the pulse to produce such a stroke. It is usually vertical, or sometimes when the heart's action is very quick will even slope

backwards. Some of the best investigators regard it as a mere jerking up of the lever at the moment when the blood enters the aorta, and therefore anterior to the true pulse. This seems to be Dr Galabin's opinion.* If it is really caused by the pulsation of the vessel, it is so intimately blended with the curve which follows that they cannot be separated by the sensation conveyed to the finger. This upright line is called the *percussion stroke.*

The first curve or wave which follows constitutes the most important part of the tracing; this it is which is mainly felt by the finger and by which we judge of the strength of the pulse and the tension of the artery. It is called the *tidal wave.* The end of this curve corresponds with the fall of the vessel and the closure of the aortic valves, and is therefore called the *aortic notch.* The next curve is only occasionally felt by the finger; when so the pulse has been long called dicrotic; this is therefore the *dicrotic wave.*

The first or tidal wave best measures the strength of the pulse. The late Dr Mahomed showed how its amplitude may be found by drawing a line from the apex of the percussion stroke to the aortic notch; if it is much outside this, great tension is denoted. It is clear that if there be little tension the tidal curve will disappear and the tracing be marked by a simple falling line; and if this line fall very low (which would be denoted by a low aortic notch) the vessel is relaxed, as in aortic valvular disease. If the line of the first curve should fall very low towards the notch which indicates the closure of the aortic valves, and then a rebound occur, so as to produce a second large wave we have in the tracing exaggerated dicrotic characters; and if the lever fall still lower, so as to reach below the basal line, and then rise to produce the dicrotic wave, the tracing is called *hyperdicrotic.* We see, then, that the larger the tidal wave and the higher the aortic notch from the base line, the greater the tension of the vessel and hardness of the pulse; whilst on the other hand, the nearer this notch approaches to the line the lower is the arterial pressure. It must be remembered that the finger can detect the hardness or contracted state of the vessel during its diastole as well as systole, although the sphygmograph cannot.

The sphygmograph shows the rhythm and number of pulsations. It exhibits well the hardness of the pulse by the high and raised tidal wave; also a soft pulse by the absence of this; a dicrotic pulse by the marked size of the second wave. Therefore in Bright's disease, aortic disease, and in fevers, the tracing is characteristic. In mitral regurgitation, with the weak ventricle and diminished discharge of blood, the tracing is a mere irregular curved line: in uncomplicated mitral stenosis there would be nothing characteristic. In aneurysm the sphygmograph is often of great service, for if the subclavian artery be given off from the sac the tension is much decreased and the pulse on that side will often be smaller and feebler than the other, but sometimes to an extent that the sphygmograph alone can palpably detect. Dr Galabin says that one of the most characteristic features of the tracing in aortic regurgitation is the extreme amplitude of the percussion wave and the flatness of the diastolic portion. The latter corresponds with the emptiness of the artery during the diastolic period.†

Symptoms of valvular disease.—These may be both subjective and objec-

* See Dr Galabin's valuable papers in the 'Guy's Reports,' 3rd series, vols. xix and xx, and the account taken therefrom, in 'Foster's Physiology.'

† On the use of the sphygmograph, and on many points in the semeiology of the pulse, see Dr Broadbent's 'Croonian Lectures' for 1887, already referred to.

tive, *i. e.* those which affect the feelings of the patient and cause him to seek medical relief, and those which are only discoverable by the physician.

It has already been explained that so long as the hypertrophy of the heart overcomes the imperfection caused by the valves, no disturbance in the circulation need arise, and consequently no unpleasant symptoms be experienced; but immediately the cavities, and particularly the ventricles, become unequal to the increased labour thrown upon them, all the symptoms denoting cardiac disease ensue. Should the imperfection in these cavities be a primary one, owing to degeneration of the muscular tissue, it is clear that the case is from the commencement one of the worst forms of heart disease. Whether the weakness of the heart is a primary or secondary affection, various distressing symytoms ensue, arising from the impeded and disturbed circulation, the most striking being the engorgement of the venous system, together with the various changes in the organs before noticed, including dropsy. In the treatment of heart disease, therefore, our efforts should be especially directed to strengthen as far as possible the weakened heart as well as to relieve the symptoms induced by the overloaded blood-vessels.

As regards the several symptoms, we will mention first the subjective, and the most important of these is *cardiac dyspnœa*. The patient becomes breathless upon increased exertion, so that walking up-hill or upstairs, or even dressing in the morning distresses him. He is seen to pant, but has not dyspnœa in the common sense of the term, for he has no difficulty in filling his chest with air. The difficulty is in the blood meeting the air. Since the blood is not being pumped through the lungs with regularity, the relation between the respiration and circulation is consequently lost and the breathing becomes irregular; and the patient has to stop breathing for a moment in order to recover the proper rhythm. If a patient describe to you such symptoms, and if they are genuine, no doubt can exist as to the fact of the heart being weak or diseased, even if it cannot be proved by physical signs.

Another symptom which patients sometimes complain of is *palpitation*. This is by no means so important as the dyspnœa, since it is a symptom so very commonly produced by nervous and other causes acting on the heart from without, as was shown before (*supra*, p. 902). Palpitation is altogether subjective and is rarely felt except the heart beats intermittingly. Now, in some of the worst forms of cardiac disease the action is slow and regular. Then nothing is perceptible to the patient; and even when the heart is very irregular, there need be no consciousness of it. It is more especially in the intermittency arising from functional disturbance that the patient is conscious of a beating or fluttering, and seeks advice for what he believes to be "heart complaint." Probably if all the cases in which a patient complains of palpitation were put together, it would be found that in a very large majority of them there is no disease of any kind and that the patient is only nervous and dyspeptic. It is worthy of remembrance, however, in forming a diagnosis in such cases that although marked dyspepsia and flatulence may be found to be the causes of the patient complaining of palpitation, yet these gastric symptoms are very frequent concomitants of heart disease. They are observed especially after a meal and very frequently on the patient lying down at night, when constant eructations take place from the stomach; the case might then be regarded as one of gastric disease when it is really cardiac. The relations of the stomach and heart through the pneumogastric nerve are very intimate, and thus we can understand how one organ participates in the weaknesses and disturbances

of the other. The dyspnœa being greater when the patient lies down is probably due to mechanical causes, the upward pressure of the stomach and intestines. This is one reason why he sometimes prefers to sit all night in a chair, with his head leaning forward.

The patient with heart disease, after describing his symptoms of breathlessness, of palpitation, or of dyspepsia seldom fails also to dilate upon his disturbed and *sleepless nights*. He says as soon as he falls off he starts up in fear of suffocation. There are many reasons for this; as the heart requires the largest expanse of lung for the circulation, so the best position for the patient is the dorsal one with the head raised, a position to which he had previously been quite unaccustomed, and therefore incompatible with the usual rest; then again, for the production of sleep a quiet regular circulation through the brain is necessary, but this is often impossible when a heart is beating too frequently and with various degress of force. Then besides these there is another cause, probably the most important, which seems to be due to some action on the medulla oblongata rendering it less susceptible of its function in carrying on the respiration; for although it is said that the spinal system never sleeps, the statement is true only in a sense; during ordinary sleep the deep breathing certainly indicates a change in the respiratory process. In disease of the heart this change, probably due to an altered vascular state of the medulla, immediately produces suffocative symptoms and awakens the patient. A peculiar form of respiration (which may generally be regarded as a fatal sign) is the Cheyne-Stokes respiration, *i. e.* ascending and descending respiration, marked by a series of respirations becoming shallower and shallower until they altogether cease; then after a long pause the series begins again.

These are the chief subjective symptoms complained of, but before long the disturbed circulation gives rise to positive objective signs in all parts of the body. In mitral disease, in a certain number of aortic cases which have advanced to the last stage, and in various primary affections of the muscular tissue of the heart, the venous circulation becomes obstructed and the changes already described ensue in the various organs of the body. The cardiac apnœa becomes aggravated by actual *congestion of the lungs:* the patient has a cough, and begins to expectorate mucus tinged with blood, and sometimes enough comes up to be called hæmoptysis.* On examining the patient, besides discovering mucous râles, we find that the lower part of the chest is imperfectly filled with air, and perhaps there is dulness on percussion at the base of both lungs; as a rule it is the lower lobe of the right lung which first becomes blocked, but no satisfactory explanation of this prevalent engorgement of the right side has yet been forthcoming. At this time the lung is in the condition known as brown induration or "splenization," and if blood has been expectorated there are probably effusions of blood in its tissue,—so-called pulmonary apoplexy, recognised after death by dark airless patches which have been compared to damson-cheese. The coloured mucus implies an intense congestion of the bronchial membrane also.

* Dr Shaw, Medical Registrar at Guy's Hospital, finds, from an analysis made for the present edition of this work, of 262 adequately reported cases of valvular disease, that hæmoptysis was most frequent in those of mitral stenosis, much less so in those of aortic regurgitation, with or without obstruction, and least frequent in those of uncomplicated mitral regurgitation. Hæmoptysis occurred in 29 per cent. of the whole number of cases; in 45 per cent. of those with mitral stenosis, and in less than 20 per cent. of those without it. Or, put another way, of the total number of cases of hæmoptysis (76 out of the 262) 45, or more than 59 per cent., were cases of mitral stenosis.

When the lungs are thus gorged, the whole venous system is necessarily obstructed; dropsy occurs in the legs and abdomen, and the veins are seen to be much enlarged in the neck. The skin of the upper part of the body is yellow; this is due to congestion of the *liver*, which is in the state called "nutmeg;" the liver is felt much enlarged, reaching to the umbilicus, and, if the hand be placed upon it before and behind, may sometimes be felt to pulsate. This enlargement of the liver is often a great help to diagnosis. A medical man may be called to a patient for the first time, whom he finds dropsical, with albumen in the urine and a state of heart which, from its weakness and the sounds of bronchitis, is not at once easy to make out. He may be discussing in his own mind the question whether cardiac or renal disease is the proper name to give to the case; but if the liver be enlarged, he at once decides in favour of the former, for no disease of the kidneys would cause this symptom. It is therefore a great aid to diagnosis.

The *kidney* also becomes gorged with blood, and the urine scanty, of high specific gravity, and albuminous; after a time casts may be found, from an actual tubular nephritis being set up in the kidney, just as a bronchitis is produced in the lung. It is important in this case also to discover, if possible, whether the albumen denote independent disease of the kidney or mere congestion, for the prognosis may turn upon the answer. Urine of the kind just spoken of arises from congestion and it does not indicate of necessity any irrecoverable disease of the organ, and yet it is a serious complication, since the impaired action of the kidney aggravates the dropsy. Very often the discovery of disease of the heart and Bright's disease combined seems to have taken away all hope of recovery or even temporary improvement, and yet it is this very combination which may suggest a reason for hopefulness. For example, a patient with cardiac disease has, owing to cold or some unknown cause, an aggravation of his symptoms; the kidneys become congested, they fail to accomplish their due amount of work, and the dropsy, together with all the other symptoms, becomes quickly aggravated; with this double affection the case seems to be desperate, but by treatment and time the kidneys recover themselves, the albumen ceases to appear, the urine begins to flow in natural quantities, the dropsy departs, and the patient once more returns to comparative health.

Congestion of other organs also occurs in heart disease, and the extreme redness of the *stomach* often seen after death is no doubt incompatible with its proper function and therefore one cause of sickness and want of appetite.

As regards the *dropsy*, this commences in the legs, then passes up until it reaches the petitoneum, and afterwards the pleura and the pericardium. Why the fluid should fill one pleura and not the other is not always evident, but it may be due to mere accidental causes, as, for example, to a closely adherent lung on one side. It is said, and probably with some truth, that all pleuritic effusions, both active and passive, affect the left side more than the right. The skin in dropsy often becomes tense, shiny, and red. It may proceed to actual inflammation, including the cellular tissue beneath. Bright observed that inflammation and sloughing were more likely to occur when the kidneys were involved, probably from the semen containing irritating urinary ingredients.

All the symptoms above described arise from venous engorgement: they are most constantly seen in cases of mitral imperfection, whether arising

from primary disease of the valve or from dilatation of the ventricle. In aortic disease, while the force of the ventricle is equal to overcome the difficulty produced by the constant reflux of blood, none of these symptoms occur; but as soon as the ventricle fails in this respect and the mitral valve is unable to close, all the symptoms of mitral disease ensue; at the same time a mitral regurgitant murmur may become audible in addition to the aortic one which was present before. Before this occurs, however, the difference between the case of aortic disease and that of mitral disease is most marked; in the latter the venous obstruction produces congestion and lividity of the face, with a general bloatedness of the body, or actual dropsy; whereas in the former, owing to a small amount of blood being retained in the tissues, they become impoverished, and the patient is universally wasted. Whilst he remains quiet and sits well propped up, no stress is put upon the circulation, so that his appearance is like that seen in wasting disease; in fact he might at first glance be considered to be suffering from phthisis. But closer observation shows strong pulsation of the great arteries in the neck; and the throbbing of the radial artery, which is felt on raising the hand, tells us at once the nature of the case.

There is a class of symptoms observed in disease of the aorta and its valves which are not seen in mitral disease; these symptoms resemble those of angina pectoris. Patients are seized with intense pain in the chest combined with a sense of constriction, and this pain passes across the chest and down the arms; in these paroxysms they sometimes die. They are relieved by nitrite of amyl, by nitro-glycerine, and other remedies which are useful in angina. Sudden death without these symptoms is common in aortic disease, more rare in mitral.

Treatment.—This is of two kinds or has two objects, the one directed against the perverted action of the heart and the other against the effects of the obstructed circulation. For the former great object we have in our hands one of the most remarkable medicines in the Pharmacopœia, one which, if given opportunely, may truly snatch the patient from the jaws of death. He may, for example, be the subject of mitral disease and all its attendant consequences, and may be approaching his end, when *digitalis*—for this is the remedy referred to—is administered; the heart becomes quiet, contracts with more energy, and with this improved action all the symptoms depart and the patient is restored to comparative health. There is no such striking effect seen from the influence of any other drug. A pill consisting of digitalis, squill, and mercury had long been known as a most valuable medicine in cardiac dropsy, but formerly its administrators scarcely understood that it had any other than a diuretic action; it is evident, however, that the digitalis was influencing the heart in a very direct and specific manner, and in this way its action was beneficial. With the knowledge, therefore, which we now have, digitalis is given alone and in larger doses in those cases where its specific action is immediately wanted. Digitalis gives strength and regularity to the heart's systole, it has been called therefore the tonic and opiate of the heart. The fact is known not only by clinical experience but by direct observation on animals. If the chest of an animal be exposed and digitalis administered, the organ is seen to contract more firmly until, becoming tighter and tighter and its action slower and slower, it ceases to beat in the mast complete systole. If the animal be previously poisoned by any substance which relaxes the heart and stops its action during the time of dilatation,

the effect of digitalis is then even more striking, the drug excites it to contraction afresh, which goes on increasing in vigour until it ceases in systole as before.

Now, if in disease we have exactly the counterpart of a heart in this weak state, we have also the same valuable agent at hand. Should a patient have a very irregular and weak action of the heart, digitalis is the appropriate medicine, and since this weak action is usually a concomitant of a dilated ventricle and diseased mitral valve, it is more especially in cases where we have a systolic mitral bruit and irregular heart that we give digitalis. When patients are able to walk about, but are breathless on any increased exertion, and the action of the heart is weak, irregular, and attended with a mitral bruit—digitalis is the remedy. We should begin with ten drops of the tincture three or four times a day for a few days, and then reduce the quantity. A favourable and good form is the combination of digitalis with ammonia and senega; the latter two remedies were highly praised by the late Dr Barlow as supporters of the heart's power. Subsequently, with the smaller amount of digitalis, iron may be beneficially given; and when the digitalis has had its effect, the iron alone or combined with ammonia. Patients with heart disease may have their lives prolonged for many years by the judicious use of these two remedies. Digitalis may often be given continually with advantage, for if its effect be to preserve the quiet action of the heart and give it tone, its long-continued use may produce a permanently tonic state in a heart which had been previously weak. Therefore, besides its immediate use as a sedative to the heart, it acts as a tonic when long continued; and many cases might be quoted where small doses given for months together have been attended by the most beneficial results. The mode in which digitalis quiets the heart is by prolonging the diastole; in cases where the organ is too quickly contracting and fluttering, this drug controls its action until the ventricle is quite filled and the systole takes place. A patient, aged fifty, having a mitral bruit, a very irregular pulse, ascites and anasarca of legs, was given digitalis in full doses until all the symptoms were relieved; he was then ordered a mixture containing the tincture to the amount of seven and a half minims three times a day. Finding the medicine useful, he continued it without intermission for seven months. When seen at that time his heart was beating slowly and deliberately at the rate of 54 per minute; the remarkable point observable was the prolonged diastole.

In cases where this drug has not been given at the onset and where dropsy and all the other symptoms of obstruction have set in, such as congested lung, enlarged liver, and albuminuria, digitalis may even then be given with the same striking results as those just mentioned, and as the heart's action becomes steadied and strengthened, all the engorgements and dropsy may disappear.

If the heart be not very irregular and feeble, and therefore affords less indications for the free use of digitalis, the diuretic pill before mentioned, composed of mercury, squill, and digitalis may be substituted for the simple drug. The combination is valuable for its diuretic effect, although no doubt much of its efficacy lays in the power of digitalis over the heart, a fact unknown to the first compounder of it. Other remedies which act as *diuretics* are also useful in cardiac dropsy, that is, always supposing the kidneys to be healthy: these are for the most part salines, such as the cream of tartar drink. The resin of copaiba also sometimes acts

as a remarkable diuretic. Caffein has been recommended as a good diuretic, but the effects are very doubtful.

Purging is also useful, and compound jalap powder, administered two or three times a week, will sometimes give more relief than any other remedy.

Occasionally, however, the engorgement of the lung will come on so quickly, and the whole venous system with the right side of the heart be so blocked, that *venæsection* is called for and may be practised with the greatest benefit; the indications for this treatment are orthopnœa, lividity of countenance, great distress of breathing, and engorgement of the jugular and other veins. After a few ounces or perhaps a pint of blood has been removed, the lividity will pass off, the breathing become tranquil and often refreshing sleep ensue.

One of the most distressing symptoms of heart disease is the sleeplessness already mentioned. For this *opiates* may be given, not only with safety but with the greatest benefit. One sixth or one fourth of a grain of morphia may be taken at bedtime, or if it produce gastric disturbance, it may be injected hypodermically.

When all remedies have failed to act and the venous obstruction is so great that the whole body has become dropsical, the only resource remaining is to let the fluid run away as it collects, and this drainage may sometimes prolong life for many months. The usual method is to puncture the legs and so allow the fluid to escape. Sometimes the skin ruptures spontaneously, or vesicles form and burst until large sores or ulcers are produced, which continually discharge and keep up the necessary drain. There is always fear, however, whether the skin be broken spontaneously or artifically, of an erysipelatous inflammation being set up and quickly bringing about the fatal end.

Just as digitalis is the true remedy in the weak, irregularly acting heart in order to steady and strengthen it, so is it almost valueless when the organ is hypertrophied and acting steadily. It is therefore of no value in uncomplicated aortic disease with its due compensating hypertrophy. When, however, the enlargement has advanced to that extreme degree that the left ventricle can no longer contract, and therefore the mitral valve is unable to close, all the conditions of primary mitral disease exist. The action of the heart will become irregular and venous engorgement with dropsy ensue. Then digitalis may be usefully administered; whenever, in fact, in aortic disease the pulse becomes feeble, frequent, fluttering, and irregular, as in mitral cases.

In the earlier stages of aortic disease, when the heart is steady, very few remedies are of use, although tonics are sometimes beneficial, as ammonia and senega or iron. Occasionally when the blood-vessels are much relaxed, belladonna has been found useful. If in aortic cases symptoms of angina pectoris occur, then the same remedies may be given as are found useful in that disease, inhalation of nitrite of amyl or the internal use of nitroglycerine.

Additional cardiac remedies.—Extracts and tincture of the flowers and stem of Lily of the Valley (*Convallaria majalis*) have been recently revived as a remedy in similar conditions to those in which digitalis is valuable. The drug is undoubtedly efficacious in raising blood-pressure and thus producing diuresis. It appears to be devoid of danger, and will sometimes no doubt prove useful in cases in which digitalis fails or is counter-indicated. It was employed for dropsy in the seventeenth century, long before Withering introduced the use of digitalis, and it is still a popular remedy in Russia.

Strophanthus, an African arrow poison (*S. cuspidus ;* nat. ord. Apocynaceæ) has been introduced by Dr Fraser as a cardiac remedy. A tincture is prepared from the seeds, which yield an active glycoside, strophanthin. Fraser found that this drug increased the length of the systole in animals, while slowing the rate of pulse, and that it was more powerful than digitalis. It is said not to have a cumulative effect. After thorough investigation in the laboratory it was tried in cases of cardiac, particularly mitral, disease (in doses of five or ten minims of the tincture or more) and with good results. Its remedial action is like that of digitalis, and it sometimes appears to succeed better, at least for a time, but at present strophanthus has shown no title to supersede the older remedy, and not unfrequently entirely fails where digitalis succeeds. See Professor Fraser's paper (in the 'British Medical Journal,' November 14th, 1885), and one by Dr Quinlan, *ibid.*, August 27th, 1887.

Arsenic is valuable as a cardiac tonic and is sometimes even more efficacious than iron. Dr Balfour has been particularly successful in its use : see his remarks in the second edition of his 'Diseases of the Heart' (p. 351).

Congenital Disease of the Heart.—This most frequently depends on a primary defect of development, but also almost certainly upon intra-uterine inflammation, sometimes perhaps of a true rheumatic character.*

Malformation from arrest of development without evidence of endocarditis.—When the heart consists (like that of a fish) of only two cavities, the auricle and ventricle, without any septum making a right and left division, life is rarely sustained after the cessation of the placental circulation at birth. One infant, however, with this defect is recorded to have lived seven days, and two others three days.

With two completely formed auricles and one ventricle (like a frog's heart), from which an aorta arises and supplies both lungs and body, life may be prolonged for weeks or even months (see Dr Peacock's collection of cases in his monograph on 'Malformations of the Human Heart,' 1866), and when the pulmonary artery and aorta are normal, adult life may be occasionally reached even though there is no trace of a septum in the ventricle.

A mere imperfection of the septum between the two ventricles (as in the chelonian reptiles) is a comparatively unimportant defect of development. It almost always occupies the "undefended space" between the base of the left ventricle and the sinus of the right. Persons with this abnormality have lived to adult age in apparent health, and died of some independent disorder.

Results of intra-uterine endocarditis.—It appears from twenty-three cases tabulated by Schipmann that this is rare before the fourth month of fœtal life. By far the most common and important effect of intra-uterine inflammation of the heart is *stenosis of the pulmonary orifice.* The limitation to the right side of the organ really comes under the same law as the corresponding limitation to the left side in extra-uterine life ; in both cases the cavity which has most work to perform suffers most. Why the tricuspid valve should so frequently escape is less clear ; probably it may depend on part of the incoming fœtal blood being diverted to the foramen ovale. Cases of congenital stenosis of the right auriculo-ventricular, however, are recorded.

The effect of this obstruction, occurring before the septum between the

* The most important contributions to the pathology of this subject are by Rokitansky and Kussmaul in Germany, and by Peacock in England.

ventricles is completed, will be to perpetuate the aperture of communication in the undefended space, the ductus arteriosus will remain permeable, and the foramen ovale will continue unclosed.

The first case of this remarkable condition recorded was by Sandifort at Leyden in 1777, and the next was by William Hunter at Glasgow in 1783. In each the patient was a boy who lived to the age of about twelve years. Numerous cases have been since observed, in most of which the aorta communicates with the right as well as the left ventricle. In the most extreme degree of the lesion, the pulmonary artery is completely obstructed (*atresia*), so that not only some but all the blood which reaches the right ventricle is expelled through the aorta, and the lungs are supplied through the ductus arteriosus. The septum ventriculorum is usually open, and under such circumstances life may be prolonged for months or possibly for years. Sometimes the constriction is not at the orifice of the pulmonary artery, but at the junction of the sinus with the corner of the right ventricle, so that the heart has that cavity divided into two, like the ventricle and bulbus arteriosus of a frog's heart. From the position of the opening in the undefended space above described, there is still free communication between the right and left ventricles, and so the results are much the same as those which accompany moderate stenosis of the pulmonary orifice.

Lastly, the effects of endocarditis of the right side may be combined with one or any of the remarkable *transpositions* which occur in the origin of the great vessels from the heart. These combinations have been particularly investigated by Kussmaul, and are of great interest from a morphological point of view, but they are scarcely of sufficient practical frequency to be of clinical importance. Moreover, as we shall see, the physical signs and symptoms of congenital lesions of the heart are rarely capable of leading to more than a general recognition of their presence, and we must be content to base further diagnosis upon the age which the patient has attained, and on our knowledge of the relative frequency of the several lesions.

Prognosis.—In many cases it is remarkable how adaptation and compensation by dilatation and hypertrophy prevent the results which beforehand would appear inevitable. The greatest mortality occurs in the first days or weeks of extra-uterine life. All the worst cases end in death before the infant is a month or two old, and if he survives the first year there is good probability of his being reared. With the development of the body at puberty fresh stress is put upon the heart, and often severe symptoms then first show themselves. Even if adult life be reached, the dangers of exposure to cold producing bronchial and pulmonary inflammation, and of muscular exertion breaking down the compensatory power of the heart are such, that of those who survive childhood few live beyond the age of thirty. The same factors, exposure and exertion, sufficiently explain the undoubtedly worse prognosis for males. Among the longest survivals in cases of (usually very moderate) congenital pulmonary stenosis, we find two cases fatal at thirty, one at thirty-seven, two at forty-four, one at fifty-seven, and two at sixty.

Rokitansky believed that cyanosis protected from tuberculosis, Lebert thought it favoured consumption. In fifty-six cases of malformation of the heart with more or less cyanosis, where patients reached the age of eight, Peacock found that nine died of tubercular disease of the lungs.

When puberty is attained (especially from thirteen to twenty-five) phthisis is undoubtedly the most common cause of death, a remarkable contrast to its rarity in stenosis of the orifices of the *left* side of the heart. The chief

peculiarity of the disease is the frequency of severe hæmorrhage. It does not often run a rapid course, and tubercles of the larynx are said to be rare.

Symptoms.—The most important clinical results of congenital disease of the heart are dyspnœa and cyanosis; to these anasarca succeed, and the other results of mucous congestion and arterial anæmia, which have been detailed in the present chapter.

Cyanosis, or the blue disease (*morbus cæruleus*), was once supposed to be characteristic of congenital cardiac lesions and to depend on mingling of the arterial and venous streams of blood. So William Hunter originally taught, and he was followed by Meckel, by Gintrac and Bouillaud, by Hope, Williams, Walshe and Chevers. But it is not an uncommon result of chronic bronchitis, especially of bronchiectasis in children; and on the other hand it may be absent for many years, notwithstanding grave malformations of the heart and vessels. Still, in its extreme form it is very seldom seen except in cases of malformation, and even when apparently absent it appears on exertion or crying.

The question has been much discussed whether cyanosis, if admitted not to be due to actual mixture of venous and arterial blood, depends more upon non-aeration of the blood or upon venous congestion. The latter view was taken by Louis and Cruveilhier, Rokitansky, Stillé and Peacock. Both conditions coexist in most of the cases now under discussion; but of the two, deficient aeration is probably the more constant. See a valuable paper by Dr Lees in the 31st vol. of the 'Pathological Transactions.'

The blueness is most marked in the lips, the tip of the nose and ears, the fingers and toes; in bad cases the whole face is of a leaden colour, the eyes are bloodshot, and the mucous membrane of the mouth purple in tint. It is increased by exertion, by coughing, and by cold. The fingers and toes are clubbed and the nails incurved. Though the patient is usually comfortable while at rest, exertion brings on dyspnœa. Orthopnœa is less constant and marked than in acquired disease of the heart.

The patient suffers much from the winter's cold, and in marked cases the hands and feet feel chill and clammy even in warm weather. The body is often well nourished and the secretions normal, while the muscular strength and mental faculties are usually not perceptibly impaired.*

In 101 cases analysed by Peacock, he found that symptoms were first noticed at or shortly after birth in 74, within the first year in 15 more, between one and two years after birth in 4, between two and five years in 5, and in the remaining two cases at thirteen and fourteen.

The *physical signs* of congenital pulmonary stenosis are as a rule (1) increased cardiac dulness transversely and to right from hypertrophy of the right ventricle, (2) a long and loud bellows murmur, systolic in rhythm, basic in position, but often unduly diffused, and frequently accompanied with a tactile thrill. The form of the chest, the seat of the apex-beat and the impulse, the character of the second sound, and the rate, volume, and regularity of pulse (when the patient is at rest) are usually unaffected. But the pulse is liable to be easily rendered frequent, irregular, feeble, or intermittent. Occa-

* Dr Peacock, however, says that children who are the subjects of congenital cardiac diseases have generally feeble mental power, and are usually thin. He quotes the following graphic description by Dr Wm. Hunter of his original case (1763): "Though he was remarkably thin, he had not the look of being emaciated by consumption; on the contrary, it appears to be his natural habit. If a man had never seen any of the canine species but the bulldog, he would be struck at the first sight of the delicate Italian greyhound. This young gentleman put me in mind of that animal, and when I looked at his legs especially, I could not but think of the legs of a wading waterfowl."

sionally a diastolic bruit has indicated pulmonary regurgitation, and, perhaps more often, there has been no abnormal sound whatever.

Treatment.—The first indication is to keep up the temperature of the patient's body. A cyanotic patient should be wrapped in cotton wool, and the room kept at an equable temperature. If it survives infancy, it should be clothed from head to foot in flannel, and carefully shielded from exposure to inclement weather, even a degree of cold or of wind which is only healthful to an ordinary child. Fits of crying should be as much as possible prevented during childhood, and all exertion should be carefully restricted. Such children instinctively sit quiet by the fireside, although unfortunately they are sometimes of an easily excitable temper.

The convulsive attacks to which cyanotic persons are subject are often relieved by the application of a few leeches to the temples or below the ears; but great care should be taken that the loss of blood shall not exceed the required amount (Peacock).

Cardiac palpitation, dyspnœa, bronchitis, and flatulence must be treated by the same measures as are found useful in acquired disease of the heart.

Cardiac Disease in Children.—Apart from the congenital lesions just described, children are frequently the subjects of acquired disease of the heart, and in them it presents certain peculiarities of practical importance.

The parts involved are almost always the pericardium and the endocardium. Primary disease of the myocardium and lesions of the aorta are practically unknown. Except in cases of chorea and in exceptional cases of adherent pericardium, there is scarcely any condition but organic lesions of the valves which produces a bruit. Even with considerable anæmia it is very rare to hear murmurs in the heart, the arteries, or the veins. The pulse is naturally frequent in children, and even irregularity of the heart's action may occur in health.

Ætiology.—The origin of valvular disease as observed in childhood is in one of the following causes:—(1) Congenital lesions from malformation or intra-uterine endocarditis of the right side. (2) Rheumatism. This is common in children, but the synovitis and pain are commonly slight and the fever moderate, so that it is easily overlooked. On the other hand, there is great likelihood of the heart suffering, since there is no doubt that fewer children affected with (acute or subacute) rheumatism escape cardiac inflammation than adults attacked in the same way. (3) Chorea. This is often combined with cardiac murmurs, and these are probably organic, but chorea is so often preceded by rheumatism that certainly many, and perhaps the large majority, of these cases are due to the latter cause. (4) Scarlatina. Valvular lesions may not very unfrequently be traced to an attack of scarlet fever; but this disease is so often followed by multiple synovitis of, in most cases, true rheumatic character, that here again most post-scarlatinal cases of endocarditis are really due to the latter malady. (5) Occasionally cardiac disease in children has followed measles, enteric fever, smallpox, or diphtheria, but so seldom that we may consider these diseases to act, if at all, only as conditions of pyæmia. In the vast majority of cases, therefore, disease of the left side of the heart in children means previous rheumatic endocarditis. Girls are more liable to valvular disease than boys (167 to 81 of Dr Goodhart's cases).

Acute ulcerative endocarditis with infective emboli may occur in children, usually as a second attack on valves previously damaged by rheumatism. Atheromatous degeneration of the valves is unknown in childhood.

Anatomy and locality.—The nodules of coagulated lymph, the puckering, contraction, and adhesion of the valves, are what has been already described in the adult. Parrot (quoted by Dr Eustace Smith) has described minute hæmatomata and fibrous nodules on the mitral curtains in infants dying under a month old; these gradually disappear without leading to further changes.

Incompetence is a more common mechanical result than obstruction, but the latter is a far from unfrequent lesion of the mitral orifice in children. An apex systolic bruit is the commonest physical sign, and the lesions are probably, in order of frequency: mitral insufficiency, mitral obstruction, aortic regurgitation, and aortic obstruction.

The resulting hypertrophy and dilatation of the cavities does not differ from that seen in adults.

Symptoms.—It is remarkable that we rarely see the familiar type of either aortic or mitral disease in children, except the latter in the final stages of their illness. The aspect of a child with cardiac disease is rather that of phthisis. He is pale and thin, with dilated pupils, a delicate skin, and a quick, easily excited but usually regular pulse; he has often a short, dry cough, and gets out of breath when he runs.

Sudden death from disease of the heart (or, indeed, from any cause) is very rare in children. The last stage of their illness is general dropsy and exhaustion.

Prognosis and treatment.—For the most part, moderate valvular lesions are well compensated in childhood, and for many years there may be no symptoms whatever. Even when pallor and dyspnœa on exertion lead to an examination of the chest and discovery of the malady, it is often long before palpitation appears, and longer still before signs of venous congestion are added to those of arterial anæmia. The period of puberty and the five or six years that follow it are very critical, and the greater exposure and liability to the strain of labour and of emotion which adult life brings are unfavourable, so that few who have acquired serious lesions of the valves in childhood reach middle age. Some of the worst cases are those of adherent pericardium.

The general treatment consists: first, in fostering the nutrition of the patient by helping the appetite and digestion, and giving a due amount of meat with, in most cases, the addition of a little ale or port wine; secondly, in protecting him from damp and changes of temperature, while, at the same time, seeking to increase his power of resistance by constant woollen clothing, by well-ventilated dwellings and bedrooms, and by life as much as possible in the open air; thirdly, in prohibiting violent games, and all but the most equable and moderate bodily exercise; lastly, in giving the heart long periods of physiological rest by sleep.

In these cases residence in the South of Europe during the winter and spring, and, for those who are old enough, a long sea voyage, if possible on a sailing vessel, often prove most salutary.

Of drugs, steel is the most constantly useful. Digitalis, in small and long-continued doses, is next in importance, while strychnine and arsenic also find their place. From the small experience of strophanthus which the editor has hitherto had in these cases, it has seemed less active than in the adult, and sometimes entirely fails to reduce the number of pulsations. In one case of advanced mitral regurgitation with dropsy, in a girl of about twelve, convallaria acted as a most useful diuretic after digitalis had apparently lost its power.

Frequency and General Prognosis in Organic Disease of the Heart.—The relative *frequency* of the several valvular lesions of the heart is not easy to determine; partly because during life it is often a question whether a murmur is due to functional or organic disease, partly because, even after death, there may be a question as to the perfect competence of a valve, and partly because there are so frequently combinations of more than one valvular lesion. Clinically, an apex systolic bruit is undoubtedly the more frequent; a to-and-fro basic bruit undoubtedly comes next, and a presystolic murmur third.

Mitral regurgitation is by far the most common form of heart disease in children; then mitral stenosis, but only after the seventh and eighth year. Mitral is more common than aortic disease in women; and mitral stenosis more common in women than in men.

Aortic lesions rarely follow mitral, but usually end with mitral regurgitation. Mitral stenosis clinically precedes regurgitation; but, as Dr Goodhart suggests, it is possible that in childhood a puckered and leaking valve may gradually thicken and contract. Tricuspid stenosis is very rare except as a complication of the same condition of the mitral valve. Aortic and mitral regurgitation often go together, aortic stenosis goes with aortic regurgitation and mitral stenosis with mitral regurgitation.

From the *post-mortem* records of Guy's Hospital, Dr Shaw has ascertained that out of 147 cases the mitral valve alone was diseased in 41, the aortic only in 26, and both in 69. On the right side, there were 14 cases of tricuspid stenosis, 7 of its dilatation and thickening, and 5 of disease of the pulmonary valves. In only 3 cases were there lesions on the right side without the left being also affected.

Reference has already been made to the relative prospects of life when a chronic lesion of a valve has been once established, according as the aortic or mitral is the one affected, and according as obstruction or regugitation is the result (p. 967), and also to the important influence of the state of nutrition of the cardiac walls upon the course of disease which follows from a valvular defect. A healthy hypertrophied auricle will often overcome all the impediments of a narrowed left ostium; and a healthy hypertrophied ventricle will compensate a narrowed or even a leaking aortic valve. But when anæmia, fatty degeneration, and dilatation befall the muscle, then the hydraulic effects of the valvular defect become apparent and too often ingravescent.

Of lesions of the right side, pulmonary stenosis is almost always a congenital defect; it leads to cyanosis, and life is rarely protracted beyond childhood, except in the slighter forms of the disease, and even then the patients usually die as young adults from phthisis. Tricuspid stenosis is occasionally seen, but almost always as a concomitant of the same change in the mitral orifice, and probably, if recognised during life, it ought not to add to the gravity of the prognosis. Tricuspid regurgitation is very common, but always as the result either of pulmonary obstruction or of primary valvular lesions on the left side of the heart. It is probably a beneficial event by allowing the high blood pressure to be distributed through the systemic veins and relieved by anasarca.

It is only in the latter stage of aortic valvular disease that dropsy and general venous congestion with albuminuria and jaundice occur, when the left ventricle and the mitral valve have yielded and become dilated. Hæmoptysis or epistaxis belong to its early stages. Severe cardiac pain, urgent

dyspnœa with orthopnœa, excessive palpitation are also miseries which appear early from aortic incompetence. Hæmoptysis is more common from mitral stenosis than from mitral regurgitation. Syncope and sudden death are usually and justly associated with aortic rather than mitral disease. Probably atheroma of the aortic orifice, with considerable stenosis and tolerably compensated incompetence, is the form of valvular disease which most often leads to absolutely sudden death. Bad cases of aortic regurgitation occurring in younger patients as the result of rheumatism produce comparatively slight symptoms, while well compensated; but when dilatation occurs the mitral valve gives, pulmonary congestion and dropsy soon follow, and the case becomes a mixed one; in such cases death may still supervene by terribly sudden syncope, but there has been ample warning beforehand. In mitral cases sudden death occurs more often with obstruction than with regurgitation.

With regard to the *patients' age* at the time of death, Dr Shaw finds that of ninety-five fatal aortic cases at Guy's Hospital (twenty-six without and sixty-nine with concomitant mitral lesions) one died under ten years old, fourteen between ten and twenty, forty between twenty and forty, thirty-three between forty and sixty, and seven above sixty. Of forty-one fatal mitral cases, none died under ten, six between ten and twenty, fourteen between twenty and forty, twenty between forty and sixty, and only one above sixty. This bears out the general experience that the duration of aortic disease is considerably less than that of mitral, *i. e.* the time of death is earlier.

Taking clinical instead of pathological data, he finds that among eighty patients (excluding cases of "malignant" endocarditis), the shortest period which elapsed between the appearance of cardiac symptoms was in "aortic" cases two weeks, in "mitral" four. The average period (not very important, since it may be so much influenced by a single exceptional case) was only two and a half years in "mitral" cases in general, one year and a half in aortic cases with mitral regurgitation also, and as much as four and a half years in simple, *i. e.* well-compensated aortic cases. In cases of mitral stenosis without symptoms or sign of regurgitation, the average duration was three and a half years.

In the most favourable form of valvular disease, aortic stenosis, Peacock records a case exhibiting the most extreme degree of obstruction in which compensation was so perfect that the patient died at seventy-five after the operation for strangulated hernia. With aortic regurgitation the same accurate observer records as the longest period of survival after the existence of the lesion was ascertained, five or perhaps seven years.

With respect to *sudden death*, Dr Shaw finds that there were thirty-four cases in the hospital in the ten years 1875–84, from all causes except injury and hæmorrhage. Of these, thirty-one were ascribed to cardiac syncope; in fourteen the valves were found normal, in twenty they were diseased, the aortic in thirteen, and the mitral in seven, and in all these seven cases the left ostium was *constricted*. Of the above fourteen cases with healthy valves, the cardiac muscle was degenerated in eleven, viz. fatty in eight, and fibroid in three.

So far as prognosis is related to the *cause* of the cardiac lesion, it is better in rheumatic than in atheromatous cases. The most favourable cases, apart from the nature and extent of the lesion, are those in young subjects whose hearts have been damaged by rheumatic endocarditis, but whose

nutrition and renal depuration are vigorous.* The worst cases are those of ingravescent atheroma with confirmed gout and granular kidneys.

As to *sex*, the prognosis is better for women than for men, because in the majority of cases their lives are easier and more tranquil.

Apart from the nature of the lesion, the most important element of prognosis is the kind of life which the patient is able and willing to live. Hard work and exposure, dissipation, starvation, and drink bring otherwise favourable cases to an early fatal result. Moderation in all things, a gentle life, an equable temper, and exemption from attacks of rheumatism, from bronchial catarrh and pneumonia, from muscular strains, and from the excitements of passion, are the conditions which prolong otherwise unfavourable cases to advanced age, and that not unfrequently after a far from useless or unhappy life.

On the whole, the hospital student will find that in private practice he will succeed far better with the same treatment than he could anticipate from his previous experience. Sir Andrew Clark, Professor Gairdner, and others have lately published remarkable facts as to the long duration of life in cases not only of cardiac murmurs, but with conclusive symptoms of organic disease of the heart. ('Brit. Med. Journ.,' Feb. 5th and 12th, 1887.)

From the point of view, however, of insurance the risk is great and difficult to estimate; so that few offices will accept on any terms "lives" which are believed to be weighted with organic disease of the cardiac valves.

* There is in early life a special power of repair and self-adjustment in the heart which warrants our expressing a more cheerful prognosis than would be justifiable in cases of cardiac disease occurring in a grown person (West). (In children), as long as the cardiac lesions gives rise to no symptoms the prognosis is very favourable (Eustace Smith).

DISEASES OF THE BLOOD-VESSELS,

INCLUDING ANEURYSMS AND OTHER THORACIC TUMOURS

ANEURYSM.—*Definition of the term—Varieties—Anatomy—Origin—Symptoms—of aneurysm of the ascending aorta—of the arch—of the descending aorta—Treatment—Abdominal aneurysms—Origin—Diagnosis—Treatment—Dissecting aneurysms—Statistics of aortic aneurysm.*

INTRATHORACIC TUMOURS.—*Of the mediastinum—of the lung.*

THROMBOSIS AND EMBOLISM.—*Causes of coagulation—Effects—Pulmonary embolism—Fibrinous infarction—Cases of recovery.*

EXOPHTHALMIC GOITRE.—*History—Ætiology—Symptoms—Pathology—Event—Treatment.*

THE physical conditions under which the thoracic organs are placed, render the clinical detection of tumours within the chest a more difficult and a less certain matter than it is in either of the other great cavities of the body. For the walls of the thorax being formed by a bony cage, a tumour cannot be felt through them in the same way as through the yielding abdominal parietes. And the contents of the thorax being largely constituted of the soft and yielding lungs, the effects of pressure are not, as in the interior of the cranium, almost necessarily perceptible while a tumour is still small; and they may be absent long after it has attained a very considerable size. These reasons further explain the fact that it is sometimes impossible to carry the diagnosis of a case beyond that of "intrathoracic tumour," using this term in its widest sense, to include aneurysms and some other affections, as well as new growths: and they justify, from a clinical point of view, the collocation of aneurysms and new growths in the present chapter.

INTRATHORACIC ANEURYSMS.—An aneurysm is a circumscribed tumour containing fluid or solid blood, communicating directly with the canal of an artery and limited by the membrane called the sac. There has long existed a question amongst authors as to the need of any further qualification of this definition: whether it should be made to include the case where the whole calibre of the vessel is dilated, and whether in the case of the more circumscribed form it should have any reference to the number of coats involved. As regards the former part of the question, the terms *aneurysm* and *dilatation* are used as expressions of degree; a mere bulging of one side of the aorta would be spoken of as a dilatation, whereas a general dilatation of the whole circumference of the vessel, if limited in length, would be called a *fusiform* aneurysm. Not infrequently one side of the aorta may be so much enlarged as to produce symptoms by pressure on neighbouring parts, and then the expression "aneurysmal dilatation" is often used. This term is one of a practical and clinical significance, as it would not only include a case

where the symptoms were referable to the impeding circulation due to dilatation, but also one where they showed the mechanical pressure of a circumscribed aneurysm. More commonly when we speak of "aneurysm" we imply the existence of a circumscribed swelling of the *sacculated* kind, communicating with the vessel by a distinct aperture.

Nomenclature.—It has been a custom to divide this form of aneurysm into two kinds, *true* and *false*, according to the number and nature of the coats. This is, however, a useless division, not only because the terms have been used in opposite senses but also because the same aneurysm may in the course of time vary with respect to the number of these coats. Scarpa, one of the earliest and most original writers, ascertained the fact that in most aneurysms some of the coats are wanting, and he distinguished them as "true" from mere dilatations or false aneurysms. Subsequent writers used the terms true and false as indicative of the perfection or absence of some of the coats of the vessel. It is better therefore to avoid these terms in any sense of this kind, and to use the word false (if adopted at all) as synonymous with *diffused*, that is to denote the case of an artery in which the blood has burst through the real coats, and becoming effused in the tissues around, has formed out of them new walls wherewith to circumscribe the blood.

Anatomy.—Probably most aneurysms arise from a morbid softening of the inner coat whereby a bulging occurs which pushes the middle and outer coat before it. Before the aneurysm has reached a large size, it is found that the inner coat has become much attenuated or in parts altogether atrophied. The internal coat may be sometimes seen over the whole of the inner surface, and continuous with that of the blood-vessel; in other cases it ceases abruptly at the neck of the sac, or only patches and shreds of it are discoverable over other parts of the interior. Very frequently we find the whole inner coat smooth, but it differs from the original endothelium, and is bound up intimately with the coats below. Whilst the internal and middle tunics are thinned or destroyed, the outer one becomes much thickened, and in many cases constitutes the real sac of the aneurysm. It were in vain therefore to limit the term aneurysm according to the number of coats of the sac or to their perfection or deficiency. In the *fusiform* aneurysm with smooth walls, there is no tendency for the blood to coagulate, but in the circumscribed or *saccular* form with the narrow orifice and roughened interior, coagulation is ever ready to occur. This is also promoted by the greater sluggishness of the circulation within it. When these more circumscribed aneurysms are examined, the interior of the sac is seen to be filled wholly or partially with fibrin: this is arranged in concentric layers, the outer, thin, hard, and pale, and intermixed with the coats, whilst the inner are thicker, softer, and darker. This shows that the deposition has taken place from the coagulation of blood and not from any exudation from vessels or from the walls of the sac. Nor does there seem to be any vascular connection between the sac and the fibrous clot. The mode in which this lamination of fibrin occurs is not very clearly ascertained; in the cases of very rapid cure by pressure the sac is merely filled with a uniform coagulum; and therefore the filling of the sac need not necessarily occur layer by layer, although out of this coagulum distinct lamination appears subsequently to take place.

Ætiology.—Aneurysms arise from special and definite causes, and are not merely due to the general changes found in the vessels from senile and other

degenerations, for they are met with in the young, in the middle-aged, and in those who are otherwise healthy.

In the smaller arteries aneurysms have been demonstrated to arise in connection with *embolism*.*

Generally, however, there is some inflammation leading to *atheroma* in the inner coat, and it is possible that this may begin under the influence of gout, syphilis, or alcohol. That *syphilis* may be a cause had for some time been conjectured, and there can now be no doubt from careful observation in all countries that this is by no means an infrequent antecedent. Then again a strain or violent exertion may seem to have been the precursor of aneurysm, but whether by producing an actual rupture of the coats or by setting up an inflammatory process is not evident. It is a well-known fact that the thoracic aorta is very liable to be affected in those who take violent exercise, especially in those who use their arms in rowing. In such persons the vessels become much altered in configuration, the coats thickened, and the interior atheromatous; thus aneurysmal sacs are liable to occur. The aortic valves also are apt to be affected under the same conditions. These are the causes which are supposed to be especially in operation in soldiers, who are found to be very liable to aneurysm; but it must be remembered that, besides the drill and other exercises, syphilis may have a share in their production.

That violent exertion is productive of aneurysm is shown in the greater liability of *men* than women to the disease. Abdominal aneurysm, for example, is very rare in the female sex. Probably *lead* poisoning may be an occasional cause, for it is remarkable that the workers in this metal not only suffer from gout but from the diseases which so often accompany gout, as granular kidney and diseased blood-vessels; consequently they have been found to suffer not infrequently from aneurysms.

The process which sets up an aneurysm is a true arteritis or inflammation of the deepest part of the intima and is quite distinct from fatty degeneration of the endothelium, which is always superficial. Arteritis involves the middle coat, and then ruptures the endothelium, forming what used to be called an "atheromatous ulcer." The first histological products are leucocytes, then fatty and calcareous degeneration follows, producing a branny detritus (atheroma). See Dr Moxon's paper in the 16th vol. of the 'Guy's Hospital Reports' (Third Series).

Symptoms.—These depend for the most part upon the position of the aneurysm and the consequent disturbance of the neighbouring organs which are implicated. It is therefore convenient to treat of the thoracic and abdominal aneurysms separately, and to divide the former into those of the ascending, transverse and descending thoracic aorta.

(1) Aneurysms of the *ascending aorta* are strikingly different from those of the rest of the arch on account of the enlargement tending towards the front of the chest and therefore not implicating the important structures which aneurysm of the transverse and descending aorta must do.

(*a*) If the aneurysm form a pouch in the sinuses of Valsalva, the valves become involved, their closure may be prevented, a diastolic bruit be produced,

* In a case under the Editor's care in 1883, ulcerative endocarditis was seen to produce first embolism and then aneurysms of the radial and posterior tibial arteries successively. The patient was a young and otherwise healthy man, and he recovered of the acute disease: having only a well-compensated imperfect aortic valve. Each aneurysm was nearly as large as a marble. See Messrs. Langton and Bowlby's important paper, 'Med.-Chir. Trans.,' 1887.

and all the other signs and symptoms dependent upon regurgitation follow. These may indeed be the only symptoms, and none exist indicative of the presence of an aneurysm. The sac never grows to a great size, for it usually bursts into the pericardium and thus causes instant death.

(β) When the aneurysm is somewhat higher up, it still may interfere with the efficiency of the valves and thus produce the before-named symptoms, but if not, it will continue to increase, and often without much inconvenience, until its presence is known by a swelling or projection of the chest. Very often previous to its appearance pains in the chest have been experienced, but not always of a severe character. When it has come forward, it may be recognised as a swelling or bulging of the chest on the right side and usually in the second space. When the hand is placed over it a distinct pulsation is felt; this is synchronous with the heart's action, as may be ascertained by placing the other hand over the apex. Sometimes the touch detects a thrill or *frémissement*. If the swelling should project so far forwards as to be grasped by the hand, a distinct expansion is experienced. This expansive character of the swelling is the most important and distinctive sign of an aneurysm. Without it there is little certainty of its nature, for a pulsating tumour may be nothing more than a growth situated over an artery, and the bruit which is present produced by pressure on the vessel; but no dilatation felt under the hand which grasps it would occur. The expansion may sometimes be well shown by covering the swelling with a piece of plaster in which a slit has been cut down its centre; if this be narrowly watched the slit will be seen to widen at every beat of the sac. If in the early stages a pulsation is felt, but not seen by looking directly at the chest, it may often be clearly observed by placing the eye on a plane with the patient's chest, either by looking over his shoulder when he is in the erect posture, or by stooping the head to a level with his body when he is supine.

On applying the ear a distinct throb or jar is communicated to the ear, and sometimes a murmur is heard. This is, however, by no means always the case, for it depends upon the relation of the sac to the vessel, and is due to the existence of a constricted opening, through which the blood passes into a larger space beyond. It is very rare to hear a diastolic bruit; and if it exists, it must depend upon some peculiar and exceptional circumstance. As a rule the second sound is not only clear, but accentuated.

The heart is not as a rule enlarged in aneurysms of the aorta; it is only when an aneurysm exists at the very commencement of the aorta near the valves that enlargement occurs (as in primary disease of the valves), and then the apex-beat would be found somewhat lower than natural. Occasionally these aneurysms press upon the vena cava, and then some enlargement of the veins of the neck may be observed, or the surface of the chest may be seen covered with distended veins. If large, they may also press upon the bronchus and impede the entrance of air into the lung. Sibilus and rhonchus would thus be produced, and if ulceration took place into the bronchus, hæmoptysis. If the aneurysm approach the axilla, the subclavian vein may be involved, and the nerves of the brachial plexus, so as to cause swelling and pain of the arm. Most frequently aneurysms of the ascending aorta push forward, and absorbing the ribs or passing through the sternum, project outside of the thorax as circumscribed tumours; sometimes they are of a very large size and irregularly bossed; at other times of the size and rounded form of an orange. The skin may become thin and red, but the aneurysm very rarely bursts externally. When it does, blebs form with

thin sanguineous contents, the surface becomes discoloured, and at last a raw surface is formed, which from time to time exudes blood. Even then, however, the elastic corium long resists pressure, and after threatening hæmorrhages have again and again been staunched, the sac usually bursts inwards. The percussion note over an aneurysm is of course dull, the extent varying with its size.

(2) In aneurysm *of the arch of the aorta* the symptoms are more numerous and more important than those of the ascending part. This is owing to the fact of many other structures being implicated. An aneurysm at this part produces all the mechanical effects of a tumour, and this is the reason why new growths and aneurysms are so constantly mistaken for one another, for if the latter be deep seated their peculiar character may be altogether wanting—that is pulsation. If we consider the close packing together of the aorta, trachea, bronchi, œsophagus, pneumogastric, recurrent laryngeal and sympathetic nerves, it is evident that any tumour like an aneurysm forming amongst them must interfere with these structures. If it touch the spine, the bones may be eaten away. The pulmonary artery may also be pressed upon or even opened. The parts usually involved, however, are the trachea or bronchus. These being for some time pressed upon, subsequently ulcerate until the mucous membrane is reached and an oozing of blood takes place. If the aneurism contain much fibrin, and the solid portion be in contact with the air-passages, this oozing of blood or occasional hæmoptysis may continue for a long time, even for weeks or months, and in some exceptional cases for years. Sooner or later, however, the blood breaks through in larger quantities, and thus causes immediate death.

The most frequent seat of aneurysm of the arch is at the origin of the innominate artery, and this vessel is often dilated so as to form part of the sac.

If the aneurysm form in the arch behind the sternum it soon shows itself on the left side as a pulsating tumour about the second left cartilage. If there have been no symptoms this, when first observed, may be mistaken for the auricle. As in the case of the aneurysm of the ascending aorta, it may be seen and felt to pulsate, and occasionally a bruit is heard. Should the aneurysm remain behind the sternum or be formed beneath the arch no external signs of it are apparent. Then all the symptoms are due to its pressure on the parts which surround it.

One of the most common is *dyspnœa*. This may arise either from pressure on the trachea or bronchus, or from implication of the pneumogastric nerves. In the former case there is, as before said, evidence of the pressure by sibilus, and by mucous expectoration tinged with blood. In other cases the difficulty of breathing is due to paralysis of the larynx or its vocal cords. It is usually the left nerve which is involved, which may be found after death closely incorporated with the coats of the sac, and perhaps much thinned. Under these circumstances the muscles of the larynx, especially the posterior crico-arytenoid, are much atrophied. The symptoms due to pressure on the nerve generally differ from those caused by direct pressure on the air-passages, the difficulty of breathing being paroxysmal and the cough peculiar. It is like that of a croupy child, ringing or brasy, and in the intervals the breathing may be free and natural. If the laryngoscope be used it will be found that one of the vocal cords, generally the left, is paralysed and motionless; and sometimes, although only one nerve is involved, both cords become paralysed, causing great difficulty of breathing, and threatening suffocation. It must be remembered that during the act

of breathing the larynx is actively opened, and therefore if the muscles be paralysed it would close during inspiration.*

Pressure on the œsophagus may occasionally cause difficulty of swallowing; but more frequently pressure on the pneumogastric and pulmonary plexus may set up an insidious form of *pneumonia*. It is frequently found after the existence of sibilus, and pressure on the lung, that febrile symptoms ensue, and that one lung is becoming solidified; this slowly brings the patient to an end. The lung is then found to be hepatised or in a state of purulent infiltration, and the cause of this is no doubt to be explained, as Sir William Gull and Dr Budd long ago observed, by an implication of the nutritive nerves.

The implication of the sympathetic nerve in the neck may affect the pupil of the eye, its paralysis causing *myosis*, or contraction of the pupil, in the same way as paralysis of the third nerve causes dilatation. The pupils ought, therefore, to be carefully examined in cases of suspected aneurysm. The left is the more often found contracted.†

As regards *pain*, this varies according to circumstances. As a rule it is a pain in the chest or down the arms which first takes the patient to the medical man. This in course of time may become very acute, and is sometimes accompanied by actual weakness of the arm. The pain may have its origin in the aorta, and so, through the sympathetic and spinal nerves, is at last referred to various parts of the chest and upper extremities. If the aneurism be situated at the lower part of the arch it may erode the spine, and so immediately involve the nerves passing to and from it.

Another important indication of the existence of an aneurysm may sometimes be found in the *pulse*, and especially in one pulse being smaller than the other. This may arise from three causes: the subclavian artery may run outside the sac and be compressed by it; or it may come off from the sac and its mouth be closed by fibrin; or, more frequently, the artery is open, though the aneurysm, being deficient in the normal tension of the aorta, fails to produce due pulsation in the vessels coming off from it. It must be remembered that when the blood is delivered by the ventricle to the aorta, the elasticity of the latter does not prevent each shock being felt in the distant arteries; but if the aorta were converted into a large sac the impulse of the heart would be lost in this space, and the blood would flow out through the efferent vessels in a continuous stream. A case once in Guy's Hospital exemplified this: the pulses at the wrists became quite imperceptible, but the warmth of the hands and their vascularity showed that the blood flowed into them as before. It is the exception, however, for the pulse, as in this instance, to cease completely; the more usual occurrence is for that in one wrist to become more feeble than that in the other. If the aneurysm should involve the innominate the right pulse would be enfeebled, as would be the left if the aneurysm should be in front of the left subclavian. The sphygmograph is found useful in demonstrating on paper the difference between the perfect pulse of one wrist with all its parts, and the imperfect tracing of the other.

* On this subject see, however, Dr Bristowe's remarks in the 3rd vol. of the 'St Thomas's Hospital Reports' (1872).

† Sweating on the same side as the contracted pupil has been recorded in cases of aneurysm by Gairdner ('Edin. Med. Journ.,' 1856), and also by Dr Bramwell (ibid., 1878); but in the latter the hyperidrosis had preceded the symptoms of aneurysm by years, and in cases of undoubted injury to the cervical sympathetic, as well as of its experimental division in animals, the secretion of sweat is checked, not increased, on the affected side (cf. 'Journ. of Physiology,' vol. viii, p. 26).

(3) Aneurysms of the *descending thoracic aorta* are necessarily productive of different symptoms from those of the arch. They soon involve the bones of the spine, which they erode, and expose the spinal nerves.

Pain, therefore, is one of the commonest symptoms and may be the earliest. It is often very defined, as in the course of a particular intercostal nerve, and so the seat of the disease may be accurately determined. The thoracic aneurysm may also involve the lung, or press upon a bronchus, or at other times may compress the œsophagus. If it advance, it may make its way backwards, and involving the ribs be felt at the back; it may even enter the canal and compressing the cord produce paraplegia (cf. p. 481). In the former case the pulsating tumour may be felt and seen, and a bruit be heard with the stethoscope.

Death may occur from implication of the lung causing pneumonia, or by rupture of the sac into the œsophagus, but more usually by its bursting into the pleural cavity.

Treatment.—The object of the physician in treating aneurysms of the aorta is the same as the surgeon has in view in treating aneurysms of a limb, viz. to retard the circulation in the vessel and aid the deposition of fibrin. This is more likely to be effected when the opening of the sac is small and the interior roughened. In the case of a popliteal aneurysm the limb is bent or the femoral artery is ligatured or compressed, and so the blood coagulates; and the same object is attempted in the case of the aorta. The method of cure is to lessen the rapidity and force of the circulation, and, if possible, to increase the deposit of fibrin from the blood. This principle of treatment is confirmed by cases of spontaneous cure of aneurysms in wasting diseases (see Dr Moxon's article in the 'Guy's Hospital Reports,' 3rd series, vol. xii).

For this end the patient must be kept absolutely at rest in the recumbent posture, and be subjected to a strictly limited diet. The original method proposed by Valsalva was bleeding the patient several times and keeping him on the smallest amount of food. This treatment is deprecated by Tufnell, who, however, has revived the method in a modified form with remarkable success. The object, he allows, is to reduce the circulation and keep the blood in a highly coagulable state; but this is not accomplished by starvation. His experience has been mainly with abdominal and local aneurysm, but the same method equally applies to aneurysms of the thoracic aorta. Tufnell allows three meals a day and a small amount of fluid, with absolute rest for at least two months. The value of rest in the recumbent posture he illustrates by the fact that in one patient the difference of the number of beats of the heart between the sitting and recumbent position was thirty-five per minute. This, he says, is equal to more than 50,000 in the twenty-four hours, and no remedy in the Pharmacopœia could produce so striking an effect. The diet he recommends is the following: for breakfast, two ounces of bread-and-butter and two ounces of milk or tea; for dinner, three ounces of mutton and three ounces of potatoes or bread, and four ounces of claret; for supper, two ounces of bread-and-butter and two ounces of tea: the total per diem being ten ounces of solid food and eight ounces of fluid.

Of the two constituents of treatment, *rest* and *low diet*, the former is the more efficient and indispensable. The patient must not sit up to take food, nor be moved to have his bed made or to relieve his bowels. Even suddenly turning over should be avoided, and he should move his arms as little as

possible. Sleep should be procured when necessary by bromides or chloral, or, if there is much pain, by opium pills or morphia injection. The latter drugs are useful in the early part of the treatment by stilling hunger as well as pain, but after a few days the pain usually subsides and the patient sleeps comfortably.

The restriction of solid food is comparatively easy; that of liquid is more difficult. Thirst may be relieved by ice, by effervescing lozenges, or by sucking slices of lemon or orange-peel. While aiming to reduce the water taken to half a pint a day, the diminution must often be gradual and sometimes may stop short of that limit without apparent harm.

After two or three weeks of this regimen, the pulse sometimes becomes very rapid and feeble, with palpitation of the heart. The strict dietetic treatment must then be relaxed, opium with digitalis given for a time, and then the food cautiously diminished again. In certain cases all attempts in this direction are baffled, and it is then wise to let a patient eat and drink moderately and to trust to the effects of rest and iodide of potassium.

In favourable cases pain rapidly subsides and then disappears, pulsation is less violent, the swelling diminishes, and after two months or more there may be neither physical sign nor symptom of the aneurysm. This has occurred even when the sternum has been perforated in two places, and the sacs have both become filled with clot and firmly contracted.

As soon as the tumour has disappeared, the patient's food should gradually be increased; and next he should be allowed to sit up. Lastly, he may be restricted from laborious occupations only, and suffered to walk about as usual; but it is wise to continue the iodide for several weeks and to avoid all excesses in meat or drink, in bodily efforts or mental emotion.

There is little to add as to the use of drugs, when from any cause the treatment by rest and diet cannot be carried out. Digitalis is often given, but probably without any benefit, and the same may be said of lead, ergotine, and many other remedies; the only one which can be said to have any real efficacy is the *iodide of potassium*. This is now given in large doses, fifteen to thirty or even sixty grains, three or four times a day for several weeks; and sometimes with marked success. Under its use the aneurysmal sac becomes hard, as if the remedy favoured the deposition of fibrin. Whilst this is taking place the pain is subsiding, which shows that the pulsation of the sac produces symptoms not due to the tumour alone; for when the latter has become hard and inert they disappear. It is true that the cured aneurysm is smaller than the active one, but this alone will not account for the subsidence of symptoms. The fact is well seen in cases of popliteal aneurysm where the pain and tenderness immediately cease after the arrest of the circulation through the sac.

Dr Balfour has published some remarkable cases of benefit by treatment with iodide, and Dr Bramwell recorded several others in the 'Edin. Med. Journ.' of 1878.

Sometimes, however, the case has gone too far for treatment before it is seen, or the patient may be intractable and perverse; or the treatment, after success for a time, is followed by more obstinate return of the disease; or the tumour threatens immediate rupture. To relieve urgent symptoms which cannot wait for the effect of rest and indeed prevent it, the most efficient remedy is small, and, if needful, repeated *bleeding*. The four or six ounces taken from the arm, recommended by the late Dr Hughes

Bennett, rarely fail of effect. More than once a patient, who had suffered many days and nights of unrelieved pain and sleeplessness, with frequent attacks of agonizing suffering, after ice-bags, morphia in large doses, and other means had failed, has obtained complete relief and sound sleep after a single venæsection.

For the radical cure of thoracic aneurysm, when the plan of treatment above described is inapplicable or has failed, various local measures have been attempted, but not with much success. One of these is *galvanism.* The poles of a battery have been passed into the sac so as to produce coagulation of the blood. The needles must be well insulated, and of course must not touch; but probably the best plan is to use the positive pole only, so as to procure the firm non-gaseous clot which forms on the anode, and to place the cathode as a large wet sponge on an indifferent part in the neighbourhood. This practice has been successful in the hands of Ciniselli, and partly so in those of Dr John Duncan. ('Edin. Med. Journ.,' April, 1866.)

Another plan, introduced by the late Mr Moore, is to pass several yards of fine iron wire into the sac. At Guy's Hospital this has been tried with imperfect success, and horsehair has also been used. The danger is setting up inflammation or sloughing of the sac. Only two patients have survived the operation more than a fortnight; about twenty of the fatal cases are recorded.

Ligature of the distal vessels has been several times performed and in some cases with temporary benefit.

Abdominal Aneurysm.—Aneurysms of the abdominal aorta occur more frequently at some parts of the vessel than at others. The most common site is just below the diaphragm (in 133 out of 177 cases collected by Sibson), at the origin of the cœliac axis which it often involves; they are more rare at the origin of the superior mesenteric artery, and are seldom found lower than this point.

Symptoms.—The first symptoms of abdominal aneurysm are generally painful feelings in various parts of the abdomen. These may be lancinating or paroxysmal, and may encircle the body like a girdle. They may be due to the sympathetic nerves being involved in the sac or to the connection of these nerves with the spinal, or the latter may be directly involved by the aneurysm eroding the vertebræ.

If the patient have an aneurysm in the upper part of the abdomen, it will be most usually found in the left hypochondrium, or if nearer the median line, to its left side. A distinct tumour may be felt, expanding under the hand, or pulsation only; this may be so great as to give considerable movement to the stethoscope when placed upon it. If not only an impulse but also an expansion be felt, the diagnosis of aneurysm is at once made. Sometimes also a thrill can be perceived. On auscultation, a systolic bruit will almost always be heard.

If the aneurysmal tumour be lower down, it can still more easily be grasped and its nature made out. If it grow backwards, it may also be detected in the loin, both by its pulsation and by the existence of a bruit; and this bruit cannot be produced by pressure of the stethoscope, as is frequently the case when it is heard in front, in a thin and anæmic subject. Various other symptoms may be present owing to the pressure of the sac. Thus occasionally actual obstruction of the intestine has occurred; the constipation, however, so frequently observed, probably arises indirectly. A very careful

examination of the femoral arteries will sometimes show a slight retardation of their beat as well as a diminished tension.

The mode of death in abdominal aneurysm is generally by rupture; this may occur directly into the abdomen causing instant death, or it may occur behind the peritoneum, whereby the blood becomes effused into the areolar tissue and a coagulum is formed for the time. This new or false sac soon, however, gives way with the same result as in the first instance. Very rarely and exceptionally the coagulum becomes fibrinous, and this being incorporated with the original aneurysmal sac, a cure is effected. Other exceptional terminations of abdominal aneurysm are sometimes met with, as in a patient in Guy's Hospital in whom a loud whizzing bruit was heard in the lower part of the back, and who also had dropsy of the lower extremities. After death a small aneurysm was found communicating with the vena cava so as to make an aneurysmal varix. The venous blood was retarded in its flow upwards and so the dropsy was produced.

Ætiology.—The causes of abdominal aneurysm are the same as in the thoracic form. Violent exertion is no doubt a very frequent cause, and this is the reason why it is so much more common in men than in women; for in women it is exceedingly rare. In several cases there has been well-marked syphilis, and no doubt can exist as to this being a frequent cause. Of three female cases in Guy's Hospital two had had syphilis; one of them occurred in 1853 under the late Dr Babington. She was a prostitute and was being treated for syphilis when she died suddenly from rupture of an abdominal aneurysm at the cœliac axis.

Diagnosis.—This is not always easy, since pulsating tumours may exist in the abdomen which are not aneurysmal, and bruits may be discovered in the abdominal aorta without disease of its coats. In women especially who are anæmic, frequent pulsations in the abdomen occur, and bruits are heard, which suggest aneurysms. Then, again, a tumour may be raised up by the aorta and pulsate, and in it a bruit may be heard, but these two signs are not sufficient to warrant the diagnosis of an aneurysm; nothing but the rhythmical expansion of the sac can afford a distinctive diagnosis. If the patient be placed on his hands and knees and the tumour be handled, any falling forwards would be in favour of a morbid growth, for an aneurysmal sac would still remain in its place.

Treatment.—The general treatment of abdominal aneurysm is the same as that of thoracic—restricted diet, absolute rest, and potassic iodide. Beside these methods, the opportunity sometimes occurs in abdominal aneurysms of using mechanical pressure as a local method. The plan has been most successfully carried out by Dr Murray, of Newcastle, in 1863, and has since been followed by others. He found that a very short period may be sufficient to cause coagulation in the sac. The aneurysm must of course be sufficiently low down. Unfortunately, the more common position at the cœliac axis is too high to allow room for any instrument being applied above the tumour. In Dr Murray's first case ('Med.-Chir. Trans.,' vol. xlvii, p. 187) pressure was used for two hours, and again for five. The aneurysm was rapidly cured, and the man remained well for six years afterwards. It was then found after death that the aneurysm came off from the aorta at the origin of the inferior mesenteric artery; this was obliterated and the circulation had been carried on through collateral branches. This case was followed by one of Dr Greenhow's, in which also one application was not sufficient; pressure was repeated again and again for a week and at last a

perfect cure resulted. Mr Durham reported a case about the same time of an abdominal aneurysm cured by pressure. This was kept upwith the patient under chloroform for ten hours until pulsation of the sac ceased. The circulation then also stopped in the femoral arteries and coldness of the legs came on. Pulsation again occurred in the aneurysm but in a less degree. The treatment was continued, but pulsation was not completely arrested in it for a month. Perfect recovery then followed. Another case occurred in Guy's Hospital where pressure was kept up for some hours on the distal side of a high aneurysm. No apparent effect upon it was produced; but collapse came on and in twenty-four hours the patient died of peritonitis. The intestine was found bruised and covered with lymph. The sac contained a coagulum which appeared as if it had been deposited during life. The bold plan has also been adopted of opening the abdomen, exposing the aneurysm, and inserting iron wire (Pringle and Morris, 'Med.-Chir. Trans.,' 1887).

Dissecting aneurysm.—In connection with aneurysm it is necessary to allude to the subject of rupture of the aorta owing to disease of its coats. It is rare for the whole of the coats to give way simultaneously; more usually the inner coat is lacerated; the blood then finds its way between the tunics and produces a dissecting aneurysm; finally the adventitia ruptures, and causes the patient's death. When the blood is effused in this manner, it tears asunder the layers of the middle coat, so that a part is found united with the intima and a part externally covered by the adventitia. Two cases may be mentioned in illustration of this remarkable form of lesion.

A former sister of Petersham Ward, about sixty years of age, was seized a month before her death with a violent pain across the chest and abdomen, her heart became quick and tumultuous, and it was thought at the time that she was dying. She, however, rallied, had some slight febrile symptoms, and in a few days was doing duty in her ward. Two days before her death she had another similar attack and again quickly recovered. On the day of her death she fell dead whilst dressing. The autopsy showed the pericardium to be full of blood which had proceeded from a rent at the commencement of the aorta. On the inside the rent was seen to be an inch long just above the valves, and through this blood had passed between the coats of the vessel; outside the external coat was ruptured, but this did not correspond to the internal opening. Some distance above this, and at the beginning of the transverse arch, was another fissure, an inch long and older in date; it was also transverse, had smooth edges connected by bands, and had quite healed. Proceeding from this was effused fibrin in the coats of the artery, which were separated from one another to the end of the abdominal aorta. The separation had occurred in the whole circumference of the vessel.

Another example was that of a gentleman about sixty years of age. He was a merchant, and after returning from the City and eating his dinner was seized with a severe pain in the chest. It continued all the evening until he went to bed. He was found afterwards dead at the side of his bed. The autopsy showed the pericardium full of blood, which proceeded from a fissure in the aorta. On examining the latter from within, a laceration of the vessel was seen to have occurred half an inch above the valves; it was transverse and ran almost completely round the vessel. Blood had passed through and separated the coats throughout the arch, the descending thoracic and the abdominal aorta. It had, however, taken only about two

thirds of the circumference. The clot was quite recent. The laceration in the outer coat was at right angles to the internal one and was an inch and a half long.

Dr Peacock, who thoroughly investigated the subject of dissecting aneurysm, speaks of a variety occasionally met with, of a chronic nature, in which a long time elapses between the rupture of the internal and external coats, so that a distinct pouch may form in the external coat like an ordinary aneurysm. This becomes in time lined by a smooth membrane.

Statistics.—During the ten years from 1875 to 1884 inclusive, there were ninety-two cases of death in Guy's Hospital from aneurysm of the aorta, or nearly 2 per cent. of the total autopsies. An abstract of the *post-mortem* records has been made by Mr Alfred Parkin, and the editor has added eight of his own cases either antecedent or subsequent to that period, so as to make the number a hundred.

As to the *sex* of the patients, 93 were men and only 7 were women.

As to their *age*, one patient was eighteen at the time of his death, 5 were between twenty and thirty, 29 were between thirty and forty, 33 between forty and fifty, 25 between fifty and sixty, 3 between sixty and seventy, one was seventy, and one (a woman) was eighty-three.

All the cases were of *saccular* aneurysm, but in no less than sixteen there were one or more other pouches, some of which were obsolete and solid, and in several others there was more or less diffused dilatation of the aorta. In three cases the inner coat only had ruptured and formed a *dissecting* aneurysm.

The part of the aorta affected was in 28 cases the ascending part before the origin of the innominate artery, in 55 cases it was some part of the arch, in 7 the descending thoracic aorta, in 2 the sac was just at the orifice in the diaphragm, and in 15 it was below that boundary, 11 above and 4 below the origin of the superior mesenteric artery. It is, however, often difficult to say precisely whether a bulky sac, with perhaps some diffused dilatation around it, should be reckoned to the arch or not.

In 47 cases there was rupture of the sac (including six in which it had opened into the pulmonary artery), in 52 the sac had not ruptured, and death was caused by pneumonia, bronchitis, or pulmonary œdema, by syncope, by embolism, or some independent disease.

Of the cases in which rupture occurred, the sac burst into the right pleura in 4, into the left in 7; into the trachea in 4, the left bronchus in 4, and the left lung in one; into the œsophagus in 4, and the posterior mediastinum in one; into the pericardium in 4, and into the left auricle in 1, beside the six above mentioned into the pulmonary artery. The abdominal aneurysms ruptured nine times into post-peritoneal tissues, once into the peritoneal cavity and once into the stomach. In the whole number of cases there was none of external rupture, although in two the threatening signs described above had appeared.

Intrathoracic New Growths.—The chief clinical interest of these growths arises from their liability to be mistaken for aneurysms, or the reverse; and therefore, though pathologically so different, they may be most conveniently considered here. From the same clinical point of view, the new growths that occur in the thoracic cavity may be divided into two groups: (1) some of them are seated in the mediastinal tissues, or at

least implicate the root of the lung on one or on both sides; (2) others affect only the substance of the lungs, perhaps reaching the pleural surface, but not interfering with their roots.

Mediastinal new growths.—These tumours differ in different cases in almost every particular: in their starting-point from one or another of the thoracic structures, in their histology, and in the various directions in which they may grow. It may therefore seem illogical to group them together under a single heading; but there is really no alternative, for no clinical distinction can be drawn between them.

As regards the *origin* of a mediastinal growth, it is seldom possible to come to any positive conclusion at the time of an autopsy, when it has generally reached a great size and involved various tissues. The common supposition is that they often start from the bronchial and other lymph-glands; and this is doubtless correct for cases in which the thoracic affection is only part of Hodgkin's disease or general lympho-sarcoma, as well as for those in which it is secondary to a growth situated in some other part of the body. But on more than one occasion the glands have been only partially attacked: some have been infiltrated with the new growth, but others, though embedded in it, have retained their natural structure. In such cases the origin may be assumed to be the thymus or the mediastinal connective tissue and fat, or perhaps the pericardium, or the periosteum of the sternum or vertebræ.

As for the *histology* of mediastinal tumours, the vast majority of them are made up principally or entirely of small round-cells, and are classified by some observers as lymphomata, by others as round-cell sarcomata. But some specimens contain a large proportion of spindle-cell tissue, others consist of little but fibrous tissue, and some have been described as having an alveolar structure, and even as being true carcinomata. In yet other cases mediastinal tumours have been found to be of a syphilitic nature, being formed of gummata embedded in a dense fibrous material. Lastly, some have been dermoid cysts, containing hair, bone, and even teeth, beside a quantity of fat.

Among the *relations* which mediastinal new growths bear to the various structures contained in the thoracic cavity, by far the most important are those that concern, on the one hand, the great systemic veins, and, on the other, the main air-passages. Indeed, it is almost a peculiarity of the affection now under consideration to produce obstruction either of the superior vena cava, or of one or other of the innominate veins, or of all three of them. In one case a tumour in the lower part of the thorax reduced the orifice of the inferior vena cava to a mere slit, through which the finger could scarcely be passed. As already observed, aortic aneurysms occasionally, though comparatively seldom, interfere with the veins; and almost the only other cases in which the flow of blood through the superior cava is retarded or prevented are certain cases of heart disease in which, during an acute attack of pericarditis, there has been inflammation of the mediastinum with thrombosis of one or both of the innominate veins, ending in obliteration of the affected vessels. But when there is a mediastinal new growth, venous obstruction is a very frequent result. Sometimes the growth penetrates the coats of the vena cava, and fungates within it as a soft smooth mass, which may be as large as a thumb. Sometimes it surrounds that vessel or one of the innominate veins, and causes extreme narrowing or even complete obliteration of the blood-channel. In either case there may be a consecutive thrombosis of the jugular and other tributary veins.

The *clinical effects* of these lesions are sometimes very marked. There may be great œdema of the arm and hand on one side or on both. The neck and the face may be enormously swollen and of a deep red or purple colour, with obvious over-distension of their veins. When the obstruction is limited to the superior cava a collateral channel for the passage of the blood may be afforded by the azygos vein, which becomes dilated. But when both innominate veins are blocked the intrathoracic vessels can do little towards carrying on the circulation, which then depends in great measure upon anastomoses between the superficial veins of the chest and back with those of the lower part of the trunk. The consequence is that the body becomes covered with dilated vessels, and may acquire a deep purple or claret colour. One can generally easily make out in which direction the blood has to flow, for when one of the veins is emptied by pressure along its course it fills far more rapidly from above downwards than from below upwards. Or passing a piece of string round the chest, one may see at once that the vessels above it remain full, whereas those below it become empty. But in many cases the appearance of the affected parts is in itself a sufficient indication. For it is a curious fact, doubtless dependent on the presence of valves in their interior, that obstructed veins are apt to become far more tortuous when the circulation through them is in the reverse, than when it is in the natural direction. Thus when the superior cava is blocked the veins may be zigzagged and varicose all over the chest and the upper part of the back, whereas those over the lower part of the body may be a little larger than natural, but almost straight. If there is obliteration of the inferior cava, on the other hand, the effect may be exactly the converse. Watson, in his sixty-third lecture, records and illustrates by diagrams two cases in which this distinction was very manifest. A man who died in Guy's Hospital of a mediastinal new growth in 1868, said that the first indication that anything was wrong with him was that a sensation of swimming in the head came on when he stooped.

Obstruction of the lower air-passages may be the effect of compression of the trachea or of the bronchi by mediastinal growths. The records of necropsies at Guy's Hospital contain very few cases in which these parts are stated to have been found entirely untouched by the disease at the time of death. But sometimes it is noted that although their walls, even to the mucous membrane, were completely infiltrated, there was yet no narrowing of their calibre. In most cases the growth extends along the bronchi into the pulmonary tissue. Sometimes it fills up a great part of the lung, forming large masses in its interior and even reaching its surface. For obvious reasons the disease is then either confined to one side, or at least far more marked on one side than on the other. Dr Powell, in 'Reynolds' System,' says that the left lung is invaded more often than the right, but among twenty-six cases taken from the records of autopsies at Guy's Hospital the numbers on each side are nearly equal.

The *physical signs* caused by mediastinal new growths are due chiefly to their size, and to their interfering with the air-passages and with the lungs. Their bulk is sometimes enormous. In one case there was a solid mass that weighed ten pounds, and another in which the measurements in three dimensions were ten inches, seven inches, and five inches respectively. Such great tumours naturally cause considerable enlargement of that side of the chest which they principally occupy. The intercostal spaces may be widened and flattened; the movements of the ribs may be much impaired. But, on

the other hand, if the growth is attended with shrinking of the lung, the measurements may be less than those of the opposite side. In some cases the tumour protrudes above the clavicle, so as to be felt at the root of the neck; in others it bulges through one or more of the intercostal spaces. It may lead to some absorption of bone, but it does not appear as a rounded swelling, projecting far beyond the natural level of the ribs, as an aneurysm does. It may, however, pulsate more or less forcibly, and a systolic murmur may be transmitted through it from the heart or the aorta.

As may be imagined, such large growths as these cause great dulness on percussion. This is commonly very marked over the sternum, and for a greater or less distance on each side of it. But it may also extend over the whole of the front of the chest on one side up to the clavicle, or over the back to a variable distance round one scapula, or between both scapulæ, or even over the whole of one half of the chest from apex to base, including the axillary region. Not only is the percussion-sound absolutely toneless, but the sense of resistance to the finger may be extreme. The condition may therefore be exactly like that which would be produced by liquid effusion into the pleural cavity; and as a matter of fact such an error of diagnosis has been often committed, and repeated exploratory punctures have been made, which of course have led to no good result. The best way to avoid this blunder is to map out carefully the area of dulness in all directions, especially over the middle of the chest in front, where it may be found to extend beyond the bounds to which it would certainly be limited were it due to pleural fluid. But the difficulty is increased by the fact that when the pleura is reached by a new growth, effusion as a rule follows, and sometimes in large quantity. Hence the success of a paracentesis is no warrant for resting satisfied with the diagnosis that there was liquid in the serous cavity; that may be merely a complication of a far more grave disease. A circumstance which should excite suspicion is the presence of altered blood in the liquid, giving to it a dark brown tint. But in some cases of mediastinal tumour the pleural effusion is straw coloured, and exactly like that seen under other conditions.

On the other hand, there are cases, especially where the growth is limited to the root of one or both of the lungs, in which percussion yields altogether negative results.

The other physical signs of mediastinal tumours are very uncertain. Tactile vibration is sometimes increased, but more often lessened or abolished. It is, of course, only in the latter case that the disease is likely to be mistaken for pleural effusion. There may be more or less loud bronchial breathing, or only a very faint vesicular murmur may be audible with the stethoscope when the patient inspires, or there may be absolute silence. The stridulous and other sounds produced by narrowing of the lower air-passages will be described in the second volume. In many cases there are râles, of varying quality, not over the tumour itself, but over parts of the lung to which pass the branches of an obstructed bronchus. To these may be added the signs of consolidation, when inflammation of pulmonary tissue sets in.

In many cases mediastinal growths invade the pericardium, generally at the base of the heart; and they may then spread downwards to a greater or less extent, both along the parietal layer of the serous membrane and also in the walls of one or both of the auricles. The pericardial sac may either become closed by adhesions, or it may become distended with liquid effusion, which is often sanguineous, or it may be affected with inflammation, leading

to the exudation of lymph or of lymph and pus. Of the great vessels, the aorta almost always seems to escape entirely, the venæ cavæ (at least the vena cava superior) are very often greatly narrowed, as has already been pointed out; the main divisions of the pulmonary artery and of the pulmonary veins are also in many instances pressed upon, so that their calibre is much reduced. At first sight it might appear probable that cases in which the pulmonary vessels are involved should be characterised by more intense dyspnœa than would otherwise be present. But in practice this is not observed, nor ought it perhaps to be expected, inasmuch as Lichtheim has shown by experiment that one fourth of the natural calibre of the pulmonary artery suffices to keep the lungs fully supplied with blood. Even when the heart is not invaded by a mediastinal growth, it is often much displaced; generally it is pushed downwards and to the left, but sometimes, when there is shrinking and contraction of the left lung, it is dragged up so as to be felt pulsating not far below the clavicle. Although the aorta itself resists the pressure of the growth, it may happen that some of its branches are more or less narrowed, causing the radial pulse to be weaker and smaller on one side than on the other.

The œsophagus does not seem to be often occluded by mediastinal growths; at least in the reports of *post-mortem* examinations at Guy's Hospital there are very few instances in which this is said to have been the case, or in which dysphagia is noted as having been among the symptoms. One patient is reported to have brought up his food almost directly after attempting to swallow it; at the autopsy the œsophagus was found pushed aside, but not invaded. In some rare instances the disease extends into the spinal canal, causing a variety of "compression-paraplegia."

One very curious effect of a mediastinal growth which was observed many years ago, in Mr Cooper Forster's ward, was apparently due to pressure upon the vaso-motor nerves. A woman, aged twenty-six, was admitted, in the summer of 1866, for a defective state of the circulation in her fingers, which were blue, cold, shrunken, and also very painful and tender. After a few weeks she was discharged, but she again came in under Dr Moxon in the following April, and died some months later. At the autopsy it was found that a growth infiltrated the fibrous tissue in front of the spine, and involved both the first dorsal nerve and the sympathetic trunk. Nothing abnormal was ever detected in the state of the pupils. But in other cases in which there is pressure upon the sympathetic trunk on one side at the root of the neck, the corresponding pupil is smaller than the opposite one, epecially when but little light reaches the eyes, so that both pupils should normally be dilated. The explanation is that when the sympathetic is paralysed, the natural balance is lost and the contracting fibres of the third nerve get the upper hand. In three cases of mediastinal growth, Rossbach is said to have further observed that both pupils dilated regularly with each inspiration; in two of these cases, in which there was a swelling above the clavicle, pressure upon this tumour caused the pupils to become widely dilated, while the frequency of the pulse also underwent a temporary alteration, being retarded in one instance and quickened in the other.

The other symptoms of mediastinal growths vary widely in different cases. There is generally more or less dyspnœa from the time when the patient first notices that anything is amiss with him. Very often he is obliged to sit up, even at night; sometimes the only position in which he

can sleep is leaning forwards, or even lying on his face. The breathing is generally quickened to twenty-four to thirty in the minute. Pain is commonly an early symptom, and sometimes (though not usually) it is very severe. It may be referred either to the side, or to the shoulder, or to the middle of the chest in front. Sir Risdon Bennett speaks of it as sometimes sudden and transitory, and attendant on physical exertion.* Most patients have a troublesome cough, which is often described as "ineffectual," giving rise to little or no expectoration. But sometimes a viscid mucus is brought up, and occasionally this contains blood so intimately mixed with it as to give it the appearance of red-currant jelly, a sign to which writers attach diagnostic importance. The spitting of pure blood, too, is not uncommon, and it may occur at the very commencement of the clinical history of the case, and more than a year before the fatal termination. Profuse hæmoptysis is seldom observed, but Dr Church has recorded in the 'Pathological Transactions' (vol. xix, p. 64) one instance in which four pints of blood were brought up immediately before death, though the bleeding probably came from broken-down lung tissue in the neighbourhood of the growth, rather than from the growth itself.

Cachexia is by no means a prominent symptom. The patient often looks well and ruddy for some time after he comes under treatment, and even at the last there is not often extreme emaciation. Pyrexia is generally absent, but Risdon Bennett relates a case (loc. cit., p. 121) in which the temperature had varied from 100° to 101·4°, but in which Dr Sutton, who made the autopsy, could detect no appreciable inflammatory changes, so that the only way of accounting for the febrile disturbance was by referring it to the active cell growth that had been going on, not only in the lungs, but also in other organs of the body.

To complete this account of the occasional symptoms of mediastinal tumours, it may be noted that in two cases of intrathoracic dermoid cysts the patient has expectorated hairs, in one of them for as long as twelve years before death.

Occasionally, however, when there is a mediastinal growth, no symptoms are present at any period of the case, which is cut short by disease of some other part of the body, most frequently by a similar tumour of the brain. Several instances of this kind have occurred at Guy's Hospital, and they are of interest, not only in themselves, but also as showing how little dependence can be placed upon the duration of chest symptoms, as an indication of the real rate of progress of the disease. On the other hand it must not be forgotten that even if we did theoretically know the time necessary for a growth to develop itself from its very commencement until it destroys life, we should still be unable, so long as we cannot fix the date at which it begins, to make any use of the knowledge in clinical practice. In nine cases at Guy's Hospital in which the duration of the symptoms was noted, it varied from two and a half to eleven months. The extremes in each direction are probably afforded by two cases cited by Hertz, in 'Ziemssen's Handbuch;' one which proved fatal in a week from the first appearance of symptoms; the other in which life was prolonged for at least seven, and possibly for fifteen years.

* Lumleian Lectures on "Intrathoracic Growths," p. 179. This refers rather to a sudden effort or injury producing a pain, to which the patient refers as his earliest symptom. The same author says, with reference to the later symptoms:—"The amount of mere pain is seldom such as to call for the use of any large quantity of opium. But the distress is often very great" (p. 186).

Diagnosis.—It will have been gathered from the last section how difficult, and sometimes impossible, it may be to recognise the existence of mediastinal tumours. In most cases, however, the physical signs point to a mass within the thorax, not pneumonic or tubercular, and distinct from, though often complicated by, pleuritic effusion. The diagnosis is then generally between new growth and aneurysm. In the absence of pulsation we depend on probabilities. Severe pain, absence of pyrexia, and the fact of the patient being a man and having been laboriously occupied, incline to aneurysm. Insidious origin, œdema of one arm, effusion into the chest, and enlarged cervical or axillary glands, together with the fact of the patient being a woman, and particularly a young woman, or a child, point decidedly to the diagnosis of tumour.

It may be laid down that, while in young subjects pleuritic effusion should make one suspect tubercle, in older subjects we should consider the probability first of Bright's disease, and then of lymphoma or sarcoma of the lung.

Ætiology.—With regard to the *causes* of mediastinal growths very little is known. In one or two recorded instances they have been attributed to injuries, such as blows upon the sternum; but it may well be doubted whether this is more than a coincidence. A remarkable contribution to the ætiology of the disease is a paper by Hesse in the 'Archiv der Heilkunde' for 1878, where it is stated that in the mines of the Schneeberg 75 per cent. of all the miners—from twenty-one to twenty-four each year—die, generally about the age of forty, from "cancer of the lungs," spreading from the root. Professor Ernst Wagner examined some specimens of the disease and found that the growth is a lympho-sarcoma. Only two explanations appear possible; one that it is the result of a tendency inherited and transmitted from generation to generation; the other, that it depends upon the nature of the minerals worked in the mines, which contain bismuth, cobalt, and nickel, with some arsenic and sulphur. With regard to the first suggestion it is noteworthy that the miners of the Schneeberg are recruited from among the sons of former miners; whether intermarriages are frequent does not appear. Dr Walshe mentions the cases of two brothers who were each affected with intrathoracic growths, and a parallel instance will presently be mentioned.

Most observers say that more men than women die of mediastinal growths, and this is confirmed by a collection of thirty-three cases from the *post-mortem* records of Guy's Hospital; the proportion being more than two to one. As regards age, I find that the numbers for each decennial period from twenty to sixty are almost exactly the same; whereas, it is generally stated that the disease is more frequent in persons between twenty and thirty than in those who are older. A few cases have been observed in children.

The *prognosis* in cases of mediastinal new growth is very grave. If recovery should take place in a case diagnosed as one of this disease, the general impression would be that a mistake had been made, and that the patient was really affected either with some inflammatory or syphilitic thickening of the intrathoracic structures, or else with aortic aneurysm. But we had one instance which goes some way towards establishing a different conclusion. It is that of a man named John Bullions, who was admitted into Guy's Hospital under Dr Habershon on February 1st, 1867, with loss of voice, stridulous breathing, great swelling of the neck, œdema

of the chest, and fulness of the veins. There was also slight deficiency of resonance on percussion over the right apex and over the root of the right lung behind. Under the administration of iodide of potassium he rapidly improved, and left the hospital on March 2nd, after which he returned to work. But on May 22nd he was readmitted with what was apparently an attack of erysipelas of the face and neck. This also quickly subsided, and from that time I lost sight of him. However, in 1871, another man, named Thomas Bullions, aged nineteen, came into the hospital and died of a mediastinal new growth, as was proved by the autopsy. Struck by his peculiar name, we inquired and found that the former patient was his elder brother, and was then in good health, though still rather short of breath. Were both cases of the same nature, like those recorded in two brothers by Dr Walshe, or were they different?

In the *treatment* of this disease a faint hope of cure is by the administration of iodide of potassium, arsenic, or mercury. When there is great venous obstruction marked relief is often afforded by venæsection, cupping, or leeches. To ease pain recourse may be had to blisters or mustard poultices, and also to the various anodynes; for the cough, Risdon Bennett recommends antimony in small doses with a sedative.

Pulmonary new growths.—Only those cases remain to be mentioned in which the lung is affected with a new growth that leaves its root free. Clinically they differ widely from the "mediastinal" cases just described. Very often, indeed, they give rise to no symptoms at all, and are almost always secondary. The position of the pulmonary in relation to the systemic circulation causes the lungs to be the most natural seat for all forms of secondary growth whenever infection takes place by the blood-current, excepting in those cases in which the primary tumour lies within the area of the portal system of vessels. But in very many instances the patient, up to the time of his death, shows no indication of anything more than a general cachexia, and it is only at the autopsy that the lungs are found studded with nodules or masses of new growth. On the other hand, there are some cases in which cough and dyspnœa, or hæmoptysis, draw attention to the state of the lungs; and in which there are physical signs of the exact position of one or more tumours, beside evidence of fluid effusion into one or both of the pleural cavities.

In most of these cases the breast or the stomach or some other part is known to be affected with a primary malignant growth. But the analogy of other viscera in which secondary tumours are apt to appear, such as the liver and the brain, would lead one to expect what occasionally does occur; a patient dies of the effects of growths in the lungs, and it is discovered for the first time at the autopsy that these growths were secondary to some primary growth in a distant organ. Such an occurrence, however, appears to be very rare. Once a young man came into Guy's Hospital under Dr Frederick Taylor with what appeared to be acute bronchitis, and died in a few days. At the *post-mortem* examination it was found that the lungs were full of sarcomatous masses, secondary to a like affection of the testis.

As to *primary* growths in the lung, they are extremely rare, and even less can be said about them than about secondary growths. Cases in which there is only a single mass of considerable size, or in which one among several masses is obviously older than the rest, have often been described as

examples of primary malignant tumour in the organ. But it is uncertain whether the other viscera, and especially the different mucous membranes, have always been searched with sufficient care to justify the conclusion. The same doubt applies to cases which have been recorded under the name of disseminated or miliary cancer of the lungs. One such was brought by the author before the Pathological Society in 1866. The patient was a man aged fifty, who died of an illness of two or three months' duration, but only two days after being admitted into hospital, with what appeared to be capillary bronchitis complicated with some pneumonia. At the autopsy the lungs were found full of round bodies like tubercles, but larger (some as large as hemp-seeds), and of a shining, white appearance. The only growths discovered elsewhere were a few in the heart and in the liver. A somewhat similar case, in a girl of seventeen, is related by Risdon Bennett; but the liver in that instance contained several very large tumours, so that the lung affection was clearly secondary.

A still more remarkable form of disease is one of which an instance occurred in 1870. A man, aged thirty-six, died in Guy's Hospital after two and a half months' illness, which was attributed to damp and cold, and which appeared clinically to have been pneumonia of the right lung, accompanied with much effusion into the pleura. At the autopsy the lung was found much enlarged, nearly white in colour, but somewhat mottled, smooth, and shining in appearance, soft and cushiony to the feel, so that one might have imagined it to be generally emphysematous but for the fact that it was absolutely airless, every part of it sinking instantly when put into water. At the root of the lung there was obvious new growth, which probably was seated in the glands, but which had also involved the superior vena cava, and narrowed it considerably. Unfortunately, the lung was thrown away before any microscopical examination of it was made. But probably it was an example of a primary diffused carcinoma of the pulmonary tissue, such as is alluded to by Hertz (in 'Ziemssen's Handbuch') as "bearing a striking resemblance to grey hepatisation."

A remarkable case of multiple sarcoma of the lungs, chiefly affecting the surface, but not causing pleurisy, occurred in a woman aged sixty four, who died in Miriam Ward, November 29th, 1886. There were malignant growths in the vertebræ, liver, and kidney, and a mass of diseased glands in the neck. These were taken for tubercular glands and the pulmonary symptoms ascribed to phthisis by the editor, under whose care she was for a day before her death: there was considerable caseation of the bronchial lymph-glands; but the true nature of the case was chronic lympho-sarcoma, lasting at least eighteen months, and then becoming generalised. One point worthy of note was a rise of temperature from 99° to 105·8° F.

Thrombosis and Embolism.—Thrombosis implies the spontaneous coagulation of the blood in a blood-vessel during life, and embolism the carrying away of a clot detached to a distant vessel, which thus becomes blocked.

As regards thrombosis, coagulation may occur under various conditions and from numerous causes. Anything which tends to render the blood stagnant will favour its coagulation, and afterwards certain changes take place in it; but if the blood be flowing during the formation of a thrombus, then the fibrin is deposited, portion by portion, until a white fibrinous mass fills the vessel. The principal cause of coagulation is some change in the interior of the vessel; whilst this is smooth and in a state of vital integrity

the blood remains fluid, but as soon as the endothelium is in any way altered in structure, or any part of it removed, or cretaceous material be formed upon it, coagulation will take place. The effect of retardation of the blood-current is seen in the case of coagulation in the iliac veins of young persons dying of phthisis and other wasting disorders, and the effects of disease in thrombosis of the vessels of the aged brain. There are no doubt other causes which favour coagulation inherent in the blood itself. For example, coagulation of the blood leading to thrombosis is often met with in diabetes, gout, typhoid, the puerperal state, and other disorders. In these cases coagulation occurs in a small vein, and then proceeds upwards, sometimes reaching to the vena cava.

The clot may set up an irritation or inflammation of the lining of the vessel, so that there is soon associated with the coagulum an endarteritis or endophlebitis. The clot then becomes organised and the vessel obliterated. Occasionally it may shrink and allow a passage of blood through it again. In other cases the clot softens and is changed into a milky or pus-like material.

Blood-vessels may become independently inflamed. Thus the intima of an artery undergoes inflammatory processes just like those of the endocardium, leucocytes may be seen infiltrating its walls, and finally fibroid patches form on the surface, which may terminate in sclerosis and calcification, or the vessel become merely thickened. Veins in connection with local inflammations may suppurate, as is often seen in the jugular in cases of disease of the temporal bone or in the portal in abscess of the liver.

Cases of thrombosis become most important when the coagulation reaches the iliacs or vena cava, for then, should a portion of the clot become detached and carried to the heart and so on to the pulmonary artery, almost sudden death ensues. The most common and best marked example of this kind occurs in puerperal cases, when a clot has formed at the commencement of the vena cava by extension from the iliac veins. Some inflammatory process having occurred about the neck of the womb, coagulation takes place in the uterine sinuses and extends through the iliac veins to the inferior cava. This may have occurred with so very few symptoms that no suspicion of the coming fatal event is excited. It is usually about a fortnight after delivery, on the patient's rising from bed, that a portion of the fibrinous clot becomes detached, passes into the pulmonary artery, and causes almost instant death. If the clot should be small and plug only one pulmonary artery or a branch, then death may not occur so speedily, but be postponed for a hour or two. In other cases the detached portions are still smaller, and not being large enough to close the vessel entirely, they stick in some of the branches, and there form a focus for further coagulation. This proceeds until the whole lung is blocked, and a fatal termination may be delayed for a few days. Under these circumstances it is possible, though very rare, for the patient to recover.

With the exception of the case of pulmonary embolism just mentioned, the most important and frequent forms of embolism are those which are met with on the arterial side of the system. A clot may form, for example, in a large artery, and portions of this may be carried into the vessels beyond, or, what is more common, vegetations become detached from the valves of the heart and are then carried into the arterioles of various parts of the body. For example, there is the sudden blocking of the middle cerebral artery, a very common cause of hemiplegia, or of the vessels of the limbs leading to gangrene. As examples of the latter result: a boy under treat-

ment for heart disease was suddenly seized with pain in the arm, which on examination was found to be pulseless; a man was attacked without warning by most excruciating pain in the leg, and the limb was found cold and with no pulsation in the arteries. It is remarkable that blood-vessels, which are regarded as insensible organs, should evince the most acute sensibility when subject to such stretching as occurs in embolism.

One of the subsequent events which occasionally takes place in the vessels so blocked is the formation of aneurysm (*vide supra*, p. 989).

When smaller fibrinous particles are detached and carried by the circulation to all parts of the body, as in the so-called ulcerative endocarditis, local effects are not so manifest, but the blood becomes infected, symptoms of a typhoid or pyæmic character are set up, and the malady, if fatal, may be protracted through several weeks. In these cases the arterioles in various organs become blocked, leading to very characteristic formations in the organs, known as "infarctions." The vessel having become blocked and the blood flowing back into the emptied tissue, coagulation occurs in these arterioles; and if exudation takes place from them, a mass resembling a portion of fibrin is formed. Such "wedged-shaped masses" are met with at the edges of the lung, kidney, or spleen, in the midst of which the remains of the tissue of the organ may still be seen. These may soften and so lead to further infection; or they may dry up and leave a cicatrix; or in the case of the lung they may become a focus for an inflammatory process.

The origin and pathology of these cases of ulcerative endocarditis have been already described (*vide supra*, p. 952), and instances of sudden death from pulmonary embolism need no illustration, but cases of slow coagulation in the lungs with more lingering symptoms are not so common, and therefore an example may be given.

A medical man, fifty years of age, was seized on the 3rd of March with difficulty of breathing, and on the following day he was carefully examined by Dr Wilks. His shortness of breath was of that kind so frequently seen in cardiac disease; the patient was panting and breathless, the respiration was very quick and on the slightest exertion it almost ceased, as happened more than once when he attempted to move out of bed. On examination of the chest nothing abnormal could be discovered; the sounds of the heart were healthy, its action quiet, and the pulse was 80. The patient had no difficulty in taking a very full breath, indeed he felt, he said, as if he must breathe too much. A suspicion of embolism caused an examination of the whole body, when it was found that he had had phlebitis of one leg, arising from an injury six months before. The patient also said that a month ago he was seized with shortness of breath, which passed off after a few hours. Dr Wilks concluded from this that he had embolism of the pulmonary artery. On the following day he was no better, and on March 6th the author saw him, but could discover no disease in the chest from the existence of any physical signs. On March 7th he was worse and was beginning to spit up tenacious, rusty, and bloody mucus. On March 8th he was dying, gasping for breath and with great lividity of the extremities, but the heart was regular and there was no bruit. The autopsy showed embolism of both branches of the pulmonary artery. In the left was a large clot, tolerably recent, and in the right an older one adherent to the walls of the vessel. There was also pneumonic consolidation in portions of both lungs. The right femoral vein contained a thrombus of exactly the same character as that found in the pulmonary artery.

Cases of recovery from pulmonary embolism are so rare that it may be also advisable to give an illustration of it.

A young man, an officer in the army, was operated on for varicocele; he rapidly recovered from the operation, and had left his bed, but had not gone out of the house. During conversation one evening, February 9th, a fortnight after the operation, he suddenly called out, fell back, and gasped for breath. It was thought he was dying. A medical man saw him soon afterwards and found him cold, pulseless, and breathing heavily. About an hour afterwards he spoke; he was very pale, and the respiration was sighing, but it was thought that his pulse was perceptible. He lay all night quiet and calm, but pale, and the pulse was only just to be felt. On the following morning he had rallied, his skin had become warm, the breathing was tranquil, and the pulse 100. February 11th.—Slight oppression of breathing with little pain; temperature normal. February 12th.—Slight crepitation heard over lower part of chest on left side. Heart normal, no bruit. February 13th.—Lying quietly, fills chest well and deeply. Crepitation over lower part of left lung in front and expectoration of a little bloody mucus. A slight murmur heard at base of heart. February 14th.—Bruit more audible and traced up in course of pulmonary artery. February 16th.—Bruit less marked and crepitation of lung less. February 20th.—Bruit gone and no râles heard, but in the spot where they existed imperfect respiration. The expectorated mucus was glairy, interspersed with streaks of blood. He continued to improve, but still spat up red, glairy, transparent mucus, and when he sat up, the pulmonary bruit became audible. March 15th.—Allowed to leave his bed, all physical signs having disappeared; but he still spits a little coloured mucus. He was kept quiet for another month, when he was allowed to go out, and gradually recovered.*

EXOPHTHALMIC GOITRE.†—In the second quarter of the present century several observers noted the concurrence of palpitation of the heart with staring eyeballs and an enlarged thyroid body. In Germany such cases are commonly spoken of as examples of "Basedow's disease," because v. Basedow in 1840 published a paper on the subject. It had, however, already been described by Graves, of Dublin, in his 'Lectures' (originally published in 1835, according to Stokes, who himself recorded one of the earliest cases); and therefore there is warrant for calling it "Graves's disease," as was proposed by Trousseau. Still earlier allusions to it have been found in the writings of Adelmann, and of Caleb Parry, of Bath (1825), but the references of Flajani (1798) are not to this affection at all according to Dr Beigell ('Reynolds' System,' vol. v), who shows that Graves first described a typical case and saw its clinical significance.

Origin.—The complaint generally sets in very gradually; as a rule no definite cause can be found for it. But sometimes it has been traceable to a severe mental shock, and writers say that it may then develop itself rapidly. Trousseau speaks of a lady who, having one night been crying for a long time on account of her father's death, "suddenly felt her eyes swell and lift up her eyelids," while at the same time she had copious epistaxis, violent

* Two cases of recovery from pulmonary embolism have come under the Editor's notice; one in a patient seen with Mr Hine, of Oxford, where it followed thrombosis of the femoral vein from an injury; the second, during recovery from pneumonia, in a healthy man, under the care of Dr J. H. Galton. In both cases the condition was early recognised, and in both the diagnosis was amply confirmed.

† *Synonyms.*—Maladie de Graves—Basedow'sche Krankheit—Cachexia exophthalmica.

palpitation of the heart, and throbbing and enlargement of the thyroid; four days later the nature of the case was recognised.

Graves's disease occurs chiefly in young women, but a patient who died of it at Guy's Hospital in 1868 was a woman of fifty-eight, and Stokes saw it in a lady upwards of sixty years of age. It is said to have been observed in a boy of fourteen, in a girl of seven, and in another girl only two and a half years old. The proportion of females to males is stated by von Graefe as six to one, by Eulenburg (in 'Ziemssen's Handbuch') as at least two to one; but Trousseau's cases give it as fifty to eight, Henoch's as twenty-three to four, and Praël's as twenty-eight to one.

Persons who are anæmic or chlorotic are said to be especially liable to exophthalmic goitre; and Stokes mentions the case of a man in whom long-continued bleeding from piles apparently caused it. It also frequently occurs in hysterical women, and in neurotic subjects, including epileptic patients and lunatics. An instance is recorded of it in a boy, aged eight, whose mother is said to have had the same disease. And a man, aged twenty-six, whose portrait was given by Dr Wilks in the 'Guy's Hospital Reports' for 1870, had an uncle and a brother who were affected with ordinary goitre. The disease is never endemic, but the vascular distension of the thyroid sometimes ends in solid overgrowth.

Symptoms.—Of the three cardinal symptoms, the first to be observed is commonly an *increased action of the heart.* This may for a time be only occasional, but afterwards it is constant. The number of beats is much augmented, reaching 120, 140, or even (it is said) 200 in the minute. The cardiac impulse is exaggerated, the sounds are loud and ringing, and a blowing systolic murmur may be audible with the stethoscope at the base or, less often, at the apex. The carotid arteries throb, and the hand feels the pulsation of the enlarged thyroid, often with a well-marked thrill. The beats of the radial arteries, however, are not unduly forcible. Trousseau, in opposition to Aran, makes it a point that there is no increase in the area of absolute cardiac dulness, and the author can confirm his statement from personal observations. This would not of itself prove that the heart is not enlarged. But in the fatal cases that have occurred at Guy's Hospital, the organ has weighed only nine, ten, or once perhaps eleven ounces. It is true that each patient was much emaciated, so that a degree of relative hypertrophy may be said to have been present. There is no cardiac bruit to be heard in uncomplicated cases, but when anæmia is considerable a systolic murmur may be sometimes audible in the subclavian or the pulmonary artery. Atheroma of the aorta has been occasionally seen as an effect of the increased strain upon that vessel.

Swelling of the thyroid body may either be observed before the exophthalmos, or not until after that symptom has attracted attention. It is sometimes symmetrical, sometimes more marked on one side, and usually on the right. In some cases it is but slight, so as hardly to deserve the name of goitre, and it never approaches in size to the very large tumours which sometimes occur in the endemic form of bronchocele. But it may cause a considerable projection of the throat, and it may press on the trachea, so as to alter the voice and to compel the patient to lie with the head thrown back upon a pillow. In Dr Wilks's case, already referred to, the lower part of the left lobe was found after death to extend down into the chest, altering the shape of the trachea, and perhaps compressing the thoracic duct. In an early stage of the disease the thyroid body sometimes undergoes rapid

variations in size, becoming larger when the heart's action is more disturbed, or even under emotional excitement. This of itself shows that the state of the gland is partly one of vascular turgescence, and its vessels have been found increased in diameter, the arteries especially being tortuous. Its tissue may be perfectly normal in appearance, or it may contain "colloid cysts," some of which may be of considerable size.

Prominence of the eyeballs varies greatly in degree; one patient merely seems to stare a little more than natural, another has a fierce expression, from a wide space left between the corneal margin and the edges of the eyelids. It is said that the points of insertion of the recti muscles into the sclerotic may be visible; and Trousseau alludes to a case in which one of the eyes "actually came out of the orbit, and had to be pushed back by the fingers." Sometimes the protrusion is more marked or begins earlier on one side than on the other; and according to certain observers it may be permanently unilateral. A point to which von Graefe attached considerable diagnostic importance is that, when the patient looks towards his feet, the upper eyelid fails to descend by an associated action, as it normally should do. He showed that this is by no means a necessary consequence of the exophthalmos; for when a tumour of the orbit causes the eye to project, the movements of the eyelid remain unimpaired. In one patient whom he saw, and whose complaint was of palpitation of the heart, the symptom in question constituted the sole ground for regarding the case as one of Basedow's disease. On the other hand, Eulenburg says that he has found "von Graefe's symptom" absent, or almost absent, when there was great protrusion of the eyeballs. In cases in which there is a high degree of exophthalmos, the eyelids may fail to meet during sleep; the cornea is then apt to become inflamed and even to slough. As a rule, patients affected with exophthalmic goitre see perfectly well, but sometimes they notice muscæ, or complain of fatigue in using the eyes. The only ophthalmoscopic appearance is said to be a dilated and tortuous state of the retinal veins. The pupils have been described as dilated, but Eulenburg says that von Graefe failed to observe this in an experience extending over nearly 200 cases, and he suggests that when it has been present it has been accidental and due to myopia.

There appears to be still a doubt as to whether the protrusion of the eyeballs is entirely due to turgescence of vessels in the orbit, or partly to an overgrowth or swelling of the fat. A third hypothesis is that it may in part be caused by contraction of Müller's non-striated orbital muscle. Exophthalmos has sometimes been noticed to increase or diminish as palpitation became more or less severe, and to vary with the menstrual periods. In some cases, but not always, the eyes are scarcely if at all prominent after death. The recti muscles have twice been found in a state of fatty degeneration, but this is attributed to disuse or to the stretching which they had undergone.

Other symptoms which have been noticed in patients suffering from exophthalmic goitre are irritability of temper, sleeplessness, headache, impairment of memory, and unfitness for employment. In some cases mental disturbance is associated with Graves's disease (see Dr Savage's paper in the 'Guy's Hospital Reports' for 1882); in others, flatulence and constipation, anæmia, amenorrhœa, leucorrhœa, or epistaxis. Irregular febrile attacks sometimes occur, in which the temperature may rise 2° or 3° F. There is often extreme emaciation. Trousseau notes that he has obtained a *tache cérébrale*. The spleen may be swollen, as ascertained during life. Enlargement of the

breasts has been mentioned, but Trousseau speaks of them as undergoing atrophy. It is perhaps worthy of note that in two fatal cases at Guy's Hospital, the patients being respectively twenty-nine and twenty-one years old, the thymus was persistent. In one it was four inches long, and had a maximum thickness of three quarters of an inch. Melanodermia and leucodermia have been observed by Trousseau and others (see Dr Drummond's lecture, 'British Medical Journal,' May 14th, 1887).

Pathology.—We need not discuss at length the many speculative views that have been advanced to account for the phenomena of exophthalmic goitre. That no one of the three cardinal symptoms can be taken as the cause of the others is now generally admitted. Some of the earlier writers on the subject held that some cardiac affection constituted the starting-point of the disease. Stokes, for instance, believed that this was usually a persistent functional excitement of the heart; and he cited Dr Parry's cases as showing that organic lesions might occasionally be followed by similar effects. Moreover, in two fatal cases at Guy's Hospital there had been an antecedent attack of rheumatic fever, and in one of them pericarditis and endocarditis were found at the autopsy. But in the great majority of cases the heart is perfectly sound. On the other hand, Koeben imagined that the goitre gave rise to the rest of the symptoms by pressing upon the cervical sympathetic; but such a notion is altogether inconsistent with the fact that enlargement of the thyroid may follow the exophthalmos; moreover, much larger endemic bronchoceles produce no such consequences.

There seems to be no escape from the conclusion that the three classical phenomena of the disease are joint effects of some other cause; and a very obvious suggestion is that of Trousseau, according to which they are due to disturbance of the lower cervical ganglia of the sympathetic. Eulenburg cites eight autopsies, in each of which changes in their structure were demonstrated; and Dr Goodhart's case in the 'Path. Trans.' for 1874 may be added to them, as also Dr Shingleton Smith's ('Med. Times and Gazette, 1878). The changes observed consisted generally in an overgrowth of the fibrous capsules of the ganglia, with or without an excess of the connective tissue in their interior, rendering them hard and tough. And it is perhaps worthy of notice that, in Dr Goodhart's case, the connective tissue of the neck and thorax also appeared to be in excess. In a few cases it is stated that there was atrophy of the nervous elements of the ganglia; but this may have been the result of reagents. On the other hand, there are two cases —one of them investigated with great care by Ranvier—in which the ganglia were found healthy. The superior cervical ganglion has been recently investigated by Dr Hale White, who finds that the variations in the size and pigmentation of the corpuscles, and the amount and density of the interstitial tissue, does not vary more than in cases taken at random.

In another case of Graves's disease in a lunatic, Dr Savage on careful microscopical examination of the cervical ganglia found them perfectly healthy ('Insanity and Allied Neuroses," p. 415). After death the heart is found normal both in the valves and the muscular tissue, and the thyroid much reduced in size, while the eyes have usually returned to their normal position, so that a case might easily fail to be recognised. Sir Henry Marsh, Basedow, and Begbie, sen., who wrote a monograph on the subject in 1849, found the spleen much enlarged, and Dr Warburton Begbie twice noticed the same thing ('Edin. Med. Journ.,' September, 1863, and April, 1868).

Even if it were proved that there is any constant lesion of the cervical sympathetic ganglia, it would be difficult to explain how the symptoms of exophthalmic goitre could be caused thereby. Boddaert succeeded in producing enlargement of the thyroid body by ligaturing the jugular and thyroid veins in animals; and when he also divided the sympathetic nerves, the eyeballs became prominent. Thus it may be that paralysis of the nerves in question, by dilating the blood-vessels, gives rise to like effects. But it is worthy of notice that the recognised effects of paralysis of the cervical sympathetic are absent. Moreover, the excited action of the heart corresponds, not with paralysis, but with irritation of sympathetic nerves. Some writers have indeed endeavoured to account for all the phenomena of the disease on a theory of "irritation." But it is a sufficient objection to such a view that a primary irritation of a nerve-centre, lasting for months or years unchanged, is as yet unknown in pathology. Lastly, the suggestion has been made that the original starting-point of exophthalmic goitre may perhaps, after all, be in the spinal cord or the bulb, or the brain.

With regard to the *diagnosis* of exophthalmic goitre little used to be said. But it is important to note that one must be on the look-out for slight cases and for rudimentary forms, in which one or other of the cardinal symptoms may be absent. This is a point on which Trousseau insisted; and Dr Wilks has recorded several instances which might have been set down as examples of ordinary chlorosis but for the failure of ferruginous medicines to cure them.

Event.—Exophthalmic goitre is not often traced to a fatal termination in hospital experience. We have indeed made six or seven autopsies at Guy's Hospital since 1868; but the only case in which death appeared directly due to the disease was that of which Dr Wilks has recorded the details; and in this there was bronchitis with expectoration of mucus tinged with blood. Two (or perhaps three) patients died of pleurisy, with or without pneumonia; one of rheumatic pericarditis and endocarditis. One patient succumbed unexpectedly, after having been ailing for a day or two; the stomach and intestines were found to be affected with a remarkable form of follicular inflammation, the solitary glands and Peyer's patches being very prominent, and the whole mucous membrane intensely injected, swollen, ecchymosed, and lined with mucus. In the remaining case death was caused by the inhalation of an anæsthetic.

In all but two of these cases the disease was of recent origin, having lasted only a few months. One patient had suffered for four years. It does not appear that those affected with exophthalmic goitre, like so many with other chronic and incurable maladies, go about from hospital to hospital until they die. The only possible inference seems to be that most cases at length end in recovery; but it is difficult to obtain positive evidence in support of this conclusion. The duration of the complaint is too long, and the natural process of cure is too gradual, to allow of its being traced in a ward of which the inmates are constantly changing. Dr Hale White has, however, followed up twelve patients previously under treatment in Guy's Hospital, and found ('Brit. Med. Journ.,' July 24th, 1886) that four were well after illnesses of two, three, five, and eight years; and that one was much better. Seven were dead: two suddenly, and no cause was found *post mortem;* one from mitral regurgitation, one from abscess in the axilla and pyæmia, one from gastric ulcer, one from phthisis, and one from unknown causes.

Treatment.—The medicinal treatment of exophthalmic goitre is very unsatisfactory. Trousseau was strongly convinced that iodine was generally injurious, although he admitted that it was sometimes useful. He also believed that the tincture of iron did harm, and most observers will at least admit that it seldom, if ever, does good. Traube, however, is said to have given iron and quinine alternately, each for three weeks at a time, with great advantage. Digitalis, which was recommended by Trousseau, seems to have no power whatever of slowing the pulse or tranquillising the heart. In two cases ergot was prescribed, but without marked benefit.

Some German physicians have applied galvanism to the neck with the object of "galvanising the sympathetic nerves." A battery of six or eight cells is used; the negative pole is placed upon the spine below the fifth cervical vertebra; the positive pole at different levels in front of the sterno-mastoid muscles. In this way the pulse was brought down from 130 to 70 or 80, and the general condition of the patient is said to have been much ameliorated. The application of a full current to the closed eyelids is also recommended, as tending to diminish the exophthalmos. The treatment has been adopted elsewhere with little advantage.

END OF THE FIRST VOLUME.